

HYPOTHYROIDISM IN PREGNANCY

Adedayo Jesutomi Eunice

Scientific adviser: docent *M. P. Kapliyeva, PhD*

Establishment of Education
«Gomel State Medical University»
Gomel, Republic of Belarus

Introduction

Pregnancy is a period that places great physiological stress on both the mother and the fetus. However, if pregnancy is compounded by endocrine disorders such as hypothyroidism, the potential for maternal and fetal adverse outcomes can be immense. Hypothyroidism is widely prevalent in pregnant women and the rate of detection, especially in a developing country like India, Nigeria and Ghana has not kept pace with the magnitude of the problem. Since hypothyroidism is easily treated, timely detection and treatment of the disorder could reduce the burden of adverse fetal and maternal outcomes, which are very commonly encountered.

Aim

To show that maternal and fetal outcomes can be immense, if untreated. Also to create awareness in young woman with hypothyroidism.

Discussion

Thyroid physiology is perceptibly modified during normal pregnancy. These alterations take place throughout gestation, help to prepare the maternal thyroid gland to cope with the metabolic demands of pregnancy, are reversible post-partum and the interpretation of these changes can pose a challenge to the treating physician.

The most notable change is the increase in thyroxine-binding globulin (TBG). This begins early in the first trimester, plateaus during midgestation, and persists until shortly after delivery. This is due to stimulation of TBG synthesis by elevated maternal estrogen levels, and more importantly, due to a reduced hepatic clearance of TBG because of estrogen-induced sialylation [1]. This increased TBG concentration leads to an expansion of the extra-thyroidal pool and results in elevated total T_3 and T_4 levels due to an increase in maternal thyroid hormone synthesis. Maternal thyroid hormone synthesis is also increased due to an accelerated renal clearance of iodide resulting from the increased maternal glomerular filtration rate.

Enhanced metabolism of T_4 in the second and third trimesters, due to a rise in placental type II and type III deiodinases, which convert T_4 to T_3 and T_4 to reverse T_3 and T_2 respectively, act as further impetus to T_4 synthesis. Plasma iodide levels decrease due to both increased thyroxine metabolism and increased renal iodide clearance. All these changes lead to an increase in the size of the thyroid gland in 15 % of pregnant women, which returns to normal in the post-partum period. Serum human chorionic gonadotropin (hCG) has intrinsic thyrotropic activity, which increases after fertilization and peaks at 10 to 12 weeks. Hence, in the first trimester, free T_3 and T_4 levels increase slightly and thyroid stimulating hormone (TSH) levels decrease in the first trimester with a readjustment in the second and third trimesters, when hCG levels decrease. As a consequence, cut-offs to determine hypothyroidism in pregnancy are different in the first trimester and the rest of the pregnancy.

The prevalence of hypothyroidism during pregnancy is estimated to be 0,3–0,5 % for overt hypothyroidism and 2–3 % for subclinical hypothyroidism. Autoimmune thyroiditis is the commonest cause of hypothyroidism during pregnancy. Other causes include radioiodine ablation of thyroid while treating hyperthyroidism or thyroid cancer, surgery of the thyroid tumors. However, worldwide, iodine deficiency still remains one of the leading causes of hypothyroidism, both overt and subclinical. Hypothyroidism during pregnancy is usually asymptomatic, especially when subclinical. Signs and symptoms which suggest hypothyroidism include inappropriate weight gain, cold intolerance, dry skin and delayed relaxation of deep tendon reflexes. Other features like constipation, fatigue, and somnolence are usually attributed to pregnancy.

Negro et al. in a pioneering study, found that LT_4 administration in euthyroid pregnant women with autoimmune thyroid disease decreased the rates of negative obstetric outcomes in women

with a TSH value greater than 2.0 mIU/liter and/or a high titer of thyroid antibodies [2]. In view of the negative maternal and fetal outcomes of hypothyroidism, carefully monitored thyroid hormone treatment of TPO antibody positive pregnant patients might be a prudent measure.

Subclinical hypothyroidism is defined as increased TSH with normal concentrations of FT₄. Women with subclinical hypothyroidism are more likely than euthyroid women to have TPO antibody positivity. Etiology is similar to overt hypothyroidism. Since multiple studies have shown that subclinical hypothyroidism is associated with an adverse outcome for the mother and offspring, most guidelines recommend thyroxine replacement in women with subclinical hypothyroidism. However, while thyroxine treatment has been shown to improve obstetrical outcome, it has not been proven to modify long-term neurological development in the offspring [3].

Isolated maternal hypothyroxinemia is defined as a low FT₄ and normal TSH, which can be found in approximately 1 to 2 % of pregnancies. In the Faster study, among the women with hypothyroxinemia and normal TSH, there was an increased odds ratio for preterm labor (1.62, 95 % CI 1.00–2.62), macrosomia (1.97, 95 % CI 1.37–2.83), and gestational diabetes (1.70, 95 % CI 1.02–2.84), but these results were not consistent. A study by Casey et al. concluded that isolated maternal hypothyroxinemia in the first half of pregnancy has no adverse effects on pregnancy outcome [4].

In some studies infants and toddlers whose mothers had reduced serum free T₄ concentrations (with normal TSH) during gestation (12 to 20 weeks) had lower mean intelligence, psychomotor, or behavioral scores compared with children born to women with normal thyroid function during gestation. However, till date, no study has shown benefit from levothyroxine treatment of isolated hypothyroxinemia during pregnancy, on pregnancy outcome or subsequent infant development.

Conclusion

The impact of maternal hypothyroidism on the fetus depends on the severity of the condition. A. Uncontrolled hypothyroidism: miscarriage, anaemia, intrauterine fetal demise and stillbirth preterm delivery, low birth weight, preeclampsia, developmental anomalies including reduced IQ, Maternal and congenital hypothyroidism resulting from severe iodine deficiency: profound neurologic impairment & mental retardation. If the condition is left untreated. Congenital cretinism: growth failure, mental retardation, and other neuropsychologic deficits including deaf-mutism. If cretinism is identified & treated in the first 3 months of life: near-normal growth and intelligence can be expected. For this reason, newborn screening for congenital hypothyroidism. B. Asymptomatic overt hypothyroidism. Women who had previously been diagnosed with hypothyroidism, (abnormal TSH and FT₄ levels), but who do not have symptoms. Impaired psychomotor development at 10 months in infants born to mothers who had low T₄ during the first 12 weeks of gestation. Low IQ scores in the offspring at 7 to 9 years of age was correlated with elevated maternal TSH levels at less than 17 weeks' gestation. An inverse correlation between a woman's TSH level during pregnancy and the IQ of her offspring confirmed that maternal hypothyroxinemia is a risk for neurodevelopmental abnormalities that can be identified as early as 3 weeks of age.

REFERENCES

1. *Vulsma, T.* Maternal-fetal transfer of thyroxine in congenital Hypothyroidism due to total organification defect or thyroid agenesis / T. Vulsma, M. Gons, J. Vijlder // N Engl. J. Med. — 2009. — Vol. 321. — P. 13.
2. *Negro, R. [et al.]* // Endocr Pract. — 2011. — Vol. 17. — P. 1–24.
3. Overt and subclinical hypothyroidism complicating pregnancy / M. Abalovich [et al.] // Thyroid. — 2002. — Vol. 12. — P. 63–66.
4. *Casey, B.* Treatment of subclinical hypothyroidism or hypothyroxinemia in pregnancy / B. Casey, E. Thom, A. Peaceman // N Engl J Med. — 2017. — Vol. 376. — P. 815–825.

УДК 615.835.3:616.831-005.4

HYPERBARIC OXYGEN THERAPY IN ISCHEMIC STROKE

Ajibade Moses Olamilekan

Scientific adviser: E.V. Serebrova

**Educational Establishment
«Gomel State Medical University»
Gomel, Republic of Belarus**

Introduction

Stroke is one of the leading causes of death in the world and the 5th cause of death in the United States. According to the American Stroke Association, 87 % of stroke cases are ischemic