

Original Article

Association between early gestational weight gain and the risk of gestational diabetes mellitus: a retrospective study

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Abstract: Objective: To investigate the correlation between excessive weight gain during early pregnancy and the risk of gestational diabetes mellitus (GDM), and to provide evidence for early clinical intervention. Methods: The clinical data of 152 women with singleton pregnancies who were admitted to the Department of Obstetrics, PLA General Hospital Hainan Branch, from January 2022 to December 2024 were retrospectively analyzed. According to weight gain during early pregnancy (before 14 weeks of gestation), the pregnant women were divided into an excessive weight gain group (> 2.0 kg/week, 68 cases) and a normal weight gain group (0.5-2.0 kg/week, 84 cases). Baseline clinical characteristics, including maternal age, pre-pregnancy body mass index (BMI), obstetric history, and family history, were collected. An oral glucose tolerance test (OGTT) was performed at 24-28 weeks of gestation to screen for GDM. Univariate analysis and multivariate logistic regression analysis were performed to evaluate the relationship between early pregnancy weight gain and the risk of GDM. Results: Among 152 pregnant women, 35 were diagnosed with GDM, with an incidence of 23.0%. The incidence of GDM in the excessive weight gain group was 32.4% (22/68), significantly higher than 15.5% (13/84) in the normal weight gain group ($\chi^2=6.214$, $P=0.013$). No significant differences were observed in baseline characteristics, including maternal age, pre-pregnancy BMI, and gravidity, between the two groups ($P > 0.05$). Multivariate logistic regression analysis showed that, after adjusting for potential confounding factors such as maternal age and pre-pregnancy BMI, excessive weight gain during early pregnancy remained an independent risk factor for GDM (OR=2.876, 95% CI: 1.543-5.362, $P=0.001$). Conclusion: Excessive weight gain during early pregnancy is associated with an increased risk of GDM. Therefore, clinical monitoring and management of weight gain during early pregnancy should be strengthened, and early intervention should be implemented for pregnant women with excessive weight gain to reduce the risk of GDM.

Keywords: Gestational diabetes mellitus, early pregnancy, weight gain, risk factors, retrospective study

Introduction

Gestational diabetes mellitus (GDM) is a common disorder of glucose metabolism during pregnancy. It is characterized by the first occurrence or detection of varying degrees of impaired glucose tolerance during pregnancy. The development of GDM is closely associated with decreased insulin sensitivity, increased insulin resistance, and dysregulated nutritional metabolism during pregnancy. GDM may lead to adverse maternal and neonatal outcomes, including premature rupture of membranes, macrosomia, and postpartum hemorrhage, posing serious threat to the health of mothers

and infants [1, 2]. Epidemiological studies have shown that, in recent years, the global prevalence of gestational hyperglycemia has continued to increase due to changes in fertility policies, a growing proportion of advanced maternal age pregnancies, and inappropriate nutritional intake during pregnancy. The incidence of GDM in China has exceeded 15%, and its incidence continues to increase annually, making it an important obstetric complication requiring clinical prevention and control [3, 4]. Weight management during pregnancy is a critical component of regulating glucose and lipid metabolism and reducing the risk of metabolic diseases during pregnancy. As a key stage of metabolic

adaptation and placental development, abnormal weight gain during early pregnancy may have a prominent impact on subsequent glucose homeostasis [5].

Currently, it is widely recognized that excessive gestational weight gain can increase insulin resistance and consequently elevate the risk of GDM. However, previous studies have primarily focused on the correlation between GDM and weight gain throughout pregnancy or during the second and third stages. Evidence on the relationship between the rate of weight gain during early pregnancy (before 14 weeks of pregnancy) and the risk of GDM remains limited, and no consensus has been established regarding early pregnancy weight control [6, 7]. Moreover, many existing studies have not fully accounted for potential confounding factors, such as maternal age and pre-pregnancy body mass index (BMI), thus limiting the reliability of their conclusions.

This study retrospectively analyzed the clinical data of women with singleton pregnancies to investigate the correlation between excessive weight gain during early pregnancy and the risk of GDM, and to determine its independent effect. The findings may provide evidence for early pregnancy weight monitoring, individualized intervention planning, and the prevention of GDM.

Data and methods

General information

Clinical data of 152 women with singleton pregnancies who were admitted to the Department of Obstetrics of our hospital between January 2022 and December 2024 were retrospectively analyzed. According to weight gain during the first trimester (before 14 weeks of gestation), the included subjects were divided into an excessive weight gain group (> 2.0 kg/week, $n=68$) and a normal weight gain group ($0.5-2.0$ kg/week, $n=84$). This study was approved by the Ethics Committee of PLA General Hospital Hainan Branch (Ethical Approval No. KYYLL-2024-05-12). It should be noted that the current authoritative guidelines both domestically and internationally (such as the IOM 2009 guidelines, and the Chinese *Guidelines for Diagnosis and Treatment of Gestational*

Hyperglycemia (2022)) provide recommendations for overall gestational weight gain and have not established a universally accepted cutoff value for the weekly weight gain during the early pregnancy. Therefore, the grouping criteria used in this study were mainly set based on the distribution characteristics of clinical data from our center and previous literature, which has certain limitations.

Sample size estimation

Given the retrospective design of this study, no priori sample size estimation was performed. A post-hoc power analysis was conducted using PASS 15.0 software. The analysis was performed using a two-sample Z-test for proportions with a two-sided $\alpha=0.05$. Based on the observed GDM incidence rates of 32.4% in the excessive weight gain group and 15.5% in the normal weight gain group, and the actual sample sizes (68 and 84 cases, respectively), the calculated statistical power was approximately 0.83, exceeding the commonly recommended threshold of 0.80.

Inclusion criteria

1. Age ≥ 18 years; 2. Single pregnancy; 3. Establishment of pregnancy records and completion of regular prenatal examinations at our hospital during the early stage of pregnancy (before 14 weeks of pregnancy); 4. Completion of oral glucose tolerance test (OGTT) screening at 24-28 weeks of gestation in our hospital; 5. Availability of complete clinical data.

Exclusion criteria

1. Pre-existing diabetes mellitus diagnosed before or during pregnancy (including type 1 and type 2 diabetes); 2. Multiple pregnancy; 3. Complicated with severe cardiac, hepatic, or renal dysfunction; 4. Thyroid dysfunction, polycystic ovary syndrome, or other endocrine disorders affecting glucose metabolism; 5. Use of medications that may affect blood glucose levels during pregnancy, such as glucocorticoids; 6. Incomplete clinical data.

Data collection

The clinical information of pregnant women was obtained from the electronic medical

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record system, including baseline characteristics, weight gain during early pregnancy, laboratory examination results, and pregnancy outcomes.

Baseline data collection: Baseline characteristics included maternal age, pre-pregnancy body mass index (BMI), gravidity, obstetric history, family history of diabetes, family history of hypertension, smoking history, and other relevant information.

Assessment of weight gain during early pregnancy: Gestational weight gain rate during early pregnancy (before 14 weeks of gestation) was calculated as the difference between the weight measured at the first prenatal examination and the last recorded weight before 14 weeks of gestation, divided by the corresponding gestational weeks. According to the rate, participants were divided into the excessive weight gain group (> 2.0 kg/week) and the normal weight gain group (0.5 - 2.0 kg/week).

Laboratory examination: An oral glucose tolerance test (OGTT) was performed at 24-28 weeks of gestation. After fasting for 8-12 hours, venous blood samples were collected in the next morning to measure fasting plasma glucose. Subsequently, participants orally ingested a solution containing 75 g of glucose, and venous blood samples were collected 1 and 2 hours after glucose administration. Glucose oxidase method (Roche Cobas c702 analyzer) was used to measure fasting, 1-hour and 2-hour plasma glucose levels. According to the criteria established by the International Association of Diabetes and Pregnancy Study Group (IADPSG), GDM was diagnosed when any of the following criteria were met: fasting blood glucose ≥ 5.1 mmol/L, 1-hour plasma glucose ≥ 10.0 mmol/L, or 2-hour plasma glucose ≥ 8.5 mmol/L.

Collection of other indicators: Additional clinical outcomes included neonatal birth weight, mode of delivery (natural delivery/cesarean section), and adverse pregnancy outcomes, including gestational hypertension, preterm birth, premature rupture of membranes, fetal distress, postpartum hemorrhage, macrosomia, neonatal hypoglycemia, and neonatal admission to the neonatal intensive care unit (NICU).

Observation indicators

Primary outcome measures: The primary outcomes included the incidence of GDM, which was compared between the two groups, and the correlation between early gestational weight gain and the risk of GDM.

Secondary outcome measures: The incidence of adverse maternal pregnancy outcomes, including gestational hypertension, premature delivery, premature rupture of membranes, cesarean section, fetal distress, and postpartum hemorrhage, was compared between the two groups.

Neonatal outcomes, including neonatal birth weight, macrosomia, neonatal hypoglycemia, and neonatal admission to NICU, were also compared between the two groups.

Statistical methods

All statistical analyses were performed using SPSS 26.0 (IBM Corp., Armonk, NY, USA). Continuous variables were first tested for normality using the Kolmogorov-Smirnov test (or the Shapiro-Wilk test when $n < 50$). Normally distributed variables were expressed as mean $\pm$ standard deviation (SD), and comparisons between two groups were performed using the independent-samples t-test. Non-normally distributed variables were expressed as median (interquartile range, IQR) and compared using the Mann-Whitney U test. Categorical variables were expressed as frequencies and percentages [n (%)] and compared using the chi-square (χ^2) test or Fisher's exact test when expected frequency was < 5 .

Univariate logistic regression analysis was first performed to identify potential factors associated with GDM, including maternal age, pre-pregnancy BMI, gravidity, family history of diabetes, and weight gain during early pregnancy. Variables with $P < 0.10$ in the univariate analysis were subsequently entered into a multivariate binary logistic regression model. Before multivariate analysis, multicollinearity was assessed using the variance inflation factor (VIF) and tolerance. A VIF < 5 and tolerance > 0.1 indicated no significant multicollinearity. The Enter method was used for variable selection, with an entry criterion set at $\alpha=0.05$ and

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Table 1. Comparison of baseline characteristics between the two groups

Project	Excessive weight gain group (n=68)	Normal weight gain group (n=84)	Statistical value (t/ χ^2 /Z)	P
Age (years)	28.5 $\pm$ 3.2	29.1 $\pm$ 3.5	-1.102	0.272
Pre-pregnancy BMI (kg/m ²)	22.3 $\pm$ 2.8	22.0 $\pm$ 2.6	0.684	0.495
Pregnancy times	1.9 $\pm$ 1.1	2.0 $\pm$ 1.0	-0.589	0.557
Number of births	0.6 $\pm$ 0.5	0.6 $\pm$ 0.5	0.000	1.000
Primipara [n (%)]	42 (61.8)	53 (63.1)	0.028	0.867
Educational level (high school and above) [n (%)]	51 (75.0)	64 (76.2)	0.029	0.865
Family monthly income ($\geq$ 5000 yuan) [n (%)]	47 (69.1)	59 (70.2)	0.022	0.882
Smoking history [n (%)]	5 (7.4)	6 (7.1)	0.003	0.958
Drinking history [n (%)]	8 (11.8)	10 (11.9)	0.001	0.979
Pregnancy complicated with hypertension [n (%)]	4 (5.9)	5 (6.0)	0.001	0.981

Note: BMI, body mass index.

Table 2. Comparison of the incidence of gestational diabetes mellitus (GDM) between the two groups

Groups	Number of cases	Number of GDM cases	The incidence of GDM (%)	χ^2 value	P value
Excessive weight gain group	68	22	32.4	6.214	0.013
Normal weight gain group	84	13	15.5		
Total	152	35	23.0		

Note: GDM, gestational diabetes mellitus.

a removal criterion of $\alpha=0.10$. The model was used to determine whether excessive weight gain during early pregnancy was an independent risk factor for GDM, and odds ratios (ORs) with 95% confidence interval (CI) were calculated. A two-sided *P*-value < 0.05 was considered statistically significant.

Results

Comparison of baseline characteristics between the two groups

No significant differences were observed between the excessive weight gain group and normal weight gain group in baseline characteristics, including age, pre-pregnancy BMI, gravidity, parity, proportion of primiparas, education level, monthly household income, smoking history, alcohol consumption history, and pregnancy complicated by hypertension ($P > 0.05$; **Table 1**).

Comparison of the prevalence of GDM between the two groups

Among the included pregnant women, 35 were diagnosed with GDM, with an overall incidence of 23.0%. The prevalence of GDM in the excessive weight gain group was 32.4% (22/68),

which was significantly higher than 15.5% (13/84) in the normal weight gain group ($\chi^2=6.214$, $P=0.013$; **Table 2**).

Comparison of oral glucose tolerance test results between the two groups

All participants underwent an OGTT at 24-28 weeks of gestation. Fasting plasma glucose, 1-hour plasma glucose, and 2-hour plasma glucose levels were measured, showing that the fasting, 1-hour, and 2-hour plasma glucose levels were higher in the excessive weight gain group than those in the normal weight gain group ($P < 0.05$; **Figure 1**). Significant differences were observed in 1-hour and 2-hour plasma glucose levels between the two groups ($P < 0.05$).

Comparison of insulin metabolism-related indicators between the two groups

The levels of fasting insulin, glycated hemoglobin (HbA1c), and homeostasis model assessment of insulin resistance (HOMA-IR) were significantly higher in the excessive weight gain group than those in the normal weight gain group (all $P < 0.001$; **Table 3**).

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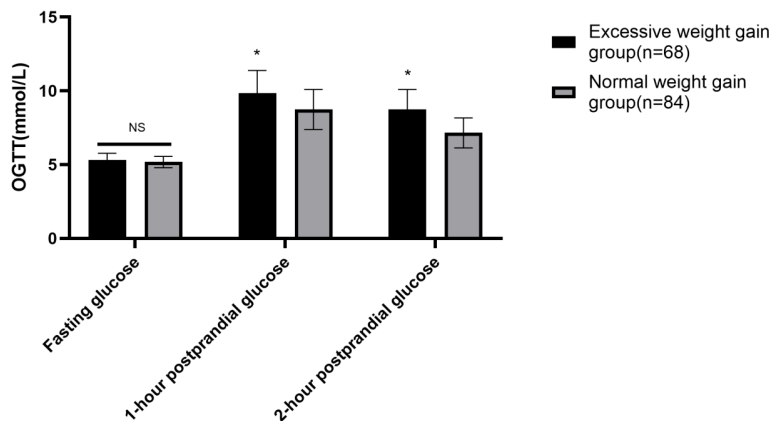


Figure 1. Comparison of OGTT blood glucose levels between the two groups ($\bar{x} \pm \text{sd}$, mmol/L). Note: OGTT, oral glucose tolerance test.

Univariate and multivariate logistic regression analyses of GDM

Univariate logistic regression analysis showed that excessive weight gain during early pregnancy was significantly associated with the risk of GDM (OR=2.612, 95% CI: 1.456-4.686, $P=0.001$), while age, pre-pregnancy BMI, family history of GDM, and other factors were not significantly associated with the risk of GDM ($P > 0.05$). Variables with $P < 0.10$ in univariate analysis, including excessive weight gain during early pregnancy, pre-pregnancy BMI, and family history of GDM, were subsequently included in the multivariate logistic regression analysis. The results showed that after adjustment for confounding factors, excessive weight gain during early pregnancy remained independently associated with an increased risk of GDM (OR=2.876, 95% CI: 1.543-5.362, $P=0.001$; **Table 4**).

To assess the robustness of the association between early pregnancy weight gain and GDM, a sensitivity analysis was performed by applying alternative cut-off values for excessive weight gain and repeating the multivariate logistic regression analysis. When excessive weight gain was defined as > 1.8 kg/week, the OR was 2.95 (95% CI: 1.58-5.51, $P=0.001$). When the cutoff was set at > 2.2 kg/week, the OR was 2.68 (95% CI: 1.42-5.06, $P=0.002$). The direction and significance of the associations remained consistent across different cutoff values, indicating that the findings were relatively robust. Multicollinearity diagnostics were performed before multivariate regres-

sion analysis. The VIF values ranged from 1.08 to 1.52 (all < 5), and tolerance values ranged from 0.66 to 0.93 (all > 0.1), indicating no significant multicollinearity among the variables included in the model.

Subgroup analysis and interaction analysis between excessive weight gain during early pregnancy and GDM

As shown in **Table 5**, subgroup analyses showed that excessive weight gain during early pregnancy was significantly associated with an increased risk of GDM across different age groups (< 35 years and ≥ 35 years) and different pre-pregnancy BMI groups (< 24 kg/m² and ≥ 24 kg/m²) (all $P < 0.05$). In pregnant women with a family history of GDM, excessive weight gain showed a stronger association with GDM risk (OR=5.000, 95% CI: 1.654-15.115, $P=0.004$). However, among those without a family history of GDM, the association did not reach statistical significance (OR=1.929, 95% CI: 0.956-3.892, $P=0.067$).

Interaction analysis showed no significant interactions between age, pre-pregnancy BMI, and excessive weight gain during early pregnancy (P for interaction > 0.05). A marginal interaction was observed between family history of GDM and excessive weight gain (P for interaction=0.086), suggesting that pregnant women with a family history of GDM may be more susceptible to the effects of excessive weight gain in early pregnancy.

Comparison of pregnancy complications and adverse pregnancy outcomes between the two groups

Regarding pregnancy complications and adverse pregnancy outcomes, the incidences of preterm birth (14.7% vs. 4.8%, $\chi^2=4.652$, $P=0.031$), premature rupture of membranes (11.8% vs. 3.6%, $\chi^2=4.012$, $P=0.045$), cesarean section (41.2% vs. 26.2%, $\chi^2=4.126$, $P=0.042$), fetal distress (10.3% vs. 2.4%, $\chi^2=4.512$, $P=0.034$), and postpartum hemorrhage (8.8% vs. 1.2%, $\chi^2=4.987$, $P=0.026$) were sig-

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Table 3. Comparison of insulin, HbA1c, and HOMA-IR between the two groups ($\bar{x} \pm sd$)

Groups	Number of cases	Fasting insulin (μ U/mL)	HbA1c (%)	HOMA-IR
Excessive weight gain group	68	12.56 $\pm$ 3.42	5.68 $\pm$ 0.45	2.98 $\pm$ 0.86
Normal weight gain group	84	9.23 $\pm$ 2.85	5.32 $\pm$ 0.38	2.12 $\pm$ 0.67
t value		5.234	4.876	6.125
P value		< 0.001	< 0.001	< 0.001

Notes: HOMA-IR, Homeostatic Model Assessment for Insulin Resistance; HbA1c, Glycated Hemoglobin.

Table 4. Univariate and multivariate logistic regression analysis of risk factors for GDM

Variable	Univariate analysis		Multivariate analysis	
	OR (95% CI)	P	OR (95% CI)	P
Age (years)	1.082 (0.956-1.225)	0.214	1.065 (0.932-1.218)	0.356
Pre-pregnancy BMI (kg/m^2)	1.124 (0.986-1.282)	0.078	1.098 (0.954-1.264)	0.196
Excessive weight gain during early pregnancy	2.612 (1.456-4.686)	0.001	2.876 (1.543-5.362)	0.001
Pregnancy (≥ 2 times)	1.156 (0.824-1.623)	0.398	-	-
Birth times (≥ 1)	1.089 (0.756-1.568)	0.642	-	-
Family history of GDM	1.856 (0.967-3.562)	0.062	1.612 (0.812-3.198)	0.168
History of hypertension	1.423 (0.654-3.098)	0.376	-	-

Note: GDM, gestational diabetes mellitus; BMI, body mass index.

Table 5. Subgroup analysis and interaction between excessive weight gain during early pregnancy and GDM

Subgroup	Excessive weight gain group (n=68)	Normal weight gain group (n=84)	OR (95% CI)	P	Interaction P value
Age					0.342
< 35 years old (n=98)	13/42 (31.0%)	8/56 (14.3%)	2.687 (1.234-5.852)	0.013	
≥ 35 years old (n=54)	9/26 (34.6%)	5/28 (17.9%)	2.431 (1.028-5.749)	0.043	
Pre-pregnancy BMI					0.218
< 24 kg/m^2 (n=112)	14/48 (29.2%)	9/64 (14.1%)	2.524 (1.198-5.316)	0.015	
≥ 24 kg/m^2 (n=40)	8/20 (40.0%)	4/20 (20.0%)	2.667 (1.082-6.571)	0.033	
Family history of GDM					0.086
Yes (n=38)	10/18 (55.6%)	4/20 (20.0%)	5.000 (1.654-15.115)	0.004	
No (n=114)	12/50 (24.0%)	9/64 (14.1%)	1.929 (0.956-3.892)	0.067	

Note: GDM, gestational diabetes mellitus.

nificantly higher in the excessive weight gain group than those in the normal weight gain group (all $P < 0.05$; **Table 6**).

Comparison of neonatal outcomes between the two groups

Regarding neonatal outcomes, the incidences of macrosomia, neonatal hypoglycemia, and neonatal NICU admission in the excessive weight gain group were significantly higher than those in the normal weight gain group (all $P < 0.05$; **Table 7**). No significant differences were

observed between the two groups in term birth rate and vaginal delivery rate ($P < 0.05$).

Discussion

Gestational diabetes mellitus (GDM) is a common metabolic disorder during pregnancy, characterized by impaired glucose metabolism during pregnancy [8]. Previous studies have shown that GDM not only increases the risk of maternal complications during pregnancy but also contributes to adverse outcomes, such as neonatal hypoglycemia and macrosomia, with

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Table 6. Comparison of pregnancy-related complications between the two groups

Complications	Excessive weight gain group (n=68)	Normal weight gain group (n=84)	χ^2 value	P value
Premature birth	10 (14.7)	4 (4.8)	4.652	0.031
Premature rupture of membranes	8 (11.8)	3 (3.6)	4.012	0.045
Cesarean section	28 (41.2)	22 (26.2)	4.126	0.042
Fetal distress	7 (10.3)	2 (2.4)	4.512	0.034
Postpartum hemorrhage	6 (8.8)	1 (1.2)	4.987	0.026

Table 7. Comparison of neonatal complications between the two groups

Complications	Excessive weight gain group (n=68)	Normal weight gain group (n=84)	χ^2 value	P value
Neonatal hypoglycemia	10 (14.7)	3 (3.6)	6.214	0.013
Newborns admitted to NICU	12 (17.6)	4 (4.8)	7.123	0.008
Full-term birth (≥ 37 weeks)	58 (85.3)	80 (95.2)	4.652	0.031
Natural childbirth	40 (58.8)	62 (73.8)	4.126	0.042

Note: NICU, neonatal intensive care unit.

long-term impacts on the long-term health of mothers and infants [9-11]. In recent years, with changes in lifestyles, the proportion of pregnant women experiencing excessive weight gain during early pregnancy has gradually increased. Studies have demonstrated an association between abnormal weight gain during early pregnancy and the development of GDM; however, the underlying mechanisms, influencing factors, and potential interactions between these factors still need further clarification [12].

Previous studies have reported a relatively high prevalence of GDM [13]. The results of this study showed that the overall prevalence of GDM among 152 women with singleton pregnancies was 23.0%, which was basically consistent with the incidence reported in previous studies conducted in China [14]. Further analysis showed that the incidence of GDM was significantly higher in the excessive weight gain group than that in the normal weight gain group (32.4% vs. 15.5%), suggesting that excessive weight gain during early pregnancy may be associated with an increased risk of GDM. In addition, OGTT results showed that 1-hour and 2-hour postprandial glucose levels in the excessive weight gain group were significantly higher than those in the normal weight gain group. These findings further support the potential adverse effects of excessive weight gain during early pregnancy on glucose metabolism.

The potential mechanisms may be as follows: First, excessive weight gain in early pregnancy may promote excessive adipose tissue accumulation and increase insulin resistance, which represents a key pathophysiological mechanism underlying GDM [15, 16]. Second, excessive adipose tissue accumulation may induce the release of pro-inflammatory cytokines (e.g., TNF- α , IL-6), thereby impairing insulin receptor signaling, reducing insulin-mediated glucose uptake and utilization, and contributing to elevated blood glucose levels and the development of GDM [17]. However, these proposed mechanisms were not directly validated in this study and require future investigations incorporating measurements of serum inflammatory biomarkers and placental function-related parameters.

Previous studies have demonstrated an intrinsic correlation between insulin resistance and body weight [18]. In the present study, insulin metabolism-related indicators were compared between the two groups of pregnant women. The results showed that fasting insulin level, HbA1c, and HOMA-IR were significantly higher in the excessive weight gain group than those in the normal weight gain group. HOMA-IR is a commonly used index for evaluating insulin resistance, and elevated levels reflect increased insulin resistance. HbA1c reflects average blood glucose levels over the preceding 2-3 months, and its elevation indicates impaired

long-term glycemic control. Elevated fasting insulin levels may represent a compensatory enhancement of insulin secretion in response to insulin resistance; however, when compensatory capacity becomes insufficient, blood glucose levels may increase. These findings are consistent with previous research conclusions [18-20].

The effects of potential risk factors on disease outcomes can be further evaluated using univariate and multivariate logistic regression analyses [21]. The results of this study showed that excessive weight gain during early pregnancy was significantly associated with an increased risk of GDM. After adjusting for potential confounding factors, including pre-pregnancy BMI and family history of GDM, excessive weight gain during early pregnancy remained independently associated with GDM. These findings highlight the potential role of early pregnancy weight management in GDM prevention and suggest that controlling excessive weight gain during early pregnancy may represent an important intervention strategy [22]. In the univariate analysis, although pre-pregnancy BMI and family history of GDM were not significantly correlated with GDM risk, they were included in the multivariate analysis because their *P* values were $P < 0.10$. The multivariate analysis showed that neither factor remained independently associated with GDM, which is consistent with the findings in previous report [23].

At the same time, subgroup and interaction analyses showed that excessive weight gain during early pregnancy was significantly associated with an increased risk of GDM across different age groups (< 35 years and ≥ 35 years) and pre-pregnancy BMI groups (< 24 kg/m² and ≥ 24 kg/m²). These findings suggest that the association between excessive weight gain during early pregnancy and GDM may be relatively consistent across different maternal age and pre-pregnancy BMI categories. Among pregnant women with a family history of GDM, the association between excessive weight gain and GDM appeared to be stronger, whereas no statistically significant association was observed among those without a family history of GDM. Interaction analysis revealed a marginally significant interaction between family history of GDM and excessive weight gain. These findings suggest that pregnant women with a family

history of GDM may have genetic susceptibility and increased vulnerability to metabolic disturbances induced by excessive weight gain during early pregnancy. This finding is consistent with previous studies [24, 25].

Previous studies have confirmed that excessive weight gain during early pregnancy may be associated with pregnancy complications [26]. The results of this study showed that excessive weight gain during early pregnancy was closely related to adverse pregnancy outcomes and neonatal complications. Specifically, the incidences of preterm birth, premature rupture of membranes, cesarean section, fetal distress, and postpartum hemorrhage were significantly higher in the excessive weight gain group than those in the normal weight gain group. In addition, the incidences of neonatal hypoglycemia and NICU admission were increased in the excessive weight gain group, whereas the rates of term birth and vaginal delivery were lower.

Several mechanisms may explain these associations. Excessive weight gain during early pregnancy may contribute to insulin resistance and impaired glucose metabolism, which may further promote vascular dysfunction and insufficient uteroplacental perfusion, thus increasing the risk of preterm birth and fetal distress. Meanwhile, poor glycemic control may contribute to excessive fetal overgrowth (macrosomia), increasing the risks of cesarean section, premature rupture of membranes, and postpartum hemorrhage. For newborns, maternal hyperglycemic environment may stimulate increased fetal insulin secretion; after birth, the abrupt interruption of maternal glucose supply may predispose neonates to hypoglycemia. Furthermore, fetal distress and macrosomia may increase the likelihood of NICU admission. These findings are consistent with previous studies showing that abnormal weight gain during early pregnancy is associated with an increased risk of adverse maternal and neonatal outcomes [27, 28].

This study has several limitations. First, this was a single-center, retrospective study with a relatively small sample size ($n=152$), and the post-hoc power analysis indicated a statistical power of 0.68 (< 0.80). Therefore, selection bias may exist, and the generalizability of the findings may be limited. Second, the grouping criteria require further validation. Cut-off value

for excessive weight gain (> 2.0 kg/week) was not directly derived from the IOM recommendations or domestic clinical guidelines, and its applicability requires verification. Third, several potential confounding factors were not available for analysis, including dietary intake, physical activity, and pregnancy-related nausea/vomiting. These factors may influence both gestational weight gain and glucose metabolism, and their absence may have resulted in residual confounding and biased effect estimates. Additionally, as this was a retrospective study, these variables were not routinely recorded in electronic medical records and could not be reliably retrieved, representing an inherent limitation of the study design. Fourth, mechanistic data were unavailable. Inflammatory markers (e.g., TNF- α , IL-6, CRP), adipokines (e.g., leptin, adiponectin), and placental function-related indicators were not measured; therefore, the potential biological mechanisms underlying the observed association could not be further elucidated. Fifth, the single-center design and lack of external validation limit the external validity of the findings. All participants were recruited from a single institution, and no independent external cohort verification was performed. This significantly limits the external validity and generalizability of the findings to broader populations. Sixth, residual confounding factors, such as socioeconomic status, psychological stress, and sleep quality, may persist. Finally, long-term follow-up data on maternal or offspring outcomes after pregnancy were unavailable. Therefore, the findings of this study should be considered exploratory and hypothesis-generating. Future prospective, multi-center, large-scale studies with comprehensive adjustment for potential confounders, external validation, and mechanistic investigation are warranted to confirm these findings and further clarify their clinical implications.

Conclusion

This study provides preliminary evidence that excessive weight gain during early pregnancy (> 2.0 kg/week) may be associated with an increased risk of GDM. However, due to the retrospective design, limited sample size, incomplete adjustment for potential confounding factors (including diet, physical activity, nausea/vomiting), and insufficient mechanistic evidence, causal relationships cannot be drawn.

Future prospective studies with adequate confounder adjustment are needed to validate these findings.

Clinical translational recommendations

Based on the findings of this study, the following preliminary recommendations are proposed (requiring validation in future prospective studies):

Intervention threshold: Weekly weight gain rate > 2.0 kg/week during early pregnancy as a warning threshold.

Monitoring protocol: Weekly weight measurement; OGTT screening when weight gain > 2.0 kg/week for two consecutive weeks.

Personalized management: For those with a family history of GDM, a stricter target (< 1.5 kg/week) and earlier OGTT screening (at 12 weeks).

Disclosure of conflict of interest

None.

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