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Diseases Thyroid Gland and Pregnancy

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Annotation: This review contains discusses of functional properties of the thyroid gland during the physiological pregnancy, particularly during in its different periods. Influence of iodine insufficiency and radioactive effects on the functional status of the pregnant women's thyroid gland. Influence of the pathology of the thyroid gland on pregnancy, a fetus and a pregnant woman. Also, methods of correction of various pathological changes of functions of the thyroid gland during the gestation are discussed, as they can lead tohabitual miscarriage and to the pathology of the newborn. Preventive measures for the women living in the region of iodine deficiency at the pregnancy planning stage and individual iodine prevention, during the all pregnancy period and in the breastfeeding period.

Keywords: thyroid gland, pregnancy, iodine deficiency, radiation.

The regulation of the reproductive function of women is carried out by various organs, one of which is the thyroid gland (TG). Thyroid pathology has a negative effect on the course of pregnancy, fetal development and adaptation of the newborn [1]. During pregnancy, the metabolism of iodine and thyroid hormones changes, which is an important factor in the diagnosis and treatment of thyroid pathology in pregnant women and women planning a pregnancy. From the first weeks of pregnancy, the functioning of the thyroid gland changes due to the influence of many factors that have a stimulating effect. First of all, this is hyperstimulation of the thyroid gland by human chorionic gonadotropin (hCG), which leads to a physiological decrease in the level of thyroid stimulating hormone (TSH) in the first trimester (due to cross-reactivity). Increased production of hCG, which has a similar structure to TSH (identical α-subunits). causes an increase in the level of free thyroxine (T4) and, as a result, suppression of TSH. Normally, at least 20% of pregnant women in the first trimester have a reduced level of TSH. [2,3]

The main function of the thyroid gland is to provide the body with sufficient thyroid hormones, since thyroid hormones (triiodothyronine - T3, thyroxine - T4) are necessary for the normal functioning of all organs and systems of the body. During pregnancy, they regulate the processes of development, maturation and renewal of almost all tissues and are of exceptional importance for the formation and development of the fetal brain, growth and maturation of the skeleton and reproductive system. Thyroid-stimulating hormone of the pituitary gland (TSH), the secretion of which is controlled by thyroliberin (TRH - thyrotropin-releasing hormone) of the hypothalamus, regulates the function of the thyroid gland. The role of thyroid hormones (TH) in the processes of formation, regulation and maintenance of normal function of the reproductive system in women has been comprehensively studied. Under physiological conditions, TH support optimal production of prolactin, synthesis of monoamines, activity of the corpus luteum and, thus, the normal level of functioning of the gonadal system. In this regard, asymptomatic hypothyroxinemia in women of childbearing age may cause infertility (disruption of the ovulation process) or miscarriage (reduction in the functional activity of the corpus luteum). It has been established that dysfunction of the thyroid gland may cause premature or late puberty, menstrual irregularities, anovulation, infertility, miscarriage, fetal and neonatal pathologies. The state of the reproductive system, in turn, has a huge impact on the thyroid gland, which is confirmed by changes in its function during pregnancy, lactation, benign tumors and hyperplastic processes of the female genital organs. Experimental work conducted in recent decades confirmed the presence of receptors for TSH and T3 in the ovary, which determines the possibility of direct influence of thyroid dysfunction on steroidogenesis, ovulation, and corpus luteum function. In the first half of pregnancy, the thyroid gland of the fetus does not yet function and its development is fully dependent on the thyroid hormones of the pregnant woman. Therefore, the need for thyroid

hormones during pregnancy increases by 40-50%. During a physiologically proceeding pregnancy, an increase in the activity of the thyroid gland is observed due to hyperproduction of chorionic gonadotropin, increased production of estrogens, thyroxine-binding globulin, increased renal blood flow and glomerular filtration, leading to increased excretion of iodine in the urine, more intense blood supply to the thyroid tissue, and some increase in tissue mass. As a result, a normally proceeding pregnancy is accompanied by an increased need for thyroid hormones and iodine with the possibility of developing hyperthyroidism. Hypotyroxinemia of any origin has the most unfavorable consequences in the early stages of pregnancy.[4]

Iodine deficiency. In the literature, the term "iodine deficiency states" refers to pathological conditions that develop as a result of iodine deficiency and that can be prevented by normalizing iodine consumption. Iodine deficiency in the body leads to disruption of the synthesis and secretion of thyroid hormones. In pregnant women, reduced iodine consumption leads to hyperstimulation of the thyroid gland and the formation of goiter in the mother and fetus. The spectrum of possible iodine deficiency pathology was determined in 2001 by WHO (Table 1). Replenishment of iodine deficiency throughout the gestation period prevents spontaneous abortion, the development of goiter in the pregnant woman and iodine deficiency pathology in the fetus. Iodine preparations are used (alone or in the form of vitamin complexes) in a dose of 200 mcg / day. Contraindication for the prescription of iodine preparations is diffuse toxic goiter (DTG) [5.6].

Hypothyroidism

The prevalence of hypothyroidism among pregnant women is 2–3%. Since uncompensated hypothyroidism has a negative impact on the course of pregnancy and the fetus, diagnosis of the thyroid gland in a pregnant woman is of great importance. Complications of uncompensated hypothyroidism during pregnancy are presented. Clinical data are not a criterion for making a diagnosis. Laboratory confirmation of overt hypothyroidism is an increase in the TSH level above the trimester-specific reference value and a simultaneous decrease in free T4 or a TSH level 10 mIU/l regardless of the free T4 level. The main diagnostic criterion for primary hypothyroidism is an elevated TSH level. Given the risk of miscarriage, premature birth and fetal malnutrition, when diagnosing overt hypothyroidism during pregnancy, immediate administration of L-thyroxine preparations and normalization of TSH at the level established for a given trimester is required [7]. Hypothyroidism in pregnant women is an absolute indication for replacement therapy. L-thyroxine is prescribed in full dose without titration [8]. TSH levels are monitored 1 month after the start of replacement therapy, then once every 1–1.5 months. In patients with hypothyroidism, the dose of L-thyroxine may increase by up to 30% upon the onset of pregnancy [9]. Most often, these are patients receiving L-thyroxine replacement therapy, in whom TSH levels of > 1.2 mIU/L were detected before pregnancy [10]. After childbirth, the L-thyroxine dose often becomes lower than it was before pregnancy.

When Graves' disease is detected in a pregnant woman, several aspects of the impact on the fetus must be taken into account: the effect of thyrotoxicosis, antithyroid drugs, and TSH receptor antibodies [10]. If maternal TSH receptor antibodies increase in the early period of gestation and the antibody level does not decrease in the second trimester, there is a real risk of developing fetal and neonatal thyrotoxicosis. This risk can be assessed by fetal ultrasonography revealing goiter, tachycardia (> 160 bpm), accelerated bone growth, and decreased fetal motor activity. In some cases, blood can be taken from the fetal umbilical vein using chordocentesis to make a diagnosis [11]. If the mother does not receive antithyroid therapy and the fetus constantly has tachycardia, it is advisable to prescribe PTU 200–400 mg/day or thiamazole 20–30 mg to the mother, and, if necessary, sodium levothyroxine to maintain euthyroidism in the mother. The consensus developed by the ATA does not recommend prescribing antithyroid therapy to the mother if thyrotoxicosis is detected in the fetus. Thyrotoxicosis and treatment with antithyroid drugs increase the potential risk of developing genetic abnormalities. Refusal to treat thyrotoxicosis as a cause of subsequent genetic diseases remains unproven. At the same time, cases of drug-dependent congenital anomalies have been described, including aplasia cutis and other serious embryopathies. There are known cases of aplasia cutis in children born to mothers using MM. To date, cases of aplasia cutis have not been detected with the use of PTU. Studies of the

effect of maternal antithyroid drugs on the fetal thyroid gland have not shown any differences between PTU and MM. Thus, the drug of choice during pregnancy, especially in the first trimester, is PTU; in its absence or allergic reactions to it, thiamazole can be used [12]. Treatment of Graves' disease during pregnancy with antithyroid drugs can induce the development of fetal hypothyroidism. Hypothyroidism in the fetus has a severe effect on the development of the central nervous system, which can be prevented by prescribing a dose of antithyroid drugs that maintain the level of thyroid hormones in the mother at the upper limit of the norm.

Literature:

1. Adylkhanov T. A., Masadykov A. S. // Features of the epidemiology of diseases of the thyroid gland in the eastern region of the Republic of Kazakhstan in modern conditions // Medicine. 2009. Pp. 42–45.
2. Aldyngurov D. K. // Medical and social aspects of improving the rehabilitation of patients with diseases of the thyroid gland: // diss. PhD. Almaty, 2013. 125 p.
3. Balabolkin, M. I. // Fundamental'naya i klinicheskaya tireoidologiya // M. I. Balabolkin, E. M. Klebanov, V. M. Kreminskij. – M.: Medicina, 2007. – 814 s.
4. Burko, I. I. Patologiya shhitovidnoj zhelezy' i beremennost': ucheb-metod. posobie / I. I. Burko, L.I. Danilova. – Minsk, 2011. – 18 s.
5. Danilova, L. I. Bolezni shhitovidnoj zhelezy' i associirovanny'e s nimi zabolevaniya / L. I. Danilova. – Minsk: Nagasaki, 2005. – 470 s.
6. Dedov, I. I. Klinika i diagnostiki e'ndokrinny'x narushenij: ucheb.-metod. posobie / I. I. Dedov. – M., 2005. – 26 s.
7. Dedov, I. I. Racional'naya farmakoterapiya zabolevanij e'ndokrinnoj sistemy' i narushenij obmena veshhestv: rukovodstvo dlya praktikuyushhix vrachej / I. I. Dedov, G. A. Mel'nichenko. – M.: Litera, 2006. – 52 s.
8. Dedov, I. I. E'ndokrinologiya: nacional'noe rukovodstvo / I. I. Dedov, G. A. Mel'nichenko. – M.: GE'OTAR-Media, 2013. – 1072 s.
9. Valdina, E. A. Zabolevaniya shhitovidnoj zhelezy' / E. A. Valdina. – SPb.: Piter, 2006. – 386 s.
10. Mel'nichenko, G. A. Zabolevaniya shhitovidnoj zhelezy' vo vremya beremennosti: posobie dlya vrachej / G. A. Mel'nichenko, V. V. Fadeev, I. I. Dedov. – M., 2003. – 47 s.
11. Mel'nichenko, G. A. Zabolevaniya shhitovidnoj zhelezy' i beremennost' / G. A. Mel'nichenko // RMZh. – 1999. – T. 7, № 3. – S. 5–10.
12. Mel'nichenko, G. A. Zabolevaniya shhitovidnoj zhelezy' vo vremya beremennosti. Diagnostika, lechenie, profilaktika: posobie dlya vrachej / G. A. Mel'nichenko, V. V. Fadeev, I. I. Dedov. – M.: MedE'kspertPress, 2003. – 48 s.