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MORPHOLOGICAL FEATURES OF THE PLACENTA IN PREGNANT WOMEN WITH EXCESSIVE GESTATIONAL WEIGHT GAIN

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▪ ***Study objective.*** To evaluate the association between the placenta and excessive gestational weight gain (GWG). ***Materials and methods.*** A prospective cohort study included the standard ultrasonography with subsequent microscopic morphology of the placenta in term pregnancy. Of 83 examined pregnant women, 46 had excessive GWG and 37 had recommended one. In addition, intensity of lipid infiltration was investigated in 24 placentas (12 in each group). ***Study results.*** Excessive GWG resulted in significant enlargement of placenta which resulted in greater neonatal weight. Ultrasonography and subsequent microscopic evaluation revealed placentas to be less efficient in case of excessive GWG. ***Conclusions.*** These findings indicated that excessive GWG influenced placental morphology. Future studies are necessary to determine accumulation of fat in placentas and membranes in case of excessive GWG, which can be defined as 'fatty degeneration'.

▪ **Keywords:** placenta; morphology; gestational weight gain.

МОРФОЛОГИЧЕСКИЕ ОСОБЕННОСТИ СОСТОЯНИЯ ПЛАЦЕНТЫ У БЕРЕМЕННЫХ С ЧРЕЗМЕРНЫМ ГЕСТАЦИОННЫМ УВЕЛИЧЕНИЕМ МАССЫ ТЕЛА

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▪ ***Цель исследования*** — выявление особенностей структуры плаценты при доношенной беременности при чрезмерном гестационном увеличении массы тела (ГУМТ). Проведены ультразвуковая плацентография при доношенной беременности и морфологическое исследование 83 плацент: 46 — при чрезмерном увеличении веса и 37 — при рекомендуемом. Дополнительно проведена оценка интенсивности жировой инфильтрации 24 плацент, по 12 в каждой группе. ***Результаты.*** Чрезмерное ГУМТ сопровождается формированием большей по размеру плаценты, величина которой коррелирует с массой плода. Ультразвуковая плацентография и последующее морфологическое исследование плаценты подтверждают увеличение риска плацентарных нарушений при чрезмерном ГУМТ. ***Выводы.*** Чрезмерное ГУМТ оказывает влияние на структурные особенности плаценты. Наличие жировой инфильтрации в плацентах в третьем триместре при чрезмерном увеличении веса можно расценивать как жировую дистрофию, последствия которой требуют дальнейшего изучения.

▪ **Ключевые слова:** плацента; морфология; гестационное увеличение веса.

The placenta represents a provisional dimorphic organ that is formed during embryogenesis and fetal development. At the stages of implantation, placentation, and fetalization, it plays an intermediary role between the mother and the fetus. The full development of the fetus involves a complex set of biological processes of active and passive penetrations of nutrients, which characterizes the aspects of the trophic functions of the placenta. At the same time, the level of macronutrients in the fetal blood is largely determined by their concentration in the mother's body. Clinical studies have confirmed that, under the conditions of diabetes mellitus, obesity, and excessive gestational weight gain (GWG) in a pregnant woman, whereby maternal hyperglycemia and hypertriglyceridemia occurs, leading to shifts in the homeostasis of fetuses as in the fetal metabolism, there are chances of subsequent possible development of macrosomia and fetopathy. It is therefore logical that, for the full energy supply to a large fetus, more intensive exchange between the maternal and fetal blood should occur, which can be prompted either by an increase in the permeability of the placenta or by an increase in its area involved in the metabolism. In particular, it has been shown that, with an excessive gravidar weight gain in a pregnant woman, not only the size of the fetus but also the size of the placenta is significantly and relatively larger than in pregnant women with the recommended weight gain [2]. However, even a minor increase in the placental weight in some cases cannot compensate for the requirement of the fetus in macrosomia during the late pregnancy, which in turn can lead to impaired development and even antenatal death [8]. This extremely important possibility was proposed in a model by Ouyang et al. [9], who suggested that an increase in the placental weight by one-third can reduce the possibility of GWG in the newborn.

The placental permeability for lipids and their infiltration of the placenta remain unclear. It is only known that the placenta contains a relatively high amount of lipids (5%–12% of the dry weight of the placenta), although the localization of lipids in the placenta varies. In addition, the role of the placenta in the synthesis of lipids and their transportation from the mother to the fetus, as well as the mechanisms involved in lipid penetration through the placenta, requires a detailed analysis by prospective studies.

In order to identify the characteristics of the structure of the placenta in full-term pregnancy with excessive GWG, we conducted a sonographic and morphological analyses of 83 placentas from 46 pregnant women with excessive weight gain and 37 with the recommended (normal) weight gain.

Materials and methods

The study was conducted by a team of the Department of Obstetrics and Gynecology with a course of prenatal diagnostics of the Smolensk State Medical University in the Obstetric Department of the Emergency Hospital of Smolensk (the Head of the Department, Professor N.K. Nikiforovsky) and the Smolensk Regional Institute of Pathology (Director Professor A.E. Dorosevich). The study groups included primigravidas with a normal baseline body mass index (BMI; 19.5–24.9 kg/m^2) and a pregnancy duration of 37–41 weeks. The average age of the participating women was 24.8 ± 0.7 years. GWG was determined according to the Guidelines for Weight Gain during pregnancy depending on the baseline BMI (IOM, 2009), thus 11.5–16 kg was regarded as the recommended weight gain and >16 kg was regarded as excessive weight gain [10]. The total GWG in the group of pregnant women with excessive weight gain was 16.1–30.5 kg (average: 18.9 ± 0.5 kg). According to the design, with the recommended weight gain, the increase in weight gain was 11.5–16 kg, with the average indicator being 12.9 ± 0.3 kg ($p = 0.000$).

A total of 35 pregnant women with excessive weight gain (76.1%) and 33 women with the normal weight gain (89.2%) had vaginal delivery. Cesarean section was performed in 11 (24.9%) and 4 (10.8%) pregnant women, respectively ($p < 0.05$).

The weight of newborns varied within a rather broad range, namely from 2160 to 4670 g (average: 3546 ± 386 g) with an excessive weight gain and from 2390 to 4300 g (average: 3379 ± 341 g) with the normal weight gain ($p = 0.001$). The difference in the mean values was attributable not only to the more frequent fetal macrosomia with excessive weight gain during pregnancy (5%–10.9%) compared with the normal weight gain (1%–2.7%) but also to the probability of having children weighing ≥3500 g (24–52.1 and 13%–35.1%, respectively). Low birth-weight babies were born in 3 (6.5%) women with excessive weight gain and in 4 (10.8%) women with the normal weight gain. The average score of newborns on the Apgar scale was, respectively, 7.9 ± 0.8 and 8.0 ± 0.2 points at 1 minute and 8.9 ± 0.9 and 8.9 ± 0.5 points at 5 minute ($p > 0.05$). One (0.9%) child was born with severe asphyxia (weakness of labors and vacuum-assisted delivery) in the group with excessive weight gain and 1 (0.9%) child was born with moderate asphyxia (cephalopelvic disproportion and cesarean section) in the group with the normal weight gain. Cerebral ischemia (2%–4.3%) and chronic intrauterine hypoxia (4%–8.7%) were

registered only in newborns born of mothers with excessive weight gain.

The indicators of thickness and sonographic maturity of the placenta at a term of 32–34 weeks were assessed and their correspondence to gestational age, weights of the newborn and placenta, morphological correspondence of the placenta to gestational age, peculiarities of maturation of the villous chorion, involutive-dystrophic changes, manifestations of compensatory-adaptive reactions, infection of the placenta (including infection of the fetal membranes and umbilical cord), and morphological aspects of the fetal membranes and umbilical cord were assessed. Moreover, the intensity of fatty infiltration of the placenta, fetal membranes, and umbilical cord was assessed in 12 afterbirths of women with excessive weight gain and in 12 afterbirths of women with normal weight gain. For morphological study, tissue pieces were incised from the specified biomaterials and fixed in 10% formalin, then histological sections were prepared on a freezing microtome, followed by staining with Sudan III and embedment in glycerol-gelatin. For plain microscopy, the sections of placental tissues after paraffin preparation were stained with hematoxylin and eosin and by van Gieson for connective tissues. Fatty infiltration in the histological preparations was assessed based on density, intensity, and distribution of inclusions (absent (–), low (+) with dusty and small-droplet fatty inclusions; moderate (++) with small and coarse fatty inclusions; intensive (+++) with large-droplet merging fatty inclusions).

Ultrasound placentography was performed at the screening study 3 at the term of 32–34 weeks. The results were evaluated by considering the national guidelines for radiation diagnostics in obstetrics [1].

Statistical processing of the results was performed by the Microsoft Excel 2007 software package for Windows XP. Depending on the purpose of the analysis, we used methods for determining the linear Pearson's correlations, Spearman's rank correlation method, χ^2 test, Fisher's exact test, and Student's *t*-test. In descriptive statistics, mean (*M*) and standard deviation were used. The significance level for statistical analysis was set to $p < 0.05$.

Results and discussion

Ultrasound placentography revealed that an excessive increase in the weight of the placenta correlated with the appearance of echographic signs of premature maturation of the placenta already at weeks 32–34 of gestation ($r = 0.26$, $p = 0.006$). With the recommended weight gain, this phenomenon was noted in 13.9% patients (5 out of 37), while, in cases of excessive weight gain, it was registered in every fourth woman (11 out of 46, 23.9%).

Comparative analysis of the sonographic and histological examinations of the placentas revealed the following characteristics: pathological weight gain was associated with the emergence of morphological signs of placental insufficiency ($r = 0.21$, $p = 0.029$). In 30 out of 87 placentas (34.5%), no pathological changes were noted. Histological examination revealed signs of inflammation in 9 (10.3%) placentas, various morphological variants of placental insufficiency were noted in 20 (23.0%) cases, and a combination of inflammatory and dystrophic changes were noted in 28 (32.2%) cases. Meanwhile, in patients with excessive GWG (29 out of 46, 63.0%), placental insufficiency was registered slightly more often than with the normal GWG (19 out of 37, 51.4%). Probably, the mechanism of these disorders was similar to those identified in the study by Bar et al. [3], who found that periconceptional obesity increases the probability of pathological disorders of the blood flow in the mother-placenta-fetus system by 54%. Simultaneously, adverse abnormalities prevailed in the blood supply to the fetal portion of the placenta and were less pronounced in the maternal one.

We considered the data obtained by comparing the results of morphological examination of the placenta, the weight of the newborn, and the level of gestational increase in body weight to be interesting. We found that the values of placental weight were proportional to the absolute values of gravidar weight gain ($r = 0.26$, $p = 0.027$). Although the trend towards an increase in the size of the placenta with a significant increase in the weight was not statistically significant, nevertheless, it was found to be 475 ± 121 g with the normal weight gain and 492 ± 118 g with excessive weight gain ($p > 0.05$). Similar changes in the weight of the placenta with excessive weight gain in pregnant women have been reported in the literature [1, 9]. The comparison of the ratio of placenta weight, fetal weight, and the size in the groups under study did not differ significantly and amounted to 7.3 ± 1.8 in patients with the normal weight gain and 7.2 ± 1.8 in patients with excessive weight gain ($p > 0.05$). Presumably, we can conclude that the larger the fetus is, greater is the weight of the placenta in order to ensure its favorable development [5, 8]. Moreover, conversely, with a decrease in the size of the placenta, a decrease in the size of the fetus was noted, which could have led to a delay in its intrauterine development and growth [2, 6]. From the point of view of the clinician, this conclusion can be

confirmed by daily practice, as larger the fetus is, larger is the placenta.

We could not find any significant relationship between GWG and the identified histological inflammatory changes in the placenta ($p > 0.05$). Meanwhile, the literature data indicated a rather frequent occurrence of inflammatory lesions of the placenta with the existing disorders of carbohydrate-fat metabolism [4, 7].

We have presented here data on the aspects of histological analyses of the placentas in postpartum women with excessive weight gain, which were identified on assessing the intensity of fatty infiltration of the placenta, fetal membranes, and umbilical cord (see Table).

Table shows that, with pathological weight gain, as a rule, the fetal membranes were predominantly lined with cuboidal amniocytes in a focal multi-row arrangement of the nuclei. On the other hand, with normal weight gain, the epithelium was cubic, with a single-row arrangement of the nuclei in the apical portion.

Lipid infiltration of the trophoblastic and decidual layers of the fetal membranes (Fig. 1) was detected quite often, although the intensity of infiltration with pathological weight gain was visually higher. In 2 cases, intense lipid infiltration (+++) was recorded, which was not noted in cases with normal weight gain.

The accumulation of lipids in the epithelial cells of the fetal membranes during pathological weight gain differed slightly from the accumulation of lipids during normal weight gain and was most often represented by a weak degree (+) in terms of intensity (Figs. 2–6). In 50% of the cases, lipid inclusions in the cytoplasm of the epithelial cells were detected in the form of dusty and small-droplet inclusions, less often (17% of cases) in the form of a moderate degree (++) (Fig. 3), when the lipid inclusions in the epithelial cells

Comparative morphological characteristics of the placenta of pregnant women with recommended and excessive GWG
Сравнительные морфологические характеристики состояния плаценты у беременных с рекомендуемым и чрезмерным гестационным увеличением массы тела

Morphological sign	Excessive GWG $n = 12$		Recommended GWG $n = 12$	
	n	%	*n*	%
Lining of fetal membranes with cuboidal amniocytes with focal multi-row arrangement of nuclei	5	41.7	2	16.7
Lining of fetal membranes with cuboidal amniocytes with single-row arrangement of nuclei in the apical part	7	58.3	10	83.3
Accumulation of lipids in epithelial cells of fetal membranes: low + moderate ++ absent	 6 2 4	 50.0 16.7 33.3	 5 2 5	 41.7 16.7 41.7
Dilation, loosening, hemorrhage in the intermediate layer of the fetal membranes	10	83.3*	0	0
Infection of the fetal membranes	7	58.3	4	33.3
Lipid infiltration of the trophoblastic and decidual layers of the fetal membranes +++ ++ +	 2 1 8	 16.7 8.3 66.7*	 0 6 3	 0 50.0 25.0
Dilation of the umbilical vein	7	58.3*	0	0
Edema of gelatin of Wharton, perivascular hemorrhages in gelatin of Wharton	10	83.3*	3	25.0
Fatty infiltration of the umbilical cord ++ +	 1 7	 8.3 58.3	 1 3	 8.3 25.0

N o t e: GWG — gestational weight gain; * by comparison of groups $p < 0.05$.

П р и м е ч а н и е: ГУМТ — гестационное увеличение массы тела; * при сравнении групп $p < 0,05$.

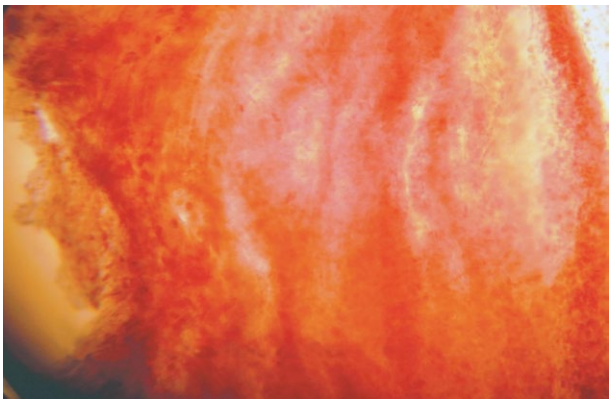

Fig. 1. Intensive fat infiltration (+++) in trophoblastic and decidual layers of the fetal membranes (400× magnification, Sudan III staining)

Рис. 1. Интенсивная жировая инфильтрация (+++) трофобластического и децидуального слоев плодных оболочек. Увеличение ×400, окраска суданом III

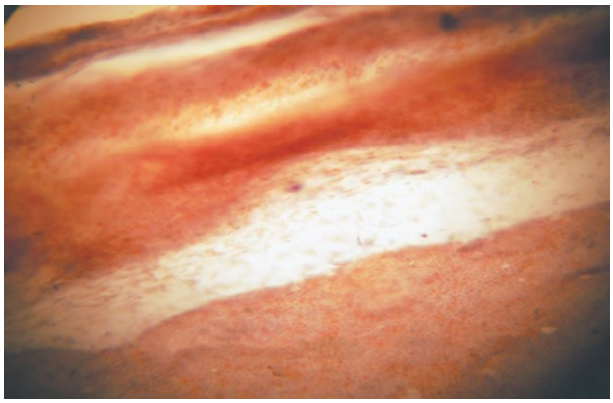

Fig. 2. Low fat infiltration in epithelial cells and decidual layer of membranes (+) (400× magnification, Sudan III staining)

Рис. 2. Слабая жировая инфильтрация эпителиоцитов и децидуального слоя оболочек (+). Увеличение ×400, окраска суданом III

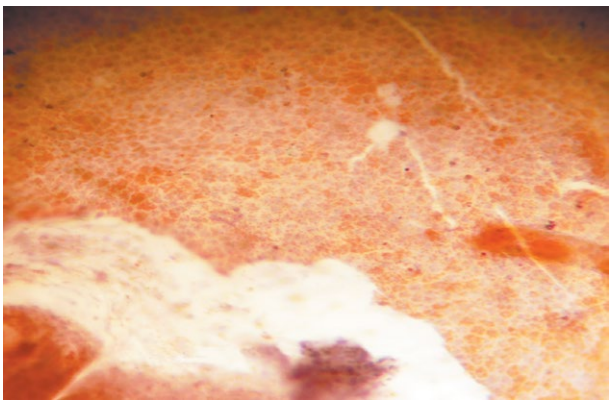

Fig. 3. Moderate fat infiltration (++) in epithelial cells of the membranes (400× magnification, Sudan III staining)

Рис. 3. Умеренная жировая инфильтрация (++) в эпителиоцитах оболочек. Увеличение ×400, окраска суданом III

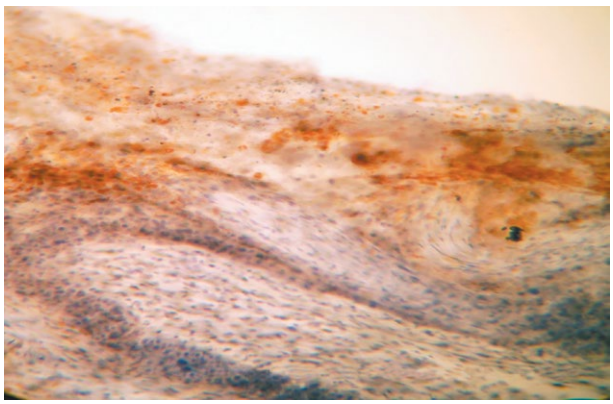

Fig. 4. Low (+) fat infiltration in the decidual layer of the membranes (400× magnification, Sudan III staining)

Рис. 4. Слабая (+) жировая инфильтрация децидуального слоя оболочек. Увеличение ×400, окраска суданом III

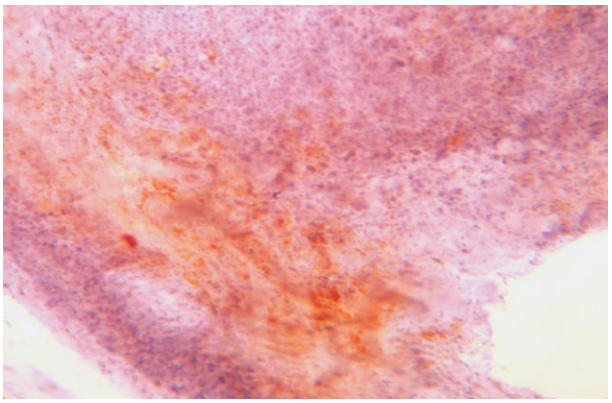

Fig. 5. Low (+) fat infiltration in epithelial cells and decidual layer (100× magnification, Sudan III staining)

Рис. 5. Слабая (+) жировая инфильтрация эпителиоцитов и децидуального слоя. Увеличение ×100, окраска суданом III

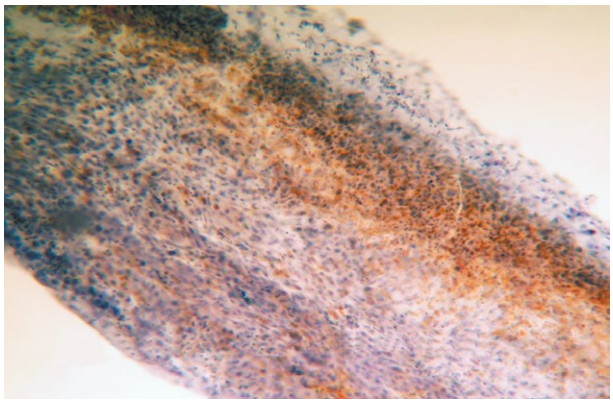

Fig. 6. Low fat infiltration in trophoblastic and decidual layers of the fetal membranes (100× magnification, Sudan III staining)

Рис. 6. Слабая жировая инфильтрация трофобластического и децидуального слоев плодных оболочек. Увеличение ×100, окраска суданом III

were presented as small-droplets. In 33% of the cases, no excess lipid accumulation occurred at all.

As a rule, the intermediate layer of the membranes in the afterbirth of puerperas with pathological weight gain was enlarged, loosened, and comprised hemorrhages. The deposition of lipids in the intermediate layer was not noted either in normal pregnancies or in pregnant women with excessive weight gain.

Infection of the fetal membranes in the pathological course of pregnancy was detected more often in women with pathological weight gain. Therefore, in 7 fetal membranes, parietal deciduitis was detected compared to that in 4 cases with normal weight gain. Infection of all layers of the membranes (membranitis) in pathological weight gain was not detected, and the membranitis was registered in only 1 case with normal weight gain.

The analysis of the results of the histological examination of the umbilical cords showed the most frequent morphological changes in the gelatin of Wharton of the umbilical cords in women with excessive GWG. Edema of gelatin of Wharton, perivascular hemorrhages in the gelatin of Wharton, and varicose veins of the umbilical cord were recorded in 58% of all cases. In our cases, this phenomenon did not occur at all during physiological pregnancy.

Funiculitis or vasculitis of the umbilical cord vessels in puerperas with pathological weight gain were not detected in our cases; however, in one women with normal weight gain, phlebitis of the umbilical cord vein was detected comorbid with membranitis.

In the walls of the umbilical cord vessels, fatty infiltration was much more common in patients with pathological weight gain (low (+) in the vein wall (n = 7), moderate (++) (n = 1), and low (+) in the arterial wall (n = 1). Fatty infiltration in the umbilical cord vessel wall with normal weight gain was more than twice less common and weaker in intensity (+).

In patients with excessive GWG, a visually more frequent impairment of the villous chorion maturation (a variant of the dissociated maturation of the villous chorion) was noted. In all placentas with excessive GWG, an expansion of the intervillous space was revealed, which was not detected in women with normal weight gain. In puerperas with pathological weight gain, vasodilatation of the venous type in the supporting villi was significantly more common.

According to our cases, compensatory-adaptive reactions with an inadequately large GWG were less pronounced than in women with normal weight gain.

Conclusions

With an excessive increase in the body weight of women during pregnancy, there is a tendency of an increase in the size of the placenta, which correlates with the weight of the fetus ($p < 0.05$).

Excessive GWG increases the risk of placental abnormalities, as evidenced by ultrasound placentography and postpartum placental morphology. Morphological changes in the placenta in postpartum women with excessive GWG can be confirmed as impaired maturation of the villous chorion, expansion of the intervillous space, dilation of the venous vessels in the supporting villi, and suppression of the compensatory-adaptive mechanisms. There is a higher lipid infiltration of the trophoblastic and decidual layers of the fetal membranes in these cases. In case of excessive weight gain, in the umbilical cords, morphological changes in the gelatin of Wharton are more often determined in the form of edema, perivascular hemorrhages, and dilation of the veins of the umbilical cord.

In cases of excessive GWG, fatty infiltration of the umbilical cord is seen in the epithelial cells of the umbilical cord, which is not typical for cases with normal weight gain. Fatty infiltration is more common in the walls of the vessels of the umbilical cord. It can thus be stated that, in the placentas of pregnant women with GWG in their third trimester, an impairment of the fat metabolism (i.e., fatty degeneration) can be registered, warranting exploration of the development of mechanisms and consequences in further studies.

The authors declare no conflict of interests.

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