Gestational diabetes: weight gain during pregnancy and its relationship to pregnancy outcomes

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Abstract

Background: Weight gain during pregnancy reflects the mother’s nutritional status. However, it may be affected by nutritional therapy and exercise interventions used to control blood sugar in gestational diabetes mellitus (GDM). This study aimed to evaluate weight gain during gestation and pregnancy outcomes among women with GDM.

Methods: A retrospective study involving 1523 women with GDM was conducted between July 2013 and July 2016. Demographic data, gestational weight gain (GWG), blood glucose, glycated-hemoglobin level, and maternal and fetal outcomes were extracted from medical records. Relationships between GWG and pregnancy outcomes were investigated using multivariate logistic regression.

Results: In total, 451 (29.6%) women showed insufficient GWG and 484 (31.8%) showed excessive GWG. Excessive GWG was independently associated with macrosomia (adjusted odds ratio [aOR] 2.20, 95% confidence interval [CI] 1.50–3.52, \( P < 0.001 \)), large for gestational age (aOR 2.06, 95% CI 1.44–2.93, \( P < 0.001 \)), small for gestational age (aOR 0.49, 95% CI 0.25–0.97, \( P = 0.040 \)), neonatal hypoglycemia (aOR 3.80, 95% CI 1.20–12.00, \( P = 0.023 \)), preterm birth (aOR 0.45, 95% CI 0.21–0.96, \( P = 0.040 \)), and cesarean delivery (aOR 1.45, 95% CI 1.13–1.87, \( P = 0.004 \)). Insufficient GWG increased the incidence of preterm birth (aOR 3.53, 95% CI 1.96–6.37, \( P < 0.001 \)).

Conclusions: Both excessive and insufficient weight gain require attention in women with GDM. Nutritional therapy and exercise interventions to control blood glucose should also be used to control reasonable weight gain during pregnancy to decrease adverse pregnancy outcomes.

Keywords: Gestational diabetes mellitus; Weight gain; Pregnancy outcomes

Introduction

Gestational weight gain (GWG) is an important index for the health and quality of life of women and their fetuses. A previous study reported excessive GWG was associated with pregnancy complications and adverse fetal outcomes.[1] Other research indicated that excessive GWG was positively associated with preterm birth in women who were overweight or obese, insuficient GWG was positively associated with preterm birth.[2] Therefore, appropriate GWG, as recommended according to a woman’s body mass index (BMI) before pregnancy, is important for the health of pregnant women and their fetuses.

Gestational diabetes mellitus (GDM) is defined as any degree of glucose intolerance with onset during pregnancy,[3] and is one of the main causes of maternal and neonatal complications (e.g., preeclampsia, fetal macrosomia, preterm birth, and cesarean delivery).[4] Therefore, we hypothesized that women with GDM and abnormal GWG would show a higher incidence of adverse pregnancy outcomes. However, the extent to which GWG is associated with adverse outcomes in GDM has not been fully elucidated. Some previous studies reported that excessive GWG was independently associated with adverse pregnancy outcomes in women with GDM,[5-7] but the effects of insufficient GWG have not been analyzed in detail.

In China, insufficient GWG occurs in 12.5% of pregnant women.[2] However, the proportion may be higher among women with GDM because of the effects of nutritional therapy and exercise, which are the main methods used to control blood glucose in GDM.[8] The prevalence of GDM in China has recently increased to 14.7–20.9%,[9] which
represents epidemic proportions. Therefore, it is essential to understand whether abnormal GWG is associated with pregnancy outcomes in women with GDM. To assess these relationships, we performed a retrospective analysis of 1523 women with GDM.

Methods
This retrospective cohort study drew on data recorded for women with GDM who delivered at a hospital in Beijing, China, between July 2013 and July 2016. The selection of participants is shown in Figure 1. Of the 9594 deliveries in the study period, we identified 2206 women with diabetes. Only women with a diagnosis of GDM based on a 75-g oral glucose tolerance test (OGTT) were included; 311 women with pre-gestational diabetes were excluded. Other exclusion criteria were: women with uncompleted medical records (n = 267), hypertension history (n = 93), fetal anomalies (n = 7), and twin pregnancy (n = 5). Finally, 1523 women were included in the analysis. All women in this study received standard antenatal examinations and nutritional guidance, which was based on the 2014 guidelines for diagnosis and treatment of GDM[10] at our hospital.

Ethical approval
This research was approved by the Hospital Ethical Review Committee (No. 2017-p2-002-01). Given the retrospective nature of this study, informed consent was not required from the women included in the analysis.

Diagnostic criteria for GDM
The International Association of Diabetes and Pregnancy Study Groups criteria were used to diagnose GDM. GDM was diagnosed when any plasma glucose value was greater than fasting plasma glucose ≥ 5.1 mmol/L, plasma glucose after 1 h ≥ 10.0 mmol/L or after 2 h of ≥ 8.5 mmol/L using a 75-g OGTT.

Data collection
Clinical data were collected from hospital medical records. The main parameters were: maternal age, family history of diabetes, parity, weight before pregnancy (self-reported), height (self-reported), maternal weight and gestational age at delivery, blood glucose after a 75-g OGTT, insulin therapy, glycated-hemoglobin (Hb) level, mode of delivery, birth weight and height of newborn, neonatal blood glucose, Apgar score, post-partum hemorrhage (PPH), premature rupture of the membranes (PROMs), gestational hypertension, preeclampsia, and amniotic fluid pollution.

BMI before pregnancy and GWG
Weight before pregnancy and height were obtained from medical records and used to calculate BMI (weight

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**Figure 1: Flow diagram of cohort derivation.**
in kg/height in m²). Pre-pregnancy BMI was categorized in 3 groups according to World Health Organization criteria: underweight (<18.5 kg/m²), normal weight (18.5–24.9 kg/m²), and overweight/obese (≥25.0 kg/m²). GWG was divided into groups according to Institute of Medicine (IOM) recommendations. The insufficient GWG group was defined as weight gain during gestation of <1.5 kg in underweight women, <11.5 kg in normal weight women, <7 kg in overweight women (BMI 25.0–30.0 kg/m²), and <5 kg in obese women (≥30.0 kg/m²). The excessive GWG group, which was defined as a weight gain during gestation of >18 kg in underweight women, >16 kg in normal weight women, >11.5 kg in overweight women, and >9 kg in obese women. All other women were classified as having sufficient GWG (within IOM recommendations).

**Pregnancy outcomes**

Maternal pregnancy outcomes included: preterm delivery (at <37 weeks of gestation); gestational hypertension (systolic blood pressure >140 mmHg [1 mmHg = 0.133 kPa] or diastolic blood pressure >90 mmHg after 20 weeks of gestation, without proteinuria); pre-eclampsia (defined as high blood pressure and proteinuria at 20 weeks of gestation); PPH (defined as hemorrhage >500 mL after 24 h of delivery); PROM (defined as premature rupture of membranes before 37 weeks of gestation or the onset of labor); and cesarean delivery (including selective and emergency cesarean delivery).

Neonatal outcomes included: macrosomia (birth weight greater than 4000 g); large for gestational age (LGA) (weight greater than the 90th percentile for gestational age); small for gestational age (SGA) (weight less than the 10th percentile for gestational age); hypoglycemia; and amniotic fluid pollution (defined by the presence of meconium in the amniotic fluid).

**Statistical analyses**

Statistical analyses were performed using SPSS version 17.0 (SPSS Inc., Chicago, IL, USA). Data distribution was tested for normality by visual inspection of histograms and the Shapiro-Wilk W test. Continuous variables are described using mean ± standard deviation (SD). Differences among the 3 groups were analyzed using analysis of variance followed by the least significant difference post-hoc test, as appropriate. P-values <0.05 were considered statistically significant. Categorical variables are described as frequencies. A Chi-squared test was used to evaluate differences in frequencies, with P <0.017 considered statistically significant.

Multinomial logistic regression was performed to investigate associations between predictors (BMI, GWG, and other relevant covariates) and each maternal and neonatal complication (including macrosomia, LGA, SGA, preterm delivery, hypertension, and cesarean delivery). Adjusted odds ratios (aOR) and 95% confidence intervals (CIs) were calculated.

**Results**

In total, 1523 women with GDM were included in our primary analyses. Table 1 shows that 451 (29.6%) women had insufficient GWG, 484 (31.8%) had excessive GWG, and 588 (38.6%) had sufficient GWG, according to the IOM recommendations for GWG. The average pre-pregnancy BMI was 23.6 ± 3.9 kg/m² (15.8–44.6 kg/m²), and was significantly higher in the excessive GWG group (24.6 ± 4.2 kg/m²) than the sufficient GWG (23.2 ± 3.8 kg/m²) and insufficient GWG (23.2 ± 3.6 kg/m²) groups (P < 0.05). The proportion of overweight/obese women (45.2%) was highest in the excessive GWG group, and women with insufficient GWG had the highest proportion (75.5%) of normal pre-pregnancy BMI. Maternal age and gestational age were significantly lower in the insufficient GWG group than in the other 2 groups (P < 0.05). Fasting plasma glucose was higher in the excessive GWG group than in the insufficient GWG group, but 1 and 2 hours glucose values during OGTT were significantly lower than in the other 2 GWG groups (P < 0.05). HbA1c was significantly higher in the excessive GWG group than in the insufficient GWG group (P < 0.05); however, somewhat counter-intuitively, women in this group needed less insulin than those in the other 2 GWG groups (P < 0.017).

Table 2 shows the influence of GWG on pregnancy complications. Neonatal height and weight were higher and neonatal blood glucose was lower in the excessive GWG group than in the other 2 groups (P < 0.05). The incidence of hypoglycemia in the excessive GWG and insufficient GWG groups were higher than in the sufficient GWG group (P < 0.05). Patients with excessive GWG had higher incidences of fetal distress, LGA, and macrosomia,

### Table 1: Maternal characteristics grouped by weight gain

| Characteristics | Insufficient (n=451) | Sufficient (n=588) | Excessive (n=588) |
|-----------------|----------------------|-------------------|------------------|
| Age (years)     | 31.5 ±3.9*          | 31.1 ±3.7*        | 30.8 ±3.7*       |
| Height (cm)     | 161.3 ±4.8          | 162.2 ±4.8        | 163.4 ±5.1       |
| Nulliparity     | 355 (78.7)          | 479 (81.3)        | 401 (82.9)       |
| Pre-pregnancy BMI (kg/m²) | 23.3 ±3.6 | 23.3 ±3.8 | 24.6 ±4.2*       |
| BMI category    |                      |                   |                  |
| Underweight     | 22 (4.9)            | 40 (6.8)          | 20 (4.1)         |
| Normal weight   | 336 (74.5)*         | 386 (65.6)*       | 245 (40.6)*      |
| Overweight-obese| 93 (20.6)           | 162 (27.6)*       | 219 (45.2)*      |
| 75-g OGTT (mg/dL) | 5.14 ±0.58  | 5.19 ±0.64       | 5.23 ±0.46*      |
| 1 h             | 9.57 ±1.75*        | 9.22 ±1.79*       | 8.96 ±1.75*      |
| 2 h             | 8.39 ±1.35*        | 8.00 ±1.46*       | 7.66 ±1.44*      |
| HbA1c (%)       | 5.19 ±0.38         | 5.21 ±0.40        | 5.26 ±0.43*      |
| Family history of diabetes | 102 (22.6) | 138 (23.5) | 90 (18.8) |
| Gestational weeks | 39.1 ±2.3*  | 39.3 ±1.4 *      | 39.5 ±2.6*       |
| Insulin therapy | 23 (5.1)           | 25 (4.3)          | 10 (2.1)*        |

Data presented as mean ± standard deviation or n (%). BMI: Body mass index; GWG: Gestational weight gain; IOM: Institute of Medicine; OGTT: Oral glucose tolerance test. *P < 0.05 vs. other two groups. †P < 0.017 vs. other two groups. ‡P < 0.05 vs. insufficient group. §P < 0.017 vs. insufficient group.
Table 2: Maternal and neonatal outcomes by maternal weight gain category

| Outcomes                        | Insufficient (n=451) | Sufficient (n=588) | Excessive (n=484) |
|---------------------------------|----------------------|--------------------|-------------------|
| Birth length (cm)               | 49.9±2.8*            | 50.2±1.9*          | 50.7±2.3*         |
| Birth weight (g)                | 3239±486E            | 3383±455E          | 3540±462E         |
| Birth glucose (mg/dL, n=1473)   | 75.5±20.6*           | 73.6±19.4*         | 71.1±19.5         |
| Hypoglycemia (n=1473)           | 9 (2.0)              | 4 (0.7)§           | 13 (2.8)          |
| Low APGAR                       | 21 (4.7)             | 20 (3.4)           | 22 (4.5)          |
| Fetal distress                  | 34 (7.5)             | 61 (10.4)          | 61 (12.6)         |
| Birth weight category           |                      |                    |                   |
| SGA                             | 24 (5.3)             | 31 (5.3)           | 13 (2.7)§         |
| AGA                             | 399 (88.5)           | 498 (84.7)         | 369 (76.2)        |
| LGA                             | 28 (6.2)             | 59 (10)            | 102 (21.1)§       |
| Macrosomia                      | 22 (4.9)             | 49 (8.3)           | 90 (18.6)         |
| Preterm birth                   | 40 (8.9)§            | 23 (3.9)           | 14 (2.9)          |
| Cesarean section                | 196 (43.5)           | 282 (48)           | 287 (59.3)        |
| Gestational hypertension        | 11 (2.4)             | 23 (3.9)           | 21 (4.3)          |
| Pre-eclampsia                   | 8 (1.8)              | 17 (2.9)           | 29 (6.0)§         |
| PPROM                           | 123 (27.3)           | 172 (29.3)         | 124 (25.6)        |
| PPH                             | 25 (5.5)             | 32 (5.4)           | 39 (8.1)          |
| Amniotic fluid pollution        | 96 (21.3)            | 143 (24.3)         | 126 (26.0)        |

Data presented as mean±standard deviation or n (%). AGA: Appropriate for gestational age; LGA: Large for gestational age; PPH: Post-partum hemorrhage; PPROM: Preterm pre-mature rupture of membranes; SGA: Small for gestational age. *P<0.05 vs. other two groups. †P<0.05 vs. excessive group. §P<0.017 vs. insufficient group. ‡P<0.017 vs. the other two groups.

Excessive GWG (aOR 2.20, 95% CI 1.50–3.35, P<0.001) was associated with increased risk for macrosomia after adjustment for maternal age, pre-pregnancy BMI, HbA1c, parity, and gestational age [Table 3]. In addition, excessive GWG (aOR 2.06, 95% CI 1.44–2.93, P<0.001) was associated with increased risk for LGA after adjustment for maternal age, parity, hypertensive disorders, pre-eclampsia, pre-pregnancy BMI, and HbA1c. SGA (aOR 0.49, 95% CI 0.25–0.97, P=0.040), preterm birth (aOR 0.45, 95% CI 0.21–0.96, P=0.041), and neonatal hypoglycemia (aOR 3.80, 95% CI 1.20–12.00, P=0.023) were associated with excessive GWG. Women with excessive GWG also had a greater likelihood of having had a cesarean delivery (aOR 1.45, 95% CI 1.13–1.87, P=0.004). Insufficient GWG had no association with SGA, hypoglycemia, and hypertensive disorders, but was highly associated with increased risk of preterm birth (aOR 3.53, 95% CI 1.96–6.37, P<0.001).

Discussion

The key findings of this study were that excessive GWG accounted for a large proportion (31.8%) of women, despite the fact that nutritional therapy and exercise interventions used to control blood sugar may help control weight gain during pregnancy. Excessive GWG increased the risk for macrosomia, LGA, neonatal hypoglycemia, and cesarean delivery, but was associated with a decreased risk for SGA. However, insufficient GWG was also common in women with GDM (accounting for 29.6% of the sample), which may be attributable to a strict diet and excessive activity. Insufficient GWG was not associated with neonatal hypoglycemia, cesarean delivery, and SGA, but significantly increased the risk for preterm birth by about 3.5 times compared with sufficient GWG. Weight management should be strengthened for patients with GDM to prevent both insufficient GWG and excessive GWG and reduce pregnancy complications.

In our study, 29.6% and 31.8% of the women showed insufficient and excessive GWG, respectively. A previous study reported insufficient weight gain occurred in 12.5% of general pregnant women and excessive weight gain in 57.9% in general pregnancy women.[24] In the present study, insufficient GWG was higher and excessive GWG was lower than in the previous study.[16] Nutritional therapy and exercise interventions were first-line treatments to control blood glucose after GDM diagnosis. However, some patients may over-limit their diet to achieve satisfactory blood glucose control, which will limit weight gain and even lead to weight loss. That may be why our study reported a lower rate of excessive GWG (31.8% vs. 57.9%) and higher rate of insufficient GWG (29.6% vs. 12.5%). Another study also showed nutritional therapy and exercise interventions had an effect on weight gain.[14] However, GWG above or below the IOM guidelines have adverse associations with maternal and infant outcomes.

Weight gain during gestation is closely related to neonatal weight. Our study found that excessive GWG increased the OR for macrosomia (aOR 1.54, 95% CI 1.07–2.21, P=0.02) and LGA (aOR 1.78, 95% CI 1.28–2.47, P=0.001). Insufficient GWG had no significant association with macrosomia (aOR 0.69, 95% CI 0.40–1.16) and LGA (aOR 0.73; 95% CI 0.30–1.74), but the aORs for macrosomia and LGA were lower. These findings were consistent with a previous study by Li et al that showed the OR for macrosomia was highest for women with GWG above the range recommended by the IOM and lowest for those with GWG below the recommendation.[15] However, Scifres et al[5] reported that both excessive GWG and insufficient GWG increased the risk for macrosomia. The differences between these data may arise from the different grouping method used by Scifres et al, which divided women into normal, overweight, and obese groups. The effect of GWG on SGA differed from that on LGA or macrosomia. Our study showed excessive GWG was associated with lower rates of SGA (aOR 0.49; 95% CI 0.25–0.97), which was consistent with a previous study.[16] However, we showed that insufficient GWG did not increase the risk for SGA. This result was not consistent with a retrospective cohort study in Taiwan,
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Table 3: Multivariate logistic regression analysis for adverse pre-natal outcomes associated with weight gain

| Outcomes                              | Unadjusted | P     | Adjusted OR | P     |
|---------------------------------------|------------|-------|--------------|-------|
| Macrosomia* (n=161)                   |            |       |              |       |
| Weight gain                           |            |       |              |       |
| Sufficient                             | Reference  |       | Reference    |       |
| Insufficient                           | 0.58 (0.34–0.97) | 0.040 | 0.67 (0.40–1.13) | 0.130 |
| Excessive                              | 2.34 (1.61–3.42) | <0.001 | 2.20 (1.50–3.25) | <0.001 |
| LGA† (n=189)                          |            |       |              |       |
| Weight gain                           |            |       |              |       |
| Sufficient                             | Reference  |       | Reference    |       |
| Insufficient                           | 0.61 (0.38–0.98) | 0.040 | 0.63 (0.40–1.00) | 0.055 |
| Excessive                              | 2.18 (1.53–3.09) | <0.001 | 2.06 (1.44–2.93) | <0.001 |
| SGA‡ (n=68)                           |            |       |              |       |
| Weight gain                           |            |       |              |       |
| Sufficient                             | Reference  |       | Reference    |       |
| Insufficient                           | 1.05 (0.61–1.82) | 0.860 | 1.23 (0.67–2.28) | 0.500 |
| Excessive                              | 0.48 (0.24–0.93) | 0.030 | 0.49 (0.25–0.97) | 0.040 |
| Neonatal Hypoglycemia (n=26)          |            |       |              |       |
| Weight gain                           |            |       |              |       |
| Sufficient                             | Reference  |       | Reference    |       |
| Insufficient                           | 3.00 (0.92–9.80) | 0.069 | 2.01 (0.59–7.50) | 0.260 |
| Excessive                              | 4.00 (1.30–12.38) | 0.016 | 3.80 (1.20–12.00) | 0.023 |
| Preterm birth‡ (n=77)                  |            |       |              |       |
| Weight gain                           |            |       |              |       |
| Sufficient                             | Reference  |       | Reference    |       |
| Insufficient                           | 2.58 (1.51–4.40) | <0.001 | 3.26 (1.82–5.82) | <0.001 |
| Excessive                              | 0.63 (0.32–1.25) | 0.190 | 0.45 (0.21–0.96) | 0.041 |
| Cesarean section‡ (n=765)              |            |       |              |       |
| Weight gain                           |            |       |              |       |
| Sufficient                             | Reference  |       | Reference    |       |
| Insufficient                           | 0.86 (0.67–1.10) | 0.220 | 0.82 (0.64–1.06) | 0.130 |
| Excessive                              | 1.44 (1.12–1.84) | 0.004 | 1.45 (1.13–1.87) | 0.004 |
| Hypertensive disorders‡ (n=108)        |            |       |              |       |
| Weight gain                           |            |       |              |       |
| Sufficient                             | Reference  |       | Reference    |       |
| Insufficient                           | 0.64 (0.36–1.13) | 0.120 | 0.61 (0.34–1.07) | 0.080 |
| Excessive                              | 1.26 (0.80–1.96) | 0.320 | 1.26 (0.80–1.98) | 0.310 |

All models include pre-pregnancy body mass index and HbA1c. In addition, regression models for each outcome included: 1. Maternal height, age, parity, gestational weeks. 2. Maternal age, parity, hypertensive disorders, pre-eclampsia. 3. Maternal age, parity, hypertensive disorders, pre-eclampsia, pre-mature rupture of membranes. 4. Maternal age, parity, hypoglycemia in newborns. However, this needs to be further confirmed by laboratory examinations.

China which reported GWG below the IOM guidelines was associated with higher rates of SGA. The reason for this difference may be the different participants (GDM vs. general pregnancy) in the 2 studies. Our participants were all women with GDM, and GDM was associated with LGA.

Neonatal hypoglycemia is a common biochemical abnormality encountered in newborns that can cause brain damage and death. Babies born at risk have an increased risk for developmental delay in later life. Infants born to mothers with GDM are more likely to be hypoglycemic. We found that excessive GWG was associated with higher incidence of neonatal hypoglycemia (aOR 2.01; 95% CI 0.59–7.50). Until now, no study had considered the association between weight gain and neonatal hypoglycemia, but recent studies have added to our understanding of a cause of hypoglycemia being hyperinsulinism. Animal studies suggest that GWG is associated with changes in the hormonal milieu, including insulin resistance. Therefore, we hypothesized that excessive weight may cause insulin levels to rise, which may increase the incidence of hypoglycemia in newborns. However, this needs to be further confirmed by laboratory examinations.

Many previous studies have investigated risk factors for preterm birth. For example, Liu et al reported that the risk for preterm delivery was increased in those showing excessive GWG (by 1.5-fold). Huang et al found that both insufficient and excessive GWG increased the risk for preterm birth in general pregnant women. The results of our study focused on GDM showed some differences from previous studies. We did not find excessive GWG increased the risk for preterm birth, in contrast, it decreased the risk.
for preterm birth (aOR 0.45, P = 0.041), whereas insufficient GWG dramatically increased the risk for preterm birth (aOR 3.26, P < 0.001). The rate of insufficient GWG was also high (29.6%) among women with GDM in the present study, which was higher than that previously reported among general pregnant women (12.5%) [13]. These findings suggest there is an urgent need to conduct patient education to avoid excessive restriction of diet and excessive exercise and ensure reasonable weight gain during pregnancy across GDM population. This will help to reduce the incidence of preterm birth.

After adjusting for many factors, including macrosomia, we found excessive GWG was associated with a higher rate of cesarean section (aOR 1.45, 95% CI 1.13–1.87), which was consistent with several previous studies involving pregnant women. Blackwell’s study concluded that in women with both treated and untreated mild GDM, excessive GWG was independently associated with cesarean delivery. [21] Even in a general pregnant cohort (that did not consider if women had GDM), women with excessive GWG had an increased likelihood of cesarean delivery (aOR 1.31; 95% CI 1.18–1.36). [12] However, other studies have concluded that GWG did not have a significant influence on the occurrence of cesarean delivery. [23] Another study showed that women with less than the recommended GWG in the second trimester had a lower risk for cesarean deliveries (risk ratio 0.82, 95% CI 0.71–0.96) than women with sufficient GWG in that trimester. However, we did not find similar results.

In our study, there was no association between GWG and hypertensive disorders (including gestational hypertension and pre-eclampsia). This observation was not consistent with data from previous investigations. Fortner et al. [25] found that pregnant woman with high GWG had a 3-fold increased risk for hypertension and a 4-fold increased risk for pre-eclampsia, compared with pregnant woman showing normal GWG. Among women with GDM, Scifres et al. [5] showed that women with excessive GWG were at higher risk for hypertensive disorders (gestational hypertension and pre-eclampsia, aOR 2.19 and aOR 1.74, respectively) than other women. In all gestational periods, GWG was found to be positively associated with concurrent blood pressure change. [24] Therefore, insufficient GWG may decrease the incidence of hypertensive disorders; however, we did not find this association in our study.

This study had some limitations. First, it was a retrospective observational study; therefore, selection and information bias cannot be excluded. Second, pre-pregnancy weight and height were self-reported, meaning pre-pregnancy BMI estimates might have been affected by reporting bias. Third, assessment of women’s diet and physical activity was not performed, meaning that the impact of these variables on perinatal outcomes could not be investigated. Fourth, because of the lack of weight measurement at the time of GDM diagnosis, it was impossible to analyze the effect of GWG specifically on maternal and neonatal outcomes after the diagnosis of GDM. Finally, this study did not distinguish weight gain at different stages of pregnancy, which implies that the effect of GWG on certain pregnancy outcomes could have been missed, overestimated, or underestimated.

In summary, a large proportion of women with GDM in the study sample had excessive GWG, even after receiving nutritional therapy and exercise for GDM. However, insufficient weight gain remains a major concern. Excessive GWG is associated with increased rates of macrosomia, LGA, pre-mature delivery, cesarean section, and hypertensive disorders. Insufficient GWG is associated with a higher rate of preterm birth. These findings suggest it is necessary to maintain a reasonable weight gain during gestation among women with GDM, as well as preventing insufficient and excessive weight gain, to reduce adverse pregnancy outcomes for newborns and mothers.

Conflicts of interest
None.

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