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## Research Article

# FEATURES OF FETOPLACENTAL INSUFFICIENCY IN PREGNANT WOMEN WITH HYPOTHYROIDISM

Submission Date: February 10, 2022, Accepted Date: February 20, 2022,

Published Date: February 28, 2022 |

Crossref doi: <https://doi.org/10.37547/TAJMSPR/Volume04Issue02-06>

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## ABSTRACT

Hypothyroidism is a thyroid dysfunction in which the thyroid gland is not ready to produce enough hormones, leading to a number of diseases such as sickness, cretinism and myxedema. Thyroid hormones play an important role in maternal development for the brain and body of the child, during the first three months of motherhood the child depends on thyroid hormones delivered through the placenta thyroid hormones until 18-20 weeks of pregnancy.

## KEYWORDS

Hypothyroidism, fetoplacental insufficiency in pregnant women, pathomorphology.

## INTRODUCTION

Adequate placental function is critical for an uncomplicated pregnancy and better fetal development, as the placenta provides the supply of nutrients, gas exchange and excretion of metabolic products as well as estrogens, progesterones and prostaglandins.

Preterm birth has been associated with low thyroid function, and high thyroid function has been associated with preeclampsia in pregnant women and fetal growth retardation, which can result from adverse pregnancy outcomes. because of impaired placentation in early pregnancy, while placental tissue is alert to TG, the authors hypothesised that early maternal thyroid function may be a regulator of placentation.

Despite a growing body of basic evidence suggesting that TG plays a role in the regulation of placental development. Furthermore, because thyroid and placental dysfunction are associated with similar birth complications, the clinical association of thyroid function with adverse pregnancy outcomes may be mediated by changes in placental function or vice versa.

The presence of specific nuclear receptors and hormones detected in the cranial medulla at the eighth week of gestation, the free T<sub>4</sub> detected in the whole-brain and amniotic fluid and the demonstration of maternal thyroid hormone transfer through the placenta underline the role of thyroid hormones in cranial brain development. The complex interactions between the deiodinases iodoamino acids D<sub>2</sub> and D<sub>3</sub> throughout pregnancy facilitate the fine-tuning of the availability of adequate amounts of T<sub>3</sub> required for traditional brain development.

A number of pioneering studies by Man et al Haddow et al and newer studies by Rovet et al and Pop et al have proved once and for all that children born to mothers with hypothyroidism have a significantly increased risk of impaired ratio, psychobiological and learning skills. children born to untreated women with hypothyroidism had an AN ratio that was seven points below the average of children born to healthy women who received T supplements. This risk extended to children born not only to untreated girls but also to girls with suboptimal complementation. A study by Rovet et al found that these children had slight defects in intellectual perception of the world, but visual-spatial ability, language, fine motor skills and learning ability were not affected. This study emphasises the need for adequate monitoring of girls at the start of treatment.

Suboptimal placental function is associated with complications of labour as well as pre-eclampsia (which in turn further worsens placental haemodynamics and worsens cranial blood supply), fetal growth retardation and preterm birth, which is one of the leading causes of maternal and perinatal mortality worldwide. Hypothyroidism is difficult to diagnose during pregnancy because the signs will belong to the mother herself. Changes in the thyroid gland have a serious negative effect on both the mother and the foetus. To date, there is no consensus on the causes of inadequate function of the fetoplacental complex in this pathology, which makes this problem urgent.

## OBJECTIVE

To investigate the features of fetoplacental insufficiency in pregnant women with hypothyroidism. To solve this problem, it is necessary to conduct a

consistent study of the frequency, cause, mechanism of development and pathomorphological manifestations of placental abnormalities in pregnant women with hypothyroidism.

## MATERIALS AND METHODS

Placental tissue of pregnant women with hypothyroidism and charts of sick women with hypothyroidism of pregnant women were used as the study material. For histological examination, 5 pieces of 1 × 1 cm were cut from different parts of the placenta for light microscopy and then fixed in 10% neutral formalin. After embedding pieces in paraffin, sections were prepared on a microtome with subsequent staining with hematoxylin and eosin.

## RESULTS OF THE STUDY

Thyroid pathology progressed throughout gestation. A comprehensive study of placentas obtained both after natural childbirth and during cesarean section was carried out. The clinical symptomatology is polymorphic, usually non-specific and mainly related to the timing and severity of the hormone deficiency. acceptable, early treatment and maintenance of conventional thyroid hormone levels minimise the chance of maternal and cerebral complications and increase the chance that motherhood can be carried to term without serious complications. Hypothyroidism during pregnancy is sometimes asymptomatic, especially in the subclinical period. When pregnant women with hypothyroidism have been observed, signs and symptoms indicative of hypothyroidism include inadequate weight gain, cold intolerance, dry skin and delayed relaxation of deep connective tissue reflexes. Various symptoms such as constipation, fatigue and drowsiness are usually associated with pregnancy.

Macroscopic examination of placenta of women in labour with hypothyroidism and healthy women was carried out. In most patients, a number of changes in placental tissue were observed, such as increased mass, change in shape, and increased elastic consistency. Attachment of the umbilical cord is more often paracentral or central. The membranes are slightly thickened, ischaemic. In some placentas, there is an area occupied by infarcts and caverns. In some cases, lymphohistiocytic infiltrates of interstitial substance were found. The analysis of the histological structure of the villous tree revealed that a variant of pathological immaturity of the placenta was observed to a greater extent than in the control group. There were e a functional zones and thrombi in the intervillous space. Intermediate-type villi with moderate vascularization of their stroma and a reduced number of lateral branches dominated over a large area. The villi were not densely arranged. The placental tissue was dominated by intermediate and terminal villi of small calibre. Microscopic examination of the placenta of women in labour with hypothyroidism revealed connective tissue fibre overgrowth, hyperelastosis, edema, fibrinoid swelling and hyalinosis in the walls of small vessels, and thrombi were detected. The number of fibrinoid-altered and sclerosed villi also increased. The syncytiotrophoblast was desquamated on a large scale. An insignificant number of syncytial nodules were observed, the number of nuclei with karyopycnosis and karyorexis increased sharply. The stroma was loose. Capillaries were ischemic. In addition, degenerative and dystrophic changes in syncytiotrophoblasts and cytotrophoblasts were observed. The dystrophic changes were proteinaceous in nature. Thus, untreated maternal hypothyroidism will lead to premature birth, low birth weight and metabolic abnormalities in the baby. Over the years, ample

evidence has accumulated for the role of thyroxine in traditional fetal development.

Children born to mothers with iodine deficiency felt even worse, and many of them had attention deficit disorder. The complications that arise depend on the severity of hypothyroidism, how timely and early treatment is initiated, the various medical specialities and the extra-genital pathologies associated with this maternity. When maternal endocrine imbalances overlap, there can be undesirable consequences for each mother and foetus. It is generally accepted that hypothyroidism in pregnant women is associated with an increased risk of abortion, stillbirth, preterm birth, intrauterine death, developmental delay and congenital fetal anomalies, congenital hypothyroidism, anaemia, postnatal depression and internal organ dysfunction, resulting in excessive maternal morbidity, perinatal pathology and mortality.

## CONCLUSIONS

Thus, the placenta against the background of maternal thyroid pathology showed signs of damage, characterised by alteration, focal sclerosis and fibrinoid necrosis. Circulatory disturbances in the form of full-thickness and thrombosis have also been observed. The combination of obstetric and endocrine pathology increases the risk of pregnancy and delivery complications with various types of maternal thyroid pathology. Pregnancy can be a period of good physiological stress for both mother and foetus. However, if maternity is combined with endocrine disorders, such as hypothyroidism, the chances of adverse outcomes for the mother and the foetus are enormous. Since hypothyroidism is fully treatable, early detection and treatment of the disease can reduce the burden of adverse outcomes.

## REFERENCES

1. Pavlova T.V., Malyutina E., Petrukhin V., Markovskaya V. Hypothyroidism in pregnant women: clinical and morphological parallels. / J. Phys. No. 4. 2015. Pp. 47-49.
2. Pavlova T.V., Malyutina E.S. Influence of thyroid pathology on the condition of pregnancy and childbirth. / / Zh. nauchnye vedomosti. 2011. № 10 (105). Issue 14. pp. 9-14.
3. Kular N.K., Samsonova L.N., Kasatkina E.P. The course of perinatal period in pregnant women with diffuse nontoxic goiter, living in the region of moderately severe iodine deficiency / J. Gynecology. 2004. T. 6 no. 2. pp. 12-16.
4. Kamalova M. I., Islamov Sh. E., Khaydarov N.K.// Morphological changes in brain vessels in ischemic stroke. Journal of Biomedicine and Practice 2020, vol. 6, issue 5, pp.280-284
5. Nikonova L.V., Davydchik E.V., Tishkovsky S.V., Gadomskaya V. I. Thyroid diseases and pregnancy. / Journal of Grodno State Medical University No 1, 2016. Pages 82-87.
6. Deryabina E.G., Bashmakova N.V., Zilber N.A., Dankova I.V. The significance of active diagnostic and therapeutic tactics in autoimmune hypothyroidism and thrombophilia in early pregnancy / J. Vestnik RUDN, series Medicine, 2009, No.6 p.142-146.
7. Abalovich .M., Gutierrez S., Alcaraz G., Maccallini G., Garcia A., Levalle O. Overt and subclinical hypothyroidism complicating pregnancy / Thyroid. 2002. V. 12. P. 63.
8. Krassas, G.E., Poppe, K. & Glinoe, D. Thyroid function and human reproductive health. / Endocr. Ed. 31, 702-755 (2010).
9. Hershman, J.M. The role of human chorionic gonadotropin as a thyroid stimulant in normal pregnancy. / J. Clin. Endocrinol. Metab. 93, 3305-3306 (2008).





10. Lazarus, J. et al. European Thyroid Association Guidelines for the Treatment of Subclinical Hypothyroidism in Pregnant Women and Children, 2014. / Euro. Thyroid J. 3, 76–94 (2014).

