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HYPOTHYROIDISM AND PREGNANCY

Demonstration of case

INTRODUCTION Hypothyroidism is the disease state in humans caused by insufficient production of thyroid hormone by the thyroid gland and is a frequent disease among women and has a particular significance during pregnancy. The manifested hypothyroidism is found in 0,9% pregnancies. Research has found that with 2,5% pregnancies, the thyroid stimulating hormone (TSH) was higher than 6 mU/L.

The most common etiological factors of hypothyroidism in the developed part of the world are the following autoimmune diseases:

1 – Hashimoto's disease

2 – Primary autoimmune hypothyroidism .

The origin of hypothyroidism in many third world countries is the decreased consumption of iodine. Pregnancy is an euthyroid condition, characterized by complex changes in the function of the thyroid gland. In different periods of pregnancy, there are changes in the globulin values of certain thyroid hormones. In the first half of pregnancy there are increased levels of globulin which binds thyroxin, called thyroxin-binding globulin (TBG) and continues throughout the pregnancy. In the first trimester, we see an increased concentration of human choriogonadotropin which causes a temporary decrease of TSH values between the 8th and 12th week, and with 20% of pregnant women, this decrease is accentuated. In the second half of pregnancy, there are changes in the peripheral metabolism of the thyroid gland metabolism.

The passage of thyroid hormones through the placenta had not been proven for a long time. Contemporary studies have demonstrated that T4 is present within the celome liquid as early as the 6th *gestational week*, and its values with the fetus with agnesium of the thyroid gland are about 30% from the normal values in the circulation, which is of special significance with these fetuses. The nuclear T3 receptors may be identified in fetal brain cells from the 10th gestational week. The fetal thyroid gains

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complete function in the 16th gestational week. The mother's T4 values and its flow to the fetus and the fetal thyroid gland are crucial for the early and late phase in the fetal brain development. During the first phase of fetal brain development with maximum growth and brain structure development, neural multiplication and organization which occurs in the third trimester, the thyroid hormones are primarily of maternal origin. The second maximum phase of fetal brain growth occurs in the third trimester and continues throughout second and third year of age, and the thyroid hormones of the thyroid gland originate from the neonate. Low concentrations of maternal tiroxine in the second trimester result in irreversible neurological deficit with newborns, while damages are less or partially reversible when the deficit occurs later.

Women with hypothyroidism are more likely to incur pregnancy complications. There are frequent miscarriages, *polihydramnions*, hypertensions and diabetes as well as fetal intrauteral delays.

Symptoms of decreased thyroid function may be unnoticed for a long period of time and masked by hypermetabolic gravidity condition. Therefore, it is of crucial importance to discover thyroid diseases with pregnant women as soon as possible and initiate proper therapy. The diagnosis is determined after finding increased TSH levels in the serum, low fT4 and values as well as antithyroid antibodies.

RESEARCH OBJECT: demonstration of a successfully completed pregnancy with a hypothyroid patient.

MATERIAL AND METHODS: In order to demonstrate the case, medical documentation from the Gynecology Department of the MC in Arilje was used.

DEMONSTRATION OF PATIENT: The patient J.D. is 36. years old, married 6 for years. Upon anamnesis, the woman is a null par who has been treated in the last two years for hypothyroidism problems and has been taking 125mg Letrox tablets. The miscarriage anamnesis revealed that she had had two previous miscarriages which occurred prior to hypothyroidism diagnosis, during the first gravidity trimester. She came for an examination due to menstruation absence. Following ultrasonograpic examination, vital pregnancy was verified, in accordance to the 8th *gestational week*. The patient was administered Letrox and vitamin therapy. During the 9th *gestational week* laboratory analysis and TSH, T4, T3 and OGTT levels revealed physiological borderlines. The dose of 125mg of Letrox remains administered. The pregnancy was being controlled pursuant to protocols of high risk pregnancies. During the 15th gestational week, the TSH values increased to 7,8 mU/L. Other laboratory results were within physiological limits. The systole and diastole blood pressure did not exceed 110/80 mmHg. The ultrasonograph fetal result indicated eutrophic growth. An endocrinologist was consulted who increased the Letrox dose to 150mg. During the 17th gestational week, early amniocentesis (RAC) was done due to age (46,xx). During the 21st gestational week a 4D ultrasound was performed with the following

results: BPD 55mm, HC 196mm, AC 172mm, FL 36mm, HC/AC 1,14, FL/AC 21, with the placenta normally positioned on the front wall, with an adequate quantity of fetal water. The morphology and dynamics of the fetal growth were completely in order. During the 22nd gestational week, the TSH levels increased to 8,9 mU/L and after a repeated consultation with the endocrinologist, the Letrox dosage was increased to 175 mg. Repeated laboratory analysis and OGTT results proved to be within physiological limits as well as the levels of measured blood pressure. In the 28th gestational week TSH results indicated to be within physiological limits and the medicine dose remained the same. An ultrasonographic fetal supervision was done once a month with CTG weekly after week 32 along with TSH values from weeks 4 to 6. After week 38, the pregnancy was completed with an elective Caesarian incision followed by live female childbirth TT 3400/51 AP 9. The following postpartum course was in order.

RESUME: After taking into consideration all the facts, it is important to recognize this disease both in clinical and subclinical form, and, if possible, treat preconceptionally and, of course, as early as possible during pregnancy. Some authors advocate routine TSH checks prior to or during early pregnancy. TSH must be determined with patients suffering from goiter, positive thyroid antibodies, diagnosed autoimmune diseases as well as those with family anamnesis of thyroid gland diseases.

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