Прилози, Одд. биол. мед. науки, МАНУ, XXX, 2, с. 115–124 (2009)
Contributions, Sec. Biol. Med. Sci., MASA, XXX, 2, p. 115–124 (2009)
ISSN 0351–3254
УДК: 616.379-008.64:618.3

GESTATIONAL *DIABETES MELLITUS* – THE IMPACT OF MATERNAL BODY MASS INDEX AND GLYCAEMIC CONTROL ON BABY'S BIRTH WEIGHT

Krstevska B.,[1] Mishevska S.,[1] Janevska E.,[1] Simeonova S.,[2] Livrinova V.,[2] Pemovska G.,[1] Velkoska Nakova V.,[1] Serafimoski V.[3,4]

[1]Endocrinology, Diabetes and Metabolic Discorders Clinic, Medical Faculty, Skopje, Macedonia
[2]Gynecology and Obstetric Clinic, Medical Faculty, Skopje, R. Macedonia
[3]Gastroenterohepatology Clinic, Medical Faculty, Skopje, R. Macedonia
[4]Macedonian Academy of Sciences and Arts, Skopje, R. Macedonia

Abstract: Objectives. To asses the influence of the maternal BMI and glycaemic control in women with GDM on the baby's birth weight (BW).

Material and methods: We analysed 180 women with GDM. Macrosomia has been defined as BW > 4000 gm, small for gestational age < 2700 gm and appropriate for gestational age between both. According to the baby´s BW the pregnant women were divided into three groups: group 1 (G1) with BW < 2700 gm (n = 26); group 2 (G2) with BW between 2700 to 4000 gm (n = 117), and group 3 (G3) with BW > 4000 gm (n = 37). We also analysed BMI (kg/m²), HbA1c (%), PPG (mmol/L) and time of delivery (WG).

Results: Comparisons between G1 and G2 showed: BMI (30.7 ± 5 & 31 ± 5.2; $p < 0.7$), HbA1c (6.4 ± 0.8 & 5.1 ± 0.8, $p < 0.002$), PPG (8.2 ± 1.7 & 6.9 ± 1.5, $p < 0.02$), time of delivery (35.2 ± 3.8 & 38.6 ± 1.5, $p < 0.0001$) and BW (2289 ± 504 & 3474 ± 334, $p < 0.0001$). Comparisons between G2 and G3 showed: BMI (31 ± 5. 2 & 33.4 ± 6.1; $p < 0.02$), HbA1c (5.2 ± 1.1 & 6.4 ± 2.3, $p < 0.02$), PPG (6.9 ± 1.5 & 8.2 ± 1.9, $p < 0.02$), time of delivery (38.6 ± 1.5 & 39.3 ± 1.4, $p < 0.01$) and BW (3474 ± 334 & 4431 ± 302, $p < 0.0001$). Comparisons between G1 and G3 showed the difference at delivery time and the baby's BW ($p < 0.0001$).

Conclusions: Maternal obesity and PPG contribute to macrosomia and also PPG to SGE.

Key words: gestational diabetes, large for gestational age, small for gestational age, birth weight, postprandial glycaemia.

Introduction

Approximately 5% of all pregnancies are complicated by gestational *diabetes mellitus* (GDM), which increases both maternal and perinatal morbidity [1]. GDM accounts for 90% of cases of *diabetes mellitus* in pregnancy [2]. GDM is defined as a carbohydrate intolerance that begins or is first diagnosed during pregnancy [3]. Maternal supply of carbohydrates leading to foetal hyperglycaemia, which in turn stimulates pancreatic islet cells and causes hyperinsulinaemia [4]. Stimulation of the insulin-sensitive tissue results in increased foetal growth, predominantly of the abdomen, and delivery of large for gestational age (LGA) newborns [5]. Women with large foetuses are at a higher risk of complications of delivery such as infection, caesarean section, pre-eclampsia and perinatal mortality [6]. For the infants the risks of immediate complications are increased, including intracranial haemorrhage, shoulder dystocia, neonatal hypoglycaemia, jaundice, and respiratory distress. The offspring of mothers with GDM have high risks of insulin resistance, obesity and type 2 diabetes later in life [7].

In treating women with this condition, many have advocated minimizing fluctuations in blood glucose concentrations to avert maternal hyperglycaemia and thus decrease the risk of foetal hyperglycemia and its consequences, foetal hyperinsulinaemia and excess foetal growth [8]. However, despite early diagnosis and aggressive dietary and insulin therapy, perinatal morbidity among infants born to women with GDM remains excessive, a fact that may or may not be attributed to suboptimal glycaemic control [9]. In the management of GDM, various methods of glucose monitoring have been proposed, including the measurement of fasting, preprandial, postprandial, and mean 24-hour blood glucose concentrations [1]. Excess nutrient delivery to the foetus causes macrosomia, but whether fasting or peak glucose values are more correlated with foetal overgrowth is less clear [2].

Evidence for an association between maternal HbA1c and the risk of foetal macrosomia is conflicting [10]. Evers *et al*. reported [11] that variations in HbA1c levels during pregnancy explained less than 5% of the variation in birth weight (BW).

Some studies attribute the greater risk of macrosomia to maternal glucose intolerance. Others have reported a stronger relationship between BW and maternal pre-pregnancy weight or weight at delivery, than between BW and measures of maternal glycaemia. Recently it was shown that maternal glycaemia during third trimester and pre-pregnancy body mass index (BMI) are independent predictors of BW in pregnancies complicated by GDA, and that obesity exerts a significant influence on the risk of delivery of LGA infants [12]. On the other hand, in one prospective study of 2,272 women with GDM, only maternal BMI predicted neonatal BW, while plasma glucose levels did not [13].

Women who have had GDM have many features of the metabolic syndrome and are at very high risk of developing type 2 diabetes. Epidemiological observations have suggested a relationship between type 2 diabetes and a low BW [13]. Whether glycaemic control in women with GDM predisposes to LGA, small for gestational age (SGA), or both, is not clear.

The aim of the research was to asses the influence of the maternal BMI and glycaemic control in women with GDM on the baby's BW.

Material and methods

We conducted this cohort study including all consecutive women who attended the Outpatient Department of Endocrinology, Diabetes and Metabolic disorders in a period from 02.2006 to 02.2009 with singleton pregnancy. The diagnosis is established by glucose tolerance testing.

According to the guidelines of the ADA [14] we used the 2 steps system. A glucose challenge test (GCT) was performed between 24th and 28th week of gestation in high and middle risk groups. In GCT, the pregnant women underwent a standard 50-g glucose load and a 1-h plasma glucose concentration was measured. A plasma glucose ≥ 7.8 mmol/l was considered positive according to these recommendations [14]. Those whose initial screening test was abnormal went on to complete a diagnostic 3-h 100-g oral glucose tolerance test (OGTT). After an overnight fast and at least 3 days of unrestricted diet (≥ 150 g carbohydrate per day) and unlimited physical activity, blood was taken to determine plasma glucose levels. Capillary blood glucose levels were measured by glucose oxidase (Glucose Analyzer; Beckman, Brea, CA). The diagnosis was made by Carpenter and Coustan's criteria [15]. Women with GDM were educated regarding an individualized diabetic diet based on pre-pregnancy weight (30 kcal · kg−1 · day−1) with caloric restriction for obese women (25 kcal · kg−1 · day−1). All women were asked to perform a daily glucose profile (fasting, preprandial and 1-h postprandial measurements) twice a week using a reflectance with electronic memory (OneTouch Basic 200–200; LifeScan, Milpitas, California, USA). Target glucose levels were: fasting glycaemia between 4.0 to 5.5 mmol/l and 1-hrs postprandial glycaemia > 7.8 mmol/l [16]. Adjustments in the insulin doses were made if any of the values were consistently higher than the target blood glucose concentrations.

Further inclusion criteria were: live birth and no foetal malformation suspected during gestation or detected postpartum. All patients gave informed consent to participate in the study.

In our research, we investigated the following parameters: patients' age, pre-pregnancy BMI, weight gain during pregnancy, gestational age when GDM was diagnosed, arterial blood pressure, fasting and postprandial glycaemia at first, second and third trimester, haemoglobin A1c (HbA1c) at first, second and third trimester, gestational age at delivery, BW and a baby capillary blood sugar level.

The BMI of women with GDM was calculated by dividing the weight by the height squared (kilograms/metres2). The patients were weighed wearing clothes without shoes in the morning with an electronic scale at the first visit. Height was measured to the nearest 1 cm with a stadiometer. The gestational age was estimated from the date of the last menstrual period. Blood pressure was measured twice in a supine position. In a case of hypertension (> 145/90 mmHg) the measurement was repeated after five minutes. Fasting and postprandial plasma glucose were measured twice a week on alternate weeks from the diagnostic moment of GDM until delivery. The blood glucose profiles performed during the entire pregnancy were averaged to calculate a mean blood glucose level. Blood samples for HbA1c were taken after overnight fasting. HbA1c was measured by an ion-exchange HPLC instrument (DS5; Drew, USA) with a reference range of 4.2–6%.

the baby's birth weights were classified as LGA, appropriate for gestational age (AGA) and SGA. LGA we defined as birth weight above 4000 g, SGA below 2700g, and AGA between the two. According to the baby's weight the women were further categorized into three groups: group 1 (G1) with BW below 2700g, group 2 (G2) with BW between 2700 to 4000g, and group 3 (G3) with BW above 4000g.

Statistical analysis

Statistical analysis was performed using the Statistics for Windows programme, version 5,0. Dates are given as mean ± standard deviation. We used t-test for independent samples to compare the variables between each of the groups. $P < 0.05$ was considered statistically significant.

Results

There were 180 women in the total sample. Basic characteristics of the patients enrolled into the study are given in Table1. Overall, 20.6% of the pregnancies resulted in birth of a LGA baby, while 14% were SGA.

Table 1 – Табела 1

Characteristics of the study group
Карактеристики на испитуваната група

Variable	Valid N	Mean	Minimum	Maximum	Std. Dev.
Age	180	31.55	18	47	5.51
Gestational age at diagnosis	178	27.3	6	40	7.68
Pre-pregnancy BMI (kg/m2)	168	26.89	16	45	5.27
Weight gain (kg/m2)	165	31.45	20	49	5.49
HbA1c (first trimester) %	16	7.113	4.3	11	2.15
HbA1c (second trimester) %	50	5.83	3.85	9.1	0.99
HbA1c (third trimester) %	117	5.87	4.3	10.0	1.07
SBP (mmHg)	144	120	90	180	15.79
DBP (mmHg)	144	76	50	120	10.63
FPG 1 (mmol/l)	13	7.388	3.7	12.5	2.36
PPG 1 (mmol/l)	13	9.532	5.17	15.4	2.74
FPG 2 (mmol/l)	64	5.527	3.3	12.9	1.49
PPG 2 (mmol/l)	65	7.466	4.37	11.5	1.76
FPG 3 (mmol/l)	167	5.338	3.07	10.75	1.16
PPG 3 (mmol/l)	166	7.385	2.9	12.7	1.91
Gestational age at delivery	180	38.264	25	42	2.39
Birth weight (g)	180	3500	700	5410	720.54
Baby's glycaemia (mmol/l)	89	3.012	0.7	7.4	1.24

BMI – body mass index, SBP – systolic blood pressure, DBP – diastolic blood pressure, FPG 1, 2, 3 – fasting plasma glucose value in first, second and third trimester, PPG 1, 2, 3 – postprandial glucose value in first, second and third trimester.
БМИ – индекс на телесна маса, СТА – систолен крвен притисок, ДТА – дијастолен крвен притисок, ФПГ 1, 2, 3 – гликемии пред оброк во прв, втор и трет триместар, ППГ 1, 2, 3 – гликемии по оброк во прв, втор и трет триместар.

Comparisons between G1 and G2 [BW (2289 ± 504 and 3474 ± 334, $p < 0.0001$)] showed statistical significant differences between the following parameters: HbA1c, PPG, and time of delivery (See, Table 2). HbA1c in the second trimester was higher in women who delivered SGA than in women who delivered AGA (6.4 ± 0.8 and 5.1 ± 0.8, $p < 0.002$). PPG in second (8.2 ± 1.7 and 6.9 ± 1.5, $p < 0.02$) and third trimester (8.3 ± 2.2 and 7.3 ± 1.8, $p < 0.02$)

was higher in women who delivered SGA than in women who delivered AGA, and time of delivery was shorter in women who delivered SGA than in women who delivered AGA (35.2 ± 3.8 and 38.6 ± 1.5, $p < 0.0001$). Mothers who delivered SGA and AGA did not have statistically different values for FPG in any trimester.

Table 2 – Табела 2

Comparisons between G1:G2, G2:G3, G1:G3
Сйоредба йомеѓу Г1 и Г2, Г2 и Г3, Г1 и Г3

Variable	Mean G1 ± SD	Mean G2 ± SD	Mean G3 ± SD	p (1 : 2)	p (2 : 3)	p (1 : 3)
Age	31.92 ± 5.1	31.55 ± 5.5	31.29 ± 5.6	0.75	0.81	0.65
Gestational age at diagnosis	24.4 ± 7.1	27.7 ± 7.4	28.1 ± 8.4	**0.04**	0.78	0.07
Prepregnancy BMI (kg/m2)	27.5 ± 5.8	26.36 ± 4.8	28.2 ± 6.2	0.31	**0.049**	0.66
Weight gain (kg/m2)	30.7 ± 5.02	32 ± 5.3	33.4 ± 6.1	0.79	0.06	0.07
HbA1c (first trimester) %	7.5 ± 3.3	7.2 ± 2.3	6.7 ± 1.4	0.87	0.68	0.65
HbA1c (second trimester) %	6,5 ± 0.8	5.5 ± 0 .8	6.1 ± 1.3	**0.002**	0.08	0.46
HbA1c (third trimester) %	6,03 ± 0.9	5.7 ± 1.03	6.23 ± 1.2	0.31	**0.04**	0.54
SBP (mmHg)	123.9 ± 13.2	117.7 ± 16.7	121.16 ± 14.1	0.1	0.31	0.47
DBP (mmHg)	79.56 ± 9.2	75.7 ± 10.8	77.3 ± 10.7	0.12	0.48	0.43
FPG 1 (mmol/l	8.7 ± 3.8	6.8 ± 1.9	7.4 ± 1.7	0.29	0.64	0.6
PPG 1(mmol/l	12.11 ± 3.03	9.1 ± 2.6	7.9 ± 1.06	0.14	0.48	0.08
FPG 2(mmol/l	5.6 ± 1.1	5.2 ± 1.2	5.8 ± 2.3	0.35	0.06	0.26
PPG 2(mmol/l	8.2 ± 1.8	6.99 ± 1.5	8.2 ± 1.9	**0.02**	**0.02**	0.99
FPG 3(mmol/l	5.4 ± 0.6	5.2 ± 1.2	5.6 ± 1.3	0.57	0.13	0.49
PPG 3(mmol/l	8.3 ± 2.2	7.3 ± 1.8	7.2 ± 1.8	**0.02**	0.91	0.06
Gestational age at delivery	35.2 ± 3.8	38.6 ± 1.6	39.3 ± 1.4	**0.000**	**0.01**	**0.000**
Birth weight (g)	2289 ± 504	3474 ± 334	4431 ± 302	**0.000**	**0.000**	**0.000**
Baby's glycaemia	2.8 ± 1.2	3.2 ± 1.3	2.5 ± 0.8	0.34	**0.02**	0.4

Comparisons between G2 and G3 [BW (3474 ± 334 and 4431 ± 302, $p < 0.0001$)] showed statistically significant differences between the following parameters: pre-pregnancy BMI, HbA1c, PPG, newborn glycaemia, and time of delivery (See Table 2). Women who delivered LGA have a higher BMI than women with AGA (33.4 ± 6.1and 31 ± 5.2, $p < 0.05$), HbA1c in third trimester is higher in women who delivered LGA than AGA (6.4 ± 2.3 and 5.2 ± 1.1, p <

0.02), PPG in second trimester is higher in women who delivered LGA than AGA (8.2 ± 1.9 and 6.9 ± 1.5, $p < 0.02$), LGA newborns had lower glycaemia than AGA newborns (2.5 ± 0.8 and 3.2 ± 1.3, $p < 0.02$), and time of delivery was longer in women who delivered LGA, but still in the normal term (39.3 ± 1.4 and 38.6 ± 1.5, $p < 0{,}01$).

Comparisons between G1 and G3 [BW (2289 ± 504 and 4431 ± 302, $p < 0.0001$)] showed statistically significant differences only between delivery time (35.2 ± 3.8 and 39.3 ± 1.4, $p < 0.0001$) (See Table 2).

Women with higher BMI are at increased risk of delivering LGA infants. Also women who delivered SGA or LGA had higher HbA1c during the second or third trimester, respectively. FPG is not associated with SGA or LGA infants, but PPG in the second and third trimester increased the risk of delivering an SGA or LGA infant.

Discussion

The results of the study showed that women with higher BMI are at increased risk of delivering an LGA infant and PPG contribute to LGA or SGA in mothers with GDM. In one study, blood glucose monitoring before meals in women with insulin-dependent *diabetes mellitus* did not provide an adequate indication of metabolic control or of the risk of macrosomia; the authors therefore recommended postprandial glucose monitoring in order to optimize glycaemic control [17]. In another study, macrosomia was related to postprandial but not to fasting blood glucose values [18], as in our study. Langer *et al*. [8] showed that concentrations of both fasting and postprandial serum glucose were directly related to BW. We found a more stringent influence of PPG on BW, LGA but also on SGA. Jovanovic-Peterson *et al*. [17] reported that among pre-gestational diabetic women an increase in the third trimester PPG glucose was a positive predictor of BW. Our results show that this relationship also exists among women diagnosed with GDM. Many of the physicians believed that pre-prandial glucose monitoring was as effective as PPG monitoring [1] but we showed that only PPG during treatment has an influence on BW in women with GDM developing LGA or SGA infants.

Other studies have found that pre-pregnancy BMI and weight gain during pregnancy are strongly associated with LGA [5, 10, 12, and 13]. In our study we found that only pre-pregnancy BMI contributed to LGA. Essentially, moderate dietary restriction before conception can decrease BMI and its influence on BW.

Aproximately 15–25% of neonates delivered from women with GDM develop hypoglycaemia during the immediate newborn period. Neonatal hypo-

glycaemia is less frequent when tight glycaemic control is maintained during pregnancy [2]. In our study the prevalence of hypoglycaemia was 13.3%. LGA newborns had lower glycaemia then AGA newborns, probably because of the higher mother glycaemia which contributea to hyperinsulinaemia in the newborn.

There were no differences in HbA1c between the groups in the first trimester, but we had a small number of women who enrolled to the study in the first trimester. Mothers with SGA and LGA newborns had higher HbA1c than mothers with AGA, although mean HbA1c was below 7%, (6.7%, 6.3%, and 6.1%, respectively). The latter showed good glycaemic control during pregnancy in the study group.

Time of delivery was significantly different between the three study groups. SGA was delivered preterm (mean 35.2 ± 3.8 gestation weeks), which is probably responsible for the high percent of SGA (14%) in our study group. Surprisingly, LGA were delivered in normal term, but statistically significant later than AGA.

Conclusion

Maternal obesity and PPG contribute to macrosomia, and also PPG to SGA. Monitoring of PPG levels rather than fasting levels are necessary to prevent SGA and LGA babies.

REFERENCES

1. De Veciana M., Ajor AM., Morgan AM. *et al*. (1995): Postprandial versus preprandial blood glucose monitoring in women with gestational diabetes mellitus requiring insulin therapy. *N Engl J Med;* 333: 1237–1241.

2. http://emedicine.medscape.com/article/127547-overview (Accessed 10.07.2009).

3. American College of Obstetricians and Gynecologists: ACOG Practice Bulletin: clinical management guidelines for obstetricians- gynecologists: number 30, September 2001: gestational diabetes. *Obstet Gynecol;* 98: 525–538.

4. Evagelidou EN., Kiortsis DN., Bairaktari ET., *et al*. (2006): Lipid profile, glucose homeostasis, blood pressure, and obesity-anthropometric markers in macrosomic offspring of nondiabetic mothers. *Diabetes Care;* 29: 1197–1201.

5. Snyder J., Gray-Donald K., Koski KG. (1994): Predictors of infant birth weight in gestational diabetes. *Am J Clin Nutr;* 59: 1509–1514.

6. Kabali C., Werler MM. (2007): Prepregnant body mass index, weight gain and the risk of delivering large babies among non-diabetic mothers. *Int J Gynaecol Obstet;* 97(2): 100–104.

7. Murphy HR., Rayman G., Lewis K. (2008): Effectiveness of continuous glucose monitoring in pregnant women with diabetes: randomised clinical trial. *BMJ;* 337: a1680.

8. Langer O., Mazze R. (1988): The relationship between large-for-gestational-age infants and glycemic control in women with gestational diabetes. *Am J Obstet Gynecol;* 159: 1478–1483.

9. Metzger BE. (1991): Summary and recommendations of the Third International Workshop-Conference on Gestational Diabetes Mellitus. *Diabetes;* 40(2): 197–201.

10. Nielsen GL., Dethlefsen C., Moller M., Sorensen HT. (2007): Maternal glycosylated haemoglobin, pre-gestational weight, pregnancy weight gain and risk of large for gestational age babies: a Danish cohort study of 209 singleton type 1 diabetic pregnancies. *Diabet Med;* 24(4): 384–7.

11. Evers IM., de Valk HW., Visser GH. (2004): Risk of complications of pregnancy in women with type 1 diabetes: nationwide prospective study in the Netherlands. *BMJ;* 328(7445): 915.

12. Ben-Haroush A., Hadar E., Chen R., Hod M., Yogev Y. (2009): Maternal obesity is a major risk factor for large-for-gestational-infants in pregnancies complicated by gestational diabetes. *Arch Gynecol Obstet;* 279: 539–543.

13. Ray JG., Vermeulen MJ., Shapiro JL., Kenshole AB. (2001): Maternal and neonatal outcomes in pregestational and gestational diabetes mellitus, and the influence of maternal obesity and weight gain: the DEPOSIT – study. *Q J Med;* 94: 347–356.

14. American Diabetes Association. Standards of medical care in diabetes-2007. (2007): *Diabetes Care;* 30 Suppl 1: S4–S41.

15. Roggenbruck L., Kleinwechter HJ., Demandt N., Dörner KM. (2004): Diagnostics of gestational diabetes: which cutoff-values are valid for capillary whole blood? *Clin Lab;* 50: 403–408.

16. Metzger BE, Buchanan TA, Coustan DR, de Leiva A, Dunger DB. (2007): Summary and Recommendations of the Fifth International Workshop-Conference on Gestational Diabetes Mellitus. *Diabetes Care;* 30: S251–S260.

17. Jovanovic-Peterson L., Peterson CM., Reed GF. *et al*. (1991): Maternal postprandial glucose levels and infant birth weight: the Diabetes in Early Pregnancy Study. The National Institute of Child Health and Human Development--Diabetes in Early Pregnancy Study. *Am J Obstet Gynecol;* 164(1 Pt 1): 103–111.

18. Combs CA., Gunderson E., Kitzmiller JL., Gavin LA., Main EK. (1992): Relationship of fetal macrosomia to maternal postprandial glucose control during pregnancy. *Diabetes Care;* 15: 1251–1257.

Р е з и м е

ГЕСТАЦИСКИ ДИЈАБЕТЕС – ВЛИЈАНИЕ НА ИНДЕКСОТ НА ТЕЛЕСНА ТЕЖИНА И ГЛИКЕМИСКА КОНТРОЛА НА МАЈКАТА ВРЗ РОДИЛНАТА ТЕЖИНА НА БЕБЕТО

Крстевска Б.,[1] Мишевска С.,[1] Јаневска Е.,[1] Симеонова С.,[2] Ливринова В.,[2] Пемовска Г.,[1] Велкоска Накова В.,[1] Серафимоски В.[3,4]

[1]Клиника за ендокринологија, дијабетес и болести на метаболизмот, Медицински факултет, Скопје, Р. Македонија
[2]Клиника за гинекологија и акушерство, Медицински факултет, Скопје, Р. Македонија
[3]Клиника за гастроентерохепатологија, Медицински факултет, Скопје, Р. Македонија
[4]Македонска академија на науките и уметностите, Скопје, Р. Македонија

Цел: Да се оцени влијанието на индексот на телесна тежина (БМИ) и гликемиската контрола кај мајки со гестациски дијабетес (ГДМ) врз родилната тежина на новороденчињата (РТ).

Материјали и методи: Анализиравме 180 жени со ГДМ. Макросомијата беше дефинирана како РТ > 4000 гр, мала телесна тежина за гестациска возраст (СГА) < 2700 гр и соодветна за гестациската возраст (АГА) помеѓу двете. Според родилната тежина бремените жени беа поделени на три групи: група 1 (Г1) со РТ < 2700 гр (n = 26); група 2 (Г2) со РТ од 2700 до 4000 гр (n = 117) и група 3 (Г3) со РТ > 4000 гр (n = 37). Анализиравме БМИ, гликолизиран хемоглобин, гликемија пред (ФПГ) и по оброк (ППГ) и гестациска недела на породување (ГН).

Резултати: Споредбата помеѓу Г1 и Г2 прикажа: БМИ (30,7 ± 5 и 31 ± 5,2; p < 0.7), HbA1c (6,4 ± 0.8 & 5,1 ± 0,8, p < 0,002), ППГ (8,2 ± 1,7 & 6,9 ± 1,5, p < 0,02), ГН (35,2 ± 3,8 и 38,6 ± 1,5, p < 0.0001) и РТ (2289 ± 504 и 3474 ± 334, p < 0,0001). Споредбата помеѓу Г2 и Г3 прикажа: BMI (31 ± 5,2 и 33,4 ± 6,1; p < 0,02), HbA1c (5,2 ± 1,1 и 6,4 ± 2,3, p < 0,02), ППГ (6,9 ± 1,5 и 8,2 ± 1,9, p < 0,02), ГН (38,6 ± 1,5 и 39,3 ± 1,4, p < 0,01) и РТ (3474 ± 334 и 4431 ± 302, p < 0,0001). Споредбата помеѓу Г1 и Г3 прикажа разлика во ГН и РТ (p < 0,0001).

Заклучок: Обезноста на мајката и ППГ придонесуваат кон макросомија, а исто така и ППГ кон СГА.

Клучни зборови: гестациски дијабетес, голема телесна тежина за гестациска возраст, мала телесна тежина за гестациска возраст, родилна тежина, гликемија по оброк.

Corresponding Author:

Krstevska B.
Clinic of Endocrinology, Diabetes and Metabolic Disorders,
Faculty of Medicine,
1000 Skopje, R. Macedonia

E-mail: branakrstevska@yahoo.com