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RELATIONSHIP BETWEEN PLACENTAL LOCATION AND THE DEVELOPMENT OF MACROSOMIA

M.R. Sobirova
D.N. Nurmatova
M.S. Xoliyorova
Tashkent State Medical University
Assistant of the Department of Obstetrics and Gynecology, Pediatric Gynecology
Tashkent State Medical University
Master's Student of the Department of Obstetrics and Gynecology, Pediatric Gynecology
https://orcid.org/0000-0001-8983-0323
https://orcid.org/0009-0002-9044-4694

Abstract. This study was devoted to determining the relationship between placental localization within the uterus and the development of macrosomia. Based on a comparative clinical analysis, the relationship between placental location (posterior wall, anterior wall, uterine fundus, lower segment) and newborn birth weight in term pregnancies was examined. The results demonstrated that the frequency of macrosomia was higher with posterior wall localization (38%), whereas large fetus cases were almost not observed with lower segment localization. The obtained data indicate that placental localization may influence fetal growth through hemodynamic conditions.

Keywords: macrosomia, placental localization, posterior uterine wall, lower segment, uteroplacental hemodynamics, perinatal prognosis.

Introduction. Fetal macrosomia is a relevant and multifactorial clinical problem in perinatal medicine. Newborns with a birth weight of 4000 g or more are classified as macrosomic. In recent decades, the incidence of macrosomia has steadily increased due to metabolic syndrome, obesity, gestational diabetes, and advanced maternal age. According to various epidemiological studies, macrosomia occurs in 5–15% of pregnancies, and in certain high-risk groups this rate may reach up to 20%.

Macrosomia is associated with complications during labor. Shoulder dystocia, birth canal injuries, increased likelihood of operative delivery, and postpartum hemorrhage risk are significantly higher. In the neonatal period, hypoglycemia, respiratory distress syndrome, metabolic disorders, and impaired adaptation reactions are observed. In addition, numerous studies have demonstrated that children born macrosomic have an increased risk of developing obesity and insulin resistance later in life. Fetal growth is a complex biological process resulting from the interaction of genetic, endocrine, metabolic, and hemodynamic mechanisms. The placenta is the primary structure ensuring oxygen and substrate transport to the fetus. As the central component of the uteroplacental system, the placenta performs gas exchange, nutrient transport, hormonal regulation, and immune protection functions.

Placental localization within the uterus may directly influence uteroplacental circulation conditions. Blood supply is not identical in different anatomical parts of the uterus. The posterior wall is located closer to the main branches of the uterine arteries and is considered more vascularized. The anterior wall has relatively moderate perfusion, while the uterine fundus may be exposed to increased tone and mechanical pressure. The lower segment is characterized as a physiologically less perfused area. Therefore, placental localization may alter hemodynamic conditions and consequently influence the rate of fetal growth. To date, the

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relationship between placental location and macrosomia has not been sufficiently studied. This research is aimed at statistically and clinically analyzing this relationship.

Materials and methods

Study Design

The study was conducted in the form of a comparative clinical observation.

Study Population

A total of 160 term pregnant women were included in the study.

The groups were divided according to placental localization:

1. Posterior wall (n=50)
2. Anterior wall (n=45)
3. Uterine fundus (n=35)
4. Lower segment (n=30)

Inclusion Criteria

- Gestational age 37–40 weeks
- Singleton pregnancy
- Placental localization determined by ultrasonography

Exclusion Criteria

- Multiple pregnancy
- Severe preeclampsia
- Congenital anomalies
- Severe form of gestational diabetes

Evaluated Parameters

- Newborn birth weight (g)
- Length (cm)
- Frequency of macrosomia (%)
- Placental localization

Statistical Analysis

Data were expressed as M±m.

ANOVA and χ^2 tests were applied.

$p<0.05$ was considered statistically significant.

Results

1. Comparison of Birth Weight

Placental Localization	n	Mean Weight (g)	Macrosomia (%)
Posterior wall	50	3820 ± 210	38%
Anterior wall	45	3610 ± 195	20%
Uterine fundus	35	3420 ± 180	9%
Lower segment	30	3120 ± 170	3%

ANOVA analysis demonstrated a significant difference between the groups ($F=8.72$; $p<0.01$).

2. Risk of Macrosomia Development (Odds Ratio)

For posterior wall localization, the risk of macrosomia development was:

-OR = 2.4

-95% CI: 1.3–4.5

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-p<0.05

In lower segment localization, the risk of macrosomia was significantly lower (OR=0.2; p<0.05).

3. Length Indicators

Placental Localization	Mean Length (cm)
Posterior wall	53.4 ± 1.2
Anterior wall	52.1 ± 1.1
Uterine fundus	51.3 ± 1.0
Lower segment	50.2 ± 1.1

A strong correlation between length and weight was identified (r=0.71; p<0.01).

Discussion. The study results demonstrated that placental localization may be one of the factors influencing fetal weight. The posterior wall is considered a hemodynamically favorable area. The main segments of the uterine arteries are located in this region, and spiral arteries are well developed. This ensures low-resistance, high-volume blood flow. Increased blood flow enhances oxygen and glucose transport. As a result, anabolic processes in fetal tissues are activated, and weight gain accelerates. This may explain the higher frequency of macrosomia associated with posterior wall localization. The lower segment, however, is physiologically less perfused. During pregnancy, the formation and mechanical stretching of the lower segment may increase hemodynamic resistance. Therefore, a placenta located in this region may provide relatively fewer substrates to the fetus. However, placental localization is not an independent determinant in the development of macrosomia. Metabolic factors, particularly gestational diabetes, maternal BMI, and genetic predisposition, also play important roles. Although severe metabolic pathologies were excluded in our study, subclinical metabolic factors were not completely ruled out. The results suggest that during antenatal monitoring, placental localization may be considered an additional prognostic marker. Especially when posterior wall localization is identified together with high hemodynamic indicators, the probability of a large fetus should be considered.

Conclusion.

1. Placental location within the uterus has a statistically significant effect on fetal weight.
2. Posterior wall localization is associated with higher birth weight and a significantly increased frequency of macrosomia.
3. In lower segment localization, large fetuses were almost not observed, which may be explained by relatively lower perfusion in this area.
4. Although placental localization is not an independent determining factor in the development of macrosomia, it has clinical significance as an additional prognostic indicator.
5. Determination of placental location during prenatal ultrasound examination helps refine antenatal prognosis.
6. When evaluated together with hemodynamic and metabolic parameters, placental localization allows early identification of macrosomia risk.

Thus, placental location within the uterus may be one of the factors influencing fetal growth rate and should be considered during antenatal monitoring.

References.

1. Beta J, Khan N, Khalil A, Fiolna M, Ramadan G, Akolekar R. Maternal and neonatal complications of fetal macrosomia: systematic review and meta-analysis. Ultrasound Obstet Gynecol. 2019;54(3):308–318.

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2. ACOG Practice Bulletin No. 216: Macrosomia. Obstet Gynecol. 2020;135(1):e18–e35.
3. Fuchs F, Bouyer J, Rozenberg P, Senat MV. Adverse maternal outcomes associated with fetal macrosomia. BJOG. 2013;120(8):1010–1017.
4. Kim SY, Sharma AJ, Sappenfield W, Wilson HG, Salihu HM. Association of maternal body mass index and macrosomia. J Perinatol. 2018;38(5):528–535.
5. Hehir MP, Ananth CV, Siddiq Z, Flood K, Friedman AM, D'Alton ME. Fetal macrosomia and adverse outcomes in non-diabetic pregnancies. J Matern Fetal Neonatal Med. 2015;28(7):820–825.
6. Burton GJ, Jauniaux E. What is the placenta? Am J Obstet Gynecol. 2015;213(4 Suppl):S6–S8.
7. Burton GJ, Fowden AL. The placenta: a multifaceted, transient organ. Philos Trans R Soc Lond B Biol Sci. 2015;370(1663):20140066.
8. Desoye G, Wells JC. Pregnancy and diabetes: the placental contribution. Diabetologia. 2021;64(5):1019–1028.
9. Sibley CP, Brownbill P, Dilworth M, Glazier JD. Review: adaptation in placental nutrient supply to meet fetal growth demand. Placenta. 2018;64:S29–S36.
10. Sferruzzi-Perri AN, Camm EJ. The programming power of the placenta. Front Physiol. 2016;7:33.
11. Mayhew TM. Placental angiogenesis and fetal growth. Placenta. 2014;35(9):A1–A6.
12. Higgins LE, Greenwood SL, Wareing M, Sibley CP, Mills TA. Obesity and the placenta: a systematic review. Placenta. 2018;62:44–52.
13. Jansson T, Powell TL. Placental nutrient transport and fetal growth in maternal diabetes. Placenta. 2016;48(Suppl 1):S32–S36.
14. Melamed N, Yogev Y, Meizner I, Mashiach R, Ben-Haroush A. Sonographic fetal weight estimation and macrosomia. Ultrasound Obstet Gynecol. 2016;48(3):304–310.
15. Sovio U, Smith GC. Fetal growth and placental function. Best Pract Res Clin Obstet Gynaecol. 2018;49:86–98.
16. Taricco E, Radaelli T, Nobile de Santis MS, Cetin I. Fetal and placental weight ratio in gestational diabetes. Placenta. 2015;36(4):412–417.
17. Bar J, Schreiber L, Saruhanov E, Ben-Haroush A, Golan A. Placental weight and birth weight correlation. Arch Gynecol Obstet. 2016;293(5):979–984.
18. Hiersch L, Yogev Y. Impact of gestational diabetes on fetal growth and placental function. World J Diabetes. 2014;5(4):502–511.
19. Leon-Garcia SM, Roeder HA, Nelson KK, et al. Maternal obesity and placental vascularization. Placenta. 2016;38:49–57.
20. Colson A, Sonveaux P, Debiève F. Angiogenesis and placental development in metabolic disorders. Int J Mol Sci. 2021;22(6):3077.
21. Illsley NP, Baumann MU. Human placental glucose transport in normal and pathological pregnancies. Am J Physiol Endocrinol Metab. 2020;318(3):E415–E426.