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The association of maternal thyroid function with placental hemodynamics during pregnancy

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Abstract. *We examined 164 pregnant women who were divided into three groups. Group I included 76 pregnant women (46.4 %) with euthyroid goiter of I–II degree. Group II consisted of 63 pregnant women (38.4 %) with subclinical hypothyroidism and diffuse thyroid goiter of I–II degree. Group III was the controls and consisted of 25 (15.2 %) pregnant women without thyroid pathology. The placenta was studied with the characteristics of ultrasound placentography, placental maturation disorders, the area and localization were determined, and pathological changes in the placental tissue were detected. Changes in the systolic-diastolic ratio in the uterine arteries and umbilical cord arteries were assessed, the resistance index in the uterine arteries, the pulsatile index in the fetal aorta and middle cerebral artery were determined using the method of color Doppler mapping of blood flow in the mother-placenta-fetus system. Study of the echographic picture of structural changes in the placenta revealed a significant impairment of its maturation, especially in the group with euthyroidism. Ultrasound screening revealed that in every second pregnant woman with thyroid disease, the condition of the placenta did not correspond to the gestational age, there were swelling, cysts and placental infarctions, a high frequency of diffuse changes in placental tissue, and hyperechogenic inclusions in the amniotic fluid. An increase in the resistance index in the uterine arteries of pregnant women, especially those with subclinical hypothyroidism, is noteworthy. With increasing gestational age, the peripheral resistance of the placental microvasculature increases due to involutional-dystrophic changes and circulatory disorders, which allows us to develop criteria for the prognosis and diagnosis of placental dysfunction, and to prevent perinatal disorders in pregnant women with thyroid disease.*

Keywords: *hypothyroidism; thyroid gland; pregnancy; placental dysfunction; dopplerometry; placentography*

Introduction

In pregnant women with thyroid disease caused by iodine deficiency (diffuse and nodular goiter), the number and weight of nodules increase, and the size of goiter increases in each pregnant woman [1–3].

Thyroid stimulation in iodine-deficiency diseases is accompanied by changes in biochemical parameters, which is manifested by an increase in thyroid volume in pregnant women by 30 % [4, 5]. In 16 % of these women, goiter is formed [6]. A goiter that occurs during pregnancy does not always have a reversible development, which leads to an increase in the incidence of thyroid pathology among women of reproductive age [7, 8].

Hyperthyroidism, goiter, hypothyroidism, and thyroiditis worsen the course of the gestational period [8–10].

The main complications of pregnancy and childbirth in pregnant women with iodine-deficiency diseases that determine the level of health of the mother and her newborn are: the threat of abortion, preeclampsia, fetal malformations, anemia, leading to placental dysfunction [11–14].

The aim was to study the state of the placenta using ultrasound to assess uteroplacental and fetal-placental blood exchange in pregnant women with subclinical hypothyroidism and euthyroidism.

Materials and methods

All 164 pregnant women examined were divided into three groups. Group I included 76 pregnant women (46.4 %) with euthyroid thyroid goiter of I–II degree. Group II consisted of 63 pregnant women (38.4 %) with subclinical hypo-

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thyroidism and the presence of diffuse thyroid goiter of I–II degree. Group III was the control group and consisted of 25 (15.2 %) pregnant women without thyroid pathology.

The condition of the placenta was assessed based on its structural integrity or degree of maturity [15]. Thus, no pregnant women with these changes were found in the control group. And the study of the echographic picture of structural changes in the placenta revealed a significant impairment of its maturation in 35 pregnant women with euthyroidism (46.1 %) and subclinical hypothyroidism in 26 observations (41.3 %), which was significantly higher than in the control group ($p < 0.05$) but did not differ between the groups of pregnant women with thyroiditis ($p > 0.05$).

The echographic criteria for premature placental maturation included the detection of grade II placental maturity by 32 weeks and grade III by 36 weeks of gestation. We did not find a clear correlation between the severity of clinical manifestations of placental dysfunction and the frequency of premature placental maturation. According to the results of the study, premature maturation of the placenta was more common (35.9 %) in pregnant women with euthyroidism, while late maturation of the placenta was more common in subclinical hypothyroidism — 27.0 % (Table 1).

Ultrasound screening revealed that in every second pregnant woman with thyroid disease, the condition of the placenta did not correspond to the gestational age. There was a higher degree of maturity, swelling, infarctions, and placental cysts. Placental thickness ranged from 0.5 to 4–5 cm. One pregnant woman in each group with thyroid disease (1.4 %) had a low placenta position, and two (2.8 %) had placenta previa. Of these, in 1 pregnant woman in group I it was central and in 3 in group II — lateral.

Ultrasound placentometry showed no differences in placental localization between the groups ($p > 0.05$). The thickness of the placenta corresponded to the normative values in 62 (81.6 %) pregnant women from group I and in 41 (65.1 %) pregnant women from group II ($p < 0.05$). In 10 (13.2 %) pregnant women of group I and 12 (19.1 %) pregnant women of group II ($p > 0.05$), the thickness of the placenta was reduced. In 4 (5.3 %) pregnant women from group I and 10 (15.9 %) pregnant women from group II, placental thickening was noted ($p < 0.05$). Pathologic changes in the form of calcifications and cystic formations were observed in 39 (51.3 %) pregnant women of group I and 41 (65.1 %) pregnant women of group II ($p > 0.05$).

Thus, in pregnant women with thyroid pathology, ultrasound examination revealed a high frequency of diffuse changes in the placenta, signs of umbilical cord wrapping around the fetal neck, and hyperechogenic inclusion in the amniotic fluid. Quite often, in group II of pregnant women ($p < 0.05$), ultrasound examination revealed indirect signs confirming the diagnosis of fetal distress (fetal muscle hypotension and increased motor activity), as well as moderate polyhydramnios.

To study the uteroplacental and fetal-placental circulation in pregnant women with thyroid disease, blood flow velocity curves were determined in the uterine arteries, umbilical cord arteries, aorta, and fetal middle cerebral artery.

The method of color Doppler mapping was used in the study of uterine arteries. Additionally, blood flow velocities were analyzed depending on the location of placentation.

For comparison, we determined the average systolic-diastolic ratio of blood flow velocities in the uterine arteries depending on the side of the placenta (Table 2).

The value of the systolic-diastolic volume of the uterine arteries in pregnant women with subclinical hypothyroidism ranged from 1.98 to 2.24 and was higher than in the control group in the second half of pregnancy. On the side of placentation, the mean values of the systolic-diastolic volume were significantly higher ($p < 0.05$) than on the other side,

Table 1. Characteristics of ultrasound placentography in pregnant women with thyroid disease (M ± m)

Indicator	Group					
	Controls, n = 25		Euthyroid goiter (I), n = 76		Subclinical hypothyroidism (II), n = 63	
	abs.	%	abs.	%	abs.	%
Disorders of placental maturation						
Premature	2	8.0	27	35.9*	9	14.3**
Late	1	3.90	8	10.5	17	27.0*, **
The area of the placenta						
Less than average	–	–	2	2.6	1	1.6
Above average	–	–	1	1.3	4	6.3
Localization of the placenta						
Back wall	13	52.0	41	54.0	37	58.7
Front wall	8	32.0	22	29.0	16	25.4
Uterine floor	4	16.0	13	17.1	10	15.9
Pathological changes in the placenta						
Swelling	–	–	–	–	3	4.8
Calcination	–	–	22	28.9*	7	11.1*, **
Cysts	–	–	14	18.4*	3	4.8**
Infarctions	–	–	3	4.0	1	1.6

Notes: * — probability of difference (p < 0.05) compared to the control group; ** — probability of difference (p < 0.05) compared to group I.

especially in complicated pregnancy (preeclampsia, threat of abortion). Similar changes were observed in the index of resistance in the uterine arteries in the compared groups of pregnant women with thyroid pathology at 28–30, 35–37 and 38–40 weeks of pregnancy (Table 3).

The increase in the index of resistance in the uterine arteries in pregnant women of group II ($p < 0.05$) and especially on the side of the placentation ($p < 0.05$) is noteworthy.

In the presence of combined severe forms of preeclampsia, abnormal values of uteroplacental blood flow indices were found.

At the same time, in most cases, the values of the vascular resistance index were within the normal range in accordance with the gestational age.

The fetal-placental circulation was studied by analyzing the curves of blood flow velocities in the umbilical cord artery (Table 4).

In pregnant women with thyroid diseases, the decrease in the indices of peripheral vascular resistance of the umbilical cord arteries was similar to the control group ($p > 0.05$). However, with increasing gestational age, the peripheral resistance of the placental microvasculature increased due to involutional and dystrophic changes and circulatory disorders (Table 4).

To determine the degree of fetal hemodynamic disorders and fetal compensatory capacities in patients with thyroplasia, we evaluated the curves of blood flow velocities in the aorta and in the fetal middle cerebral artery (Table 5).

In our studies, the absence of significant differences in fetal cerebral circulation in the group of pregnant women with thyroid disease did not differ from the control group ($p > 0.05$). This can be explained by the fact that disorders in the mother-placenta-fetus system against the background of drug therapy aimed at normalizing fetoplacental and utero-

Table 2. Systolic-diastolic ratio in the uterine arteries (M ± m)

Indicator	Group		
	Controls, n = 25	Euthyroid goiter (I), n = 76	Subclinical hypothyroidism (II), n = 63
Term in weeks			
23–25	1.93 ± 0.02	1.91 ± 0.03	2.01 ± 0.04
28–30	1.80 ± 0.04	1.76 ± 0.04	2.24 ± 0.02*, **
35–37	1.62 ± 0.04	1.74 ± 0.04*	1.98 ± 0.04*, **
38–40	1.62 ± 0.04	1.72 ± 0.03*	1.99 ± 0.05*, **
Side			
Placentation	1.60 ± 0.05	1.68 ± 0.05	2.12 ± 0.04*, **, ***
Nonplacentation	1.65 ± 0.05	1.72 ± 0.04	1.98 ± 0.05*, **

Notes: here and in Table 3: * — probability of difference (p < 0.05) compared to the control group; ** — probability of difference (p < 0.05) compared to group I; *** — probability of difference (p < 0.05) between the sides of placentation in group II.

Table 3. Indicators of uterine artery resistance index (M ± m)

Indicator	Group		
	Controls, n = 25	Euthyroid goiter (I), n = 76	Subclinical hypothyroidism (II), n = 63
Term in weeks			
23–25	0.47 ± 0.01	0.45 ± 0.02	0.50 ± 0.02
28–30	0.45 ± 0.02	0.44 ± 0.02	0.52 ± 0.02*, **
35–37	0.40 ± 0.02	0.42 ± 0.04	0.54 ± 0.02*, **
38–40	0.42 ± 0.03	0.40 ± 0.03	0.516 ± 0.020*, **
Side			
Placentation	0.44 ± 0.01	0.42 ± 0.03	0.53 ± 0.02*, **, ***
Nonplacentation	0.42 ± 0.02	0.40 ± 0.02	0.48 ± 0.01*, **

Table 4. Systolic-diastolic ratio in the umbilical artery (M ± m)

Term in weeks	Group		
	Controls, n = 25	Euthyroid goiter (I), n = 76	Subclinical hypothyroidism (II), n = 63
23–25	2.61 ± 0.04	2.60 ± 0.05	2.63 ± 0.04
28–30	2.40 ± 0.02	2.48 ± 0.04	2.50 ± 0.04
35–37	1.76 ± 0.02	2.13 ± 0.05*	2.01 ± 0.04*
38–40	1.90 ± 0.04	2.05 ± 0.04*	2.12 ± 0.05*

Note: * — probability of difference (p < 0.05) compared to the control group.

Table 5. Pulsatile index in the fetal aorta and middle cerebral artery (M ± m)

Indicator	Group		
	Controls, n = 25	Euthyroid goiter (I), n = 76	Subclinical hypothyroidism (II), n = 63
Fetal aorta	2.36 ± 0.05	2.40 ± 0.05	2.42 ± 0.04
Fetal middle cerebral artery	1.62 ± 0.06	1.70 ± 0.03	1.74 ± 0.05

placental blood circulation, as well as the state of the mother's thyroid gland, led to compensatory centralization of fetal blood circulation.

Discussion

Placental abruption, which has incidence of approximately 1 % [15, 16], is the premature separation of the placental lining from before delivery of the fetus [17]. Acute lesions may not show evidence of ischemia; however, older lesions may be accompanied by chorionic villi demonstrating chronic infarctions with degenerated villi and thrombi [12].

Placentas from hypothyroid pregnancies had villous dysmaturity, as observed in the different cases, and decreased vascularization of villi [18].

The uteroplacental interface is susceptible to both thrombosis and hemorrhage, particularly in association with structurally defective placentation. Several factors may mediate such pathogenesis: tissue factor production in response to aberrant vascular endothelial growth factor and inflammatory cytokine release, which promotes thrombosis [13]. In addition, shallow extravillous trophoblast (EVT) invasion may lead to placental ischemia and hemorrhage generating thrombin locally, which mediates the degradation of extracellular matrix, thus triggering premature placental separation [19].

How hypothyroidism might have a role in the pathogenesis of placental abruption remains speculative. Triiodothyronine affects trophoblast behavior, including promotion of EVT invasion [17], so thyroid insufficiency at this critical stage of placental development (11–16 weeks) might result in shallow EVT invasion into maternal decidua and uterine spiral arteries, leading to placental ischemia and abruption [4].

Abnormal thyroid status could influence immune-mediated processes that are critical in placentation as well as other thrombogenic and hemorrhagic events at the uteroplacental interface. Outside the context of pregnancy, higher concentrations of circulating thyroxine within the normal physiological ranges directly enhance innate and adaptive immunity in healthy individuals [6].

Thromboembolism and bleeding tendencies have been associated with hypothyroidism, even in subclinical disease, which could be mediated by defects in the coagulation-fibrinolysis system [7, 13].

In addition to abnormal thyroid function, the described cases also involved high TPO-Ab titers. The presence of TPO-Ab is associated with various non-thyroidal autoimmune diseases and altered immune responses globally [20]. Pregnancy involves a shift in the regulation of cytokine networks within the local placental-decidual environment, and it is conceivable that dysregulation of inflammatory processes in TPO-Ab-positive women is associated with abnormal placentation and adverse pregnancy outcomes.

Conclusions

The study of Doppler blood flow in the mother-placenta-fetus system showed significant disorders of placental maturation with the presence of edema, cystic formations, placental tissue infarctions, a high frequency of its diffuse changes and an increase in the resistance index in the uterine arteries in pregnant women with thyroid disease with the presence of involutional dystrophic changes and circulatory disorders.

These studies make it possible to develop criteria for predicting and diagnosing the development of placental insufficiency in order to prevent perinatal disorders in pregnant women with thyroid diseases.

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Зв'язок функції щитоподібної залози матері з плацентарною гемодинамікою під час вагітності

Резюме. Проведено обстеження 164 вагітних жінок, які були розділені на три групи. До I групи увійшли 76 вагітних (46,4 %) з еутиреоїдним зобом I–II ступеня. Другу групу становили 63 жінки (38,4 %) із субклінічним гіпотиреозом та дифузним зобом I–II ступеня. Третя група була контрольною та складалася з 25 (15,2 %) вагітних без патології щитоподібної залози. Вивчали стан плаценти із характеристикою ультразвукової плацентографії, визначали порушення дозрівання плаценти, площу, локалізацію, виявляли патологічні зміни плацентарної тканини. Оцінювали зміни систолічно-діастолічного співвідношення в маткових артеріях, артеріях пуповини, визначали індекс резистентності в маткових артеріях та пульсаційний індекс в аорті та середній мозковій артерії плода за допомогою методу кольорового доплерівського картування кровотоку в системі «мати — плацента — плід». При вивченні ехографічної картини структурних змін плаценти виявлено вірогідне порушення її дозрівання, особливо в групі з еутиреоїдним зобом. Ультразвуковий скринінг встановив, що в кожної другої вагітної з тиреопатією стан плаценти не відповідав терміну вагітності, відзначалися набряклість, кісти та інфаркти плаценти, висока частота дифузних змін плацентарної тканини, гіперехогенні включення в навколоплідних водах. Звертає на себе увагу підвищення індексу резистентності в маткових артеріях, особливо у вагітних із субклінічним гіпотиреозом. Зі збільшенням терміну гестації периферичний опір мікросудинного русла плаценти підвищується у зв'язку з інволюційно-дистрофічними змінами та циркуляторними розладами, що дозволяє розробити критерії прогнозування та діагностики плацентарної дисфункції, попередити перинатальні порушення у вагітних із тиреопатіями.

Ключові слова: гіпотиреоз; щитоподібна залоза; вагітність; плацентарна дисфункція; доплерометрія; плацентографія