УДК 618.3-06:618.36-008.64
https://doi.org/10.17816/JOWD69423-28

PREDICTION OF PLACENTAL INSUFFICIENCY IN PREGNANT WOMEN WITH DIFFERENT SOMATOTYPES

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For citation: Tomayeva KG. Prediction of placental insufficiency in pregnant women with different somatotypes. *Journal of Obstetrics and Women's Diseases.* 2020;69(4):23-28. https://doi.org/10.17816/JOWD69423-28

Received: June 5, 2020 Revised: July 16, 2020 Accepted: August 10, 2020

▪ ***Hypothesis/aims of study.*** Poor placental vascularization can lead to placental insufficiency, due to which the metabolism of nutrients and microelements between the maternal and fetal blood circulations subsequently decreases. Due to poor perfusion of placental vessels, placental dysfunction occurs. Chronic fetal hypoxia causes fetal growth retardation. The aim of this study was to assess the frequency of placental insufficiency in women with different somatotypes and to develop a model for predicting the risk of this pathology.

Study design, materials and methods. A total of 390 women were examined, of whom 110 were macrosomatic, 173 mesosomatic, and 107 microsomatic. Somatometry was performed according to R.N. Dorokhov for women in the early stages of pregnancy (up to 9-10 weeks). Placental insufficiency markers (VEGF, PlGF, IL-6, and endocan-1) were determined spectrophotometrically in blood serum at the gestational age of 12–13 and 22–23 weeks using ELISA methods.

Results. Placental insufficiency was significantly more prevalent among the women of the macro- and microsomatic body type compared with those of mesosomatotypes ($p < 0.05$). In pregnant women with subsequent placental insufficiency, VEGF and PlGF serum levels at 12–13 weeks were lower, when compared to those in patients who did not develop pathology ($p < 0.05$), and the levels of serum endocan-1 and IL-6 were higher in comparison with those in individuals who did not develop pathology ($p < 0.05$). Using multiple regression analysis, we obtained the regression equation (formula), which predicts the development of placental insufficiency in women of different somatotypes.

Conclusion. The resulting formula allows us to accurately predict the development of placental insufficiency and to form high-risk groups among women for the development of this disease. This will contribute to the effective implementation of therapeutic and preventive measures to avert the development of this pathology.

▪ **Keywords:** pregnancy; placental insufficiency; risk prediction; somatotype.

ПРОГНОЗИРОВАНИЕ ПЛАЦЕНТАРНОЙ НЕДОСТАТОЧНОСТИ У БЕРЕМЕННЫХ С РАЗЛИЧНЫМИ СОМАТОТИПАМИ

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Для цитирования: Томаева К.Г. Прогнозирование плацентарной недостаточности у беременных с различными соматотипами // Журнал акушерства и женских болезней. – 2020. – Т. 69. – № 4. – С. 23–28. https://doi.org/10.17816/JOWD69423-28

Поступила: 05.06.2020 Одобрена: 16.07.2020 Принята: 10.08.2020

▪ ***Актуальность.*** Нарушение процесса васкуляризации плаценты приводит к плацентарной недостаточности, из-за чего впоследствии снижается обмен питательных веществ и микроэлементов между материнским кровообращением и кровообращением плода. Вследствие плохой перфузии сосудов плаценты наступает плацентарная дисфункция. Хроническая гипоксия плода вызывает задержку роста плода.

Цель — изучить частоту встречаемости плацентарной недостаточности у женщин с различными типами телосложения и разработать модель прогнозирования риска возникновения данной патологии.

Материалы и методы исследования. Обследовано 390 женщин, из которых 110 были макросоматического, 173 — мезосоматического, а 107 — микросоматического типа телосложения. Проводили соматотипирование по Р.Н. Дорохову у женщин в ранние сроки беременности (в сроке до 9–10 нед.). В сыворотке крови в сроке

гестации 12–13 и 22–23 нед. определяли маркеры плацентарной недостаточности (VEGF, PlGF, ИЛ-6 и эндокан-1) спектрофотометрически с использованием методов ELISA.

Результаты исследования. Плацентарная недостаточность достоверно чаще встречалась у представительниц макро- и микросоматического типов по сравнению с женщинами с мезосоматотипами ($p < 0,05$). У беременных с развившейся в последующем плацентарной недостаточностью уровень сывороточных VEGF и PlGF в сроке 12–13 нед. был ниже в сравнении с уровнем у женщин, у которых патология не развилась ($p < 0,05$), а уровень сывороточных эндокана-1 и ИЛ-6 был выше в сравнении с уровнем у пациенток, у которых патология не развилась ($p < 0,05$). В ходе множественного регрессионного анализа получено уравнение регрессии (формула), с помощью которого прогнозируют развитие плацентарной недостаточности у женщин разных соматотипов.

Заключение. Выведенная формула позволяет с высокой точностью предсказать развитие плацентарной недостаточности и формировать группы высокого риска по развитию данного заболевания, что будет способствовать эффективной реализации лечебно-профилактических мероприятий по предупреждению развития этой патологии.

▪ **Ключевые слова:** беременность; плацентарная недостаточность; прогнозирование риска; соматотип.

Introduction

Placental insufficiency remains one of the most essential problems in obstetrics, neonatology and perinatology. It is widespread throughout the world and ranges from 17% to 45% from recent studies. An important function of the placenta is in maintaining metabolic processes that are necessary for fetal development. The placenta develops during angiogenesis and vascularization. In case of any disorder in this development, placental dysfunction occurs, representing the main cause of pregnancy complications. Dysregulation of placental angiogenesis is one of the main pathophysiological aspects in the development of placental insufficiency and its clinical consequences [1–5]. Placental insufficiency alters all metabolic processes between mother and fetus, including the processes of amino acid metabolism. Therefore, the growth and development of the fetus deteriorates. The morbidity of newborns associated with intrauterine growth restriction depends on the term of the onset and severity of placental insufficiency, as well as the gestational age at birth. This causes both short-term and long-term consequences for the fetus. Thus, placental insufficiency correlates with a high risk of metabolic diseases in childhood and adulthood, as well as cardiovascular diseases and mortality in adulthood [6–9].

Many papers have been published in which one of the important roles in the emergence and development of pathology of various organs and systems is assigned to the human body type, as well as specific clinical aspects in the course of pathological processes, taking into account the body type, have been noted [10–14]. In recent decades, many scientific works have been conducted in Russia, using classification and methodology of R.N. Dorokhov for somatotyping. When somatotyping according to R.N. Dorokhov, morphometric characters are assessed according to overall component and proportional levels. The body type can be general or individual, and the terms "somatotype" and "body type" are comparable [15, 16].

Placental insufficiency is diagnosed using an obstetrical ultrasound examination (decreased thickness, premature aging of the placenta, and presence of oligohydramnios) and Doppler measurements of the mother-placenta-fetus system [17]. There are little or no studies on markers of placental insufficiency for the prognosis of pathology in women, taking into account the somatotype

Therefore, we aimed at determining the incidence of placental insufficiency in pregnant women with different somatotypes and developing a model for predicting the risk of this pathology.

Materials and methods

We examined 390 pregnant women of which 110 had a macrosomatic body type (MaS), 173 had a meso-somatotype (MeS), and 107 had a microsomatotype (MiS). Somatotyping of women was performed according to the R.N. Dorokhov's method at 9–10 weeks of gestational age [15, 16]. We included pregnant women with no severe somatic morbidity from history, with gestational age no more than 9–10 weeks, singleton pregnancy, and with an informed voluntary consent.

We obtained serum levels of pro-angiogenic factors, such as vascular endothelial growth factor (VEGF), placental growth factor (PlGF),

interleukin-6 (IL-6), and the glycocalyx base protein endocan-1. Blood from the cubital vein was sampled in the morning on an empty stomach into a vacutainer, which contained a coagulation activator and a barrier gel. After blood sampling, the incubation period was 30 min at room temperature (20°C–25°C). Then the samples were centrifuged at 3000 rpm for 10 minutes. Serum markers of placental insufficiency (VEGF, PlGF, IL-6 and endocan-1) were determined spectrophotometrically using ELISA.

We processed and analyzed data using Statgraphics Plus version 5.0 and SPSS version 15.0 software. To assess the significance of the differences in indicators between the groups, we employed the Student's *t*-test, and for relative values, we employed the Pearson's χ^2 test. Multiple regression analysis was performed. Differences in indicators in the groups were considered significant at $p < 0.05$.

Results and discussion

The age of the study population ranged from 18 to 38 years (mean age was 27.5 ± 2.8 years). There were 233 primiparas (59.7%) and 157 (40.3%) multiparas.

Placental insufficiency was often registered in patients with macro- and micro-somatotypes compared to women with meso-somatotypes ($p < 0.05$) (Table 1).

All markers varied significantly across the groups (VEGF, PlGF, endocan-1, IL-6) ($p < 0.05$) (Table 2). Moreover, the indicators at 22–23 weeks of gestation differed significantly from the data obtained at 12–13 weeks of gestation ($p < 0.05$). In pregnant women with developed placental insufficiency at 12–13 weeks of gestation, all indicators differed significantly from those of patients without the pathology. Thus, in pregnant women with placental insufficiency at 12–13 weeks, the levels of serum VEGF and PlGF were lower than the levels in women without the pathology ($p < 0.05$). Conversely, the levels of serum endocan-1 and IL-6 were higher compared to levels in patients without pathology ($p < 0.05$). It should be noted that endocan-1 is a marker of endothelial dysfunction and is released from the glycocalyx when exposed to interleukins. It is also regulated by pro-angiogenic factors, VEGF in particular [18, 19].

We performed correlation and regression analysis using the SPSS program in order to predict placental insufficiency. The analysis revealed the relationship of placental insufficiency with the somatotype of women ($r = -0.77$; $p < 0.05$), the fat component of body weight ($r = 0.84$; $p < 0.05$), the level of serum VEGF at 12–13 weeks of gestation ($r = -0.78$; $p < 0.05$), serum PlGF level at 12–13 weeks ($r = -0.81$; $p < 0.05$), the level of serum endocan-1 at 12–13 weeks ($r = -0.84$; $p < 0.05$), and serum IL-6 level at 12–13 weeks ($r = -0.79$; $p < 0.05$). This confirms the choice of indicators for the prognosis of this pathology. Regarding the significant correlation between placental insufficiency and the levels of VEGF, PlGF, endocan-1 and IL-6 in the blood serum of pregnant women at a term of 12–13 weeks of gestation and the possibility of timely implementation of the necessary preventive measures already in this period, to create a predicting model, indicators were taken in the indicated period of pregnancy. When performing multiple regression analysis, we developed an equation for predicting placental insufficiency in pregnant women of different somatotypes, which included the above indicators due to their conjugation.

Table 1 / Таблица 1

Frequency of placental insufficiency in the examined women
Частота встречаемости плацентарной недостаточности у обследованных женщин

Group	Somatotype					
	MaS (*n* = 110)		MeS (*n* = 173)		MiS (*n* = 107)	
	n	%	*n*	%	*n*	%
Pregnant women without placental insufficiency	90	81.8	158*	91.3	86#	80.4
Pregnant women with placental insufficiency	20	18.2	15*	8.7	21#	19.6

N o t e: * the differences between the types of MaS and MeS are statistically significant ($p < 0.05$); # the differences between the types of MeS and MiS are statistically significant ($p < 0.05$); MaS — macro-somatic type; MeS — meso-somatic type; MiS — micro-somatic type.

Table 2 / Таблица 2

Markers of placental insufficiency in the study groups
Маркеры плацентарной недостаточности в обследованных группах

Group	Indicator	Somatotype of women		
		MaS (*n* = 110)	MeS (*n* = 173)	MiS (*n* = 107)
12–13 weeks of pregnancy				
Pregnant women without placental insufficiency	VEGF, pg/ml	27.9 ± 1.2	36.7 ± 1.3*	29.6 ± 0.8**
	PlGF, pg/ml	241.3 ± 6.5	359.6 ± 4.2*	249.5 ± 5.3**
	Endocan-1, ng/ml	2.2 ± 0.07	0.7 ± 0.03*	2.1 ± 0.04**
	IL-6, pg/ml	1.7 ± 0.05	0.5 ± 0.06*	1.5 ± 0.03**
Pregnant women with subsequent placental insufficiency	VEGF, pg/ml	17.5 ± 2.5$^{\delta}$	23.3 ± 2.1$^{\delta}$	16.4 ± 2.2**$^{\delta}$
	PlGF, pg/ml	166.7 ± 4.6$^{\delta}$	207.2 ± 5.3*$^{\delta}$	158.4 ± 6.8**$^{\delta}$
	Endocan-1, ng/ml	15.7 ± 1.2$^{\delta}$	10.8 ± 0.8*$^{\delta}$	17.2 ± 0.9**$^{\delta}$
	IL-6, pg/ml	7.4 ± 0.4$^{\delta}$	6.8 ± 0.2$^{\delta}$	7.8 ± 0.1**$^{\delta}$
22–23 weeks of pregnancy				
Pregnant women without placental insufficiency	VEGF, pg/ml	25.8 ± 1.6	29.8 ± 1.8*	26.7 ± 1.6
	PlGF, pg/ml	228.4 ± 4.9	309.2 ± 5.4*	231.2 ± 6.3**
	Endocan-1, ng/ml	6.2 ± 0.8	2.1 ± 0.4*	5.6 ± 0.9**
	IL-6, pg/ml	2.7 ± 0.6	1.8 ± 0.3*	2.5 ± 0.6**
Pregnant women with subsequent placental insufficiency	VEGF, pg/ml	13.2 ± 1.2$^{\#\delta}$	19.3 ± 1.1*$^{\#\delta}$	12.6 ± 1.4**$^{\#\delta}$
	PlGF, pg/ml	108.4 ± 6.6$^{\#\delta}$	150.3 ± 6.4*$^{\#\delta}$	103.6 ± 7.6**$^{\#\delta}$
	Endocan-1, ng/ml	26.4 ± 1.2$^{\#\delta}$	18.9 ± 1.1*$^{\#\delta}$	28.1 ± 1.3**$^{\#\delta}$
	IL-6, pg/ml	16.8 ± 0.7$^{\#\delta}$	14.4 ± 0.5$^{\#\delta}$	17.7 ± 0.5**$^{\#\delta}$

N o t e: * the differences between the types of MaS and MeS were statistically significant ($p < 0.05$); ** the differences between the types of MeS and MiS are statistically significant ($p < 0.05$); # the differences between indicators at 12–13 and 22–23 weeks of gestation were statistically significant ($p < 0.05$); $^{\delta}$ differences between indicators in pregnant women without placental insufficiency and in pregnant women with developed placental insufficiency were statistically significant ($p < 0.05$); MaS — macro-somatic type; MeS — meso-somatic type; MiS — micro-somatic type.

$PDPI = -90.2651 - (122.62 \cdot A) + (7.88 \cdot B) + (0.39 \cdot C) - (0.11 \cdot D) - (0.04 \cdot E) + (0.12 \cdot F)$, where PDPI is the probability of developing placental insufficiency (%); A is somatotyping scores; B is the adipose mass of the woman (%); C is serum VEGF level at 12–13 weeks of gestation (pg/ml); D is the blood serum PlGF level at 12–13 weeks of gestation (pg/ml); E is the level of endocan-1 in the blood serum at 12–13 weeks of gestation (ng/ml); F is the level of IL-6 in the blood serum at 12–13 weeks of gestation (pg/ml).

Indicators were substituted into the resulting formula and the occurrence of placental insufficiency in a particular woman was predicted. The risk of a pathological process could be low (up to 30%), moderate (30%–60%) or high (more than 60%) [20].

Examples of calculating the probability of placental insufficiency

Woman L., 22 years old, at 6 weeks of gestation, underwent somatotyping according to the R.N. Dorokhov's method. Anthropometrically, it was revealed that she weighs 44.3 kg; the height was 156.1 cm; the body mass index (BMI) was 18.3 kg/m^2; the adipose mass was 12.46 kg (28.21%); the muscle component of weight amounted to 15.82 kg (35.53%). The patient L. had a micro-somatotype (0.342 points). In the study of serum markers at 12–13 weeks of pregnancy, serum VEGF was 21.3 pg/ml, serum PlGF was 155.1 pg/ml, serum endocan-1 was 13.3 ng/ml, and serum IL-6 was 8.8 pg/ml. According to the above formula, we obtained a PDPI of 84.7%. The pregnancy was complicated by the development of placental insuf-

ficiency in the third trimester of gestation. Thus, the actual results corresponded to the calculated PDPI.

Woman P., 32 years old, at six weeks of gestation, underwent somatotyping according to the R.N. Dorokhov's method. Anthropometrically, it was revealed that she weighs 62 kg; the height was 169 cm; BMI was 21.7 kg/m^2; the adipose mass was 15.58 kg (25.2%); the muscle component of weight amounted to 26.74 kg (43.1%). The patient P. examined had a mesosomatotype (0.523 points). At 12–13 weeks of pregnancy, serum VEGF was 28.7 pg/ml, serum PlGF was 361.2 pg/ml, serum endocan-1 was 2.1 ng/ml, and serum IL-6 was 0.6 pg/ml. From the above formula, we obtained a PDPI of 14.7%. The pregnancy proceeded without placental insufficiency. Thus, the actual data corresponded to the calculated PDPI.

Placenta formation depends on the ratio of pro- and anti-angiogenic factors. Normal placental formation occurs when pro-angiogenic factors predominate, contributing to the rapid and physiological formation of the placenta and the growth of blood vessels. Given that the placenta is a hypervascularized organ, the endothelium of blood vessels occupies a large area and has a regulatory effect on the development.

Conclusion

In women with macro- and microsomatotypes, the risk of placental insufficiency is higher compared to mesosomatotype women. The resulting formula is highly accurate and enables us to predict placental insufficiency in women, taking into account the body type in the trimester I of pregnancy. This helps to form high-risk groups for the development of placental insufficiency among pregnant women registered in the antenatal clinic, and to take timely preventive measures, which will help reduce the frequency of this pathology. The method of calculating the prognosis of the onset of pathology is simple, and it can be performed using a calculator or in Microsoft Excel.

Additional information

Conflict of interest. The authors declare no conflict of interest.

Funding. The study had no external funding

Ethical considerations. The Local Ethical Committee of the North Ossetian State Medical Academy approved our study (protocol No. 5.7 of 08.12.2015).

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