MODERN MEDICINE AND PRACTICE
https://inovatus.es/index.php/ejmmp
EUROPEAN JOURNAL OF MODERN MEDICINE AND PRACTICE
Vol. 4 No. 11 (Nov - 2024) EJMMP ISSN: 2795-921X

THE SPECIFICITY OF AUTOIMMUNE THYROIDITIS IN PREGNANCY

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Abstract: Currently, diseases of the endocrine system, including thyroopathy, are amongthe top five among extragenital pathology during pregnancy. It is known that thyroiddysfunction has a negative effect on all parts of metabolism, microcirculation, vasculartone, immune and hormonal homeostasis, which, in turn, is the cause of complicatedpregnancy and childbirth and a high perinatal risk factor. That is why much attention ispaid in the scientific world to improving medical care for such patients. However, manyissues remain debatable to date, in particular the problem of individual iodine prophylaxisduring pregnancy in patients with autoimmune thyroiditis.

Keywords: pregnancy, autoimmune thyroiditis, iodine prophylaxis, TSH.

Thyroid diseases are becoming one of the most important problems, especially in the elderly. The disproportionate changes in our nature are also reflected in the health of the population. Studies conducted by various authors have shown that the introduction of pharmacological doses of iodine can lead to the appearance of thyroid antibodies in genetically predisposed individuals [2, 4, 15, 18]. At the same time, a number of studies did not determine the negative effect of physiological doses of iodine on autoimmune processes in the thyroid gland. In addition, some studies suggest that iodine deficiency may predict thyroid dysfunction in patients with AIT.

Thyroid autoimmunity affects 5–20% of the female population of childbearing age. Even in the absence of overt maternal thyroid dysfunction, thyroid autoimmunity has been reported to be associated with an increased risk of adverse pregnancy outcomes and impaired fetal neurodevelopment. Present evidence indicates that thyroid autoimmunity poses a risk for miscarriage and preterm delivery. By contrast, with regard to infertility most studies failed to find significantly different likelihood of pregnancy in euthyroid women with and without thyroid antibodies undergoing assisted reproduction procedures. Finally, uncertainty surrounds the role of thyroid autoantibodies on fetal and neonatal outcomes, since an increased risk for perinatal death, intrauterine growth restriction (IUGR), and respiratory distress syndrome has been inconsistently reported, and only few studies addressed the impact of maternal thyroperoxidase antibodies (TPOAbs) during pregnancy on cognitive functioning of the child, once again providing mixed results. Whether thyroid antibodies have a direct effect on neonatal outcomes, including neurodevelopment in the progeny, or whether the observed associations are related to maternal autoimmune-related thyroid insufficiency needs to be still clarified.

Thyroid disease is the second most common endocrine condition in women of childbearing age1 . If untreated, these conditions may affect mother and fetus. Last few decades knowledge regarding thyroid dysfunction associated with pregnancy was expanded. No less a problem are subclinical forms of disease2 , that are very frequent but not easily recognized without specific screening programs3 . The effect of thyroid dysfunction on conception, pregnancy and the outcome of the same is a current issue of the recent studies. It

is estimated that approximately 8-12% of all pregnancy losses are the result of endocrine factors4 . At least 2-3% of women have some form of thyroid dysfunction during pregnancy, and circa 10% of women have thyroid autoimmune disease despite euthyroidism5 . Thyroid dysfunction in pregnancy is classified as forms of hypothyroidism (positivity of thyroid autoantibody, isolated hypothyroidism, and subclinical or overt hypothyroidism), hyperthyroidism, and autoimmune disease, but also thyroid nodules and cancer, iodine insufficiency and postpartum thyroiditis that refer to thyroid dysfunction within the first year after delivery or miscarriage, when the known immunosuppressive effect of pregnancy disappears. It is the bidirectional relationship between autoimmunity and reproduction of great interest 6 , but detailed medical history and thyroid examination of the pregnant women are needed. It remains controversial if pregnant women should be screened for thyroid dysfunction. Some scientific societies have proposed to assess thyroid function during the first trimester of pregnancy and ideally before week 10 of gestational age8 . Reasons for screening because of existing high-risk conditions for thyroid diseases in pregnant women should be: history of previous thyroid dysfunction, goiter, positive thyroid antibodies, cervical irradiation, or thyroid surgery, age more than 30 years, family history of thyroid disease, presence of clinical signs or symptoms of hypothyroidism, diagnosis of type 1 diabetes mellitus or any other autoimmune disease, history of repeated abortions, prematurity, or infertility, morbid obesity, treatment with lithium, amiodarone, or recent administration of iodinated contrast and living in an area with moderate to severe deficiency of iodine.

Limitations in our current knowledge of physiologic changes in thyroid function during the periconceptional period and the pregnancy enable us to make a consensus over screening, diagnosis and treatment of thyroid disease. The aim of this review was to examine recent studies on the assessment of thyroid dysfunction in pregnancy, its treatment and newly perspective of thyroid autoimmunity in pregnant euthyroid women in achieving fertility.

Thyroid gland faces great challenge in pregnancy when many hormonal changes occur. These changes include the estrogen stimulated increase in thyroxin-binding globulin (TBG), resulting in an increase in total thyroxin (TT4), thyroglobulin, and the increase in serum concentration of human chorionic gonadotropin (hCG)10 . Increased HCG directly stimulates the thyroid gland; increases free thyroxin (FT4), and suppress serum thyroid-stimulating hormone (TSH) concentrations. Also, peripheral metabolism of thyroid hormones is modified, under the impact of the placental “type 3” iodothyronine deiodinase. Iodide and iodothyronines are transported from maternal circulation to the fetus; the recommended iodine intake is 250 µg/day, not more than 500 µg/day11 . Iodine deficiency cause goiter in mother and fetus. TSH levels increase and reach the highest value in the third trimester, on the other hand free fractions of thyroid hormones decrease. TSH levels are higher in the second half of pregnancy, because of decreased hCG and free thyroid hormones levels. In the last months of pregnancy, FT4 levels were often below the reference interval 12 , but an increase in reverse T3 during pregnancy was found. No optimal diagnostic tests for fT4 during pregnancy exist 13 . Thyroid volume is positively correlated with total body water14 , the increased vascularity may be the reason for the increase of thyroid volume15 . Fetal thyroxin production does not occur until 8-10 week of gestation. Other hormonal changes occur, like increase of mean plasma levels of testosterone and androstenedione, and luteinizing hormone (LH) secretion. Thyroid hormone, triiodothyronine (T3), receptors are found in the trophoblast. In vitro studies show that thyroid hormones act directly on the early development of the placenta, stimulating angiogenesis and promoting invasion and differentiation of embryonic cells. Thyroid receptors (TR) α1 and β1 and THRAb are expressed in human endometrium, the highest level is seen in the receptive endometrium, and paracrine factors like leukemia inhibitory factor and leptin are of importance for successful embryo implantation. Abalowich et al 17 established upper limit for thyrotrophin 2.5 mIU/L in the first trimester, and 3.0 mIU/L in the second and third trimesters. The lower physiological limit could be 0.1 mIU/L in the first trimester, 0.2 mIU/L in the second, and 0.3 mIU/L in the third.

Conclusions: Thyroid hormones are involved in control of menstrual cycle and in achieving fertility affecting the actions of follicle-stimulating hormone and luteinizing hormone on steroid biosynthesis by specific T3 sites on oocytes, therefore affect all aspects of reproduction. Further randomized trials are needed to expand our knowledge of physiologic changes in thyroid function during the pregnancy, so pathological changes should be better analyzed. It is necessary to delineate women with thyroid dysfunction, including thyroid autoimmunity in euthyroid women, who require treatment and follow up during pregnancy. Further studies are needed to reveal mechanisms by which thyroid autoimmunity in euthyroid women affect fertility, especially the success of assisted reproductive technology in achieving the same and validity of levothyroxine administration in TAI positive women.

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