



## Trabajo Original

### Implication of gestational diabetes treatment on maternal weight gain and low neonatal weight: a large retrospective cohort study

#### *Implicación del tratamiento de la diabetes gestacional en el aumento de peso materno y bajo peso neonatal: gran estudio de cohorte retrospectivo*

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### Abstract

**Objective:** the treatment for gestational diabetes is based on diet, and this may modify maternal weight gain. The limited maternal weight gain is related to newborns with small weight for their gestational age (SGA), and many studies have found an increase of SGA in women with gestational diabetes (GD), but the reason for this is not clear. The objective of this study is to evaluate the effects of gestational diabetes treatment on maternal weight gain and neonatal weight.

**Methods:** a retrospective cohort study of 1,765 patients with GD, according to the National Diabetes Data Group (NDDG) criteria. We assessed: pre-pregnancy BMI, total maternal weight gain (MWG), weight gain during the third trimester, gestational week of starting the treatment, and treatment modality (diet or diet plus insulin). Birth weight was adjusted by gestational age and gender: SGA ( $\leq 10^{\text{th}}$ ) and large for gestational age (LGA) ( $> 90^{\text{th}}$ ).

**Results:** the percentage of newborns with weight percentile  $\leq 10$  was 14.8%. The diet and the time of initiation of the treatment were related to maternal weight gain (MWG) in the third trimester. For every 1 kcal/kg of variation in the diet (increase or decrease), a MWG variation of 0.03 (0.001-0.06) kg occurred ( $p < 0.01$ ). For each week before the beginning of treatment, the mother did not gain  $0.13 \pm [(-0.15) - (-0.11)]$  kg in the third trimester ( $p < 0.01$ ). The SGA was related to the lowest MWG in total gestation: 7.0 (IQR 3.0-10.4) kg vs. 8.4 (IQR 5.0-11.6) kg ( $p < 0.01$ ), and in the third trimester: 0.3 (IQR -0.9-1.5) kg vs. 0.9 (IQR -0.3-2.2) kg ( $p < 0.01$ ).

**Conclusion:** the dietary treatment for gestational diabetes leads to a lower maternal weight gain and induces an impact on neonatal weight.

#### Key words:

Gestational diabetes.  
Birth weight.  
Pregnancy. Weight gain. Gestational age.

### Resumen

**Objetivo:** el tratamiento para la diabetes gestacional se basa en la dieta y esto puede modificar el aumento de peso materno. Un aumento de peso materno limitado está relacionado con recién nacidos con bajo peso para su edad gestacional (SGA). Muchos estudios han encontrado un aumento de niños con bajo peso en mujeres con diabetes gestacional, pero la razón de esto no está clara. El objetivo de este estudio es evaluar los efectos del tratamiento de la diabetes gestacional sobre el aumento de peso materno y el peso neonatal.

**Métodos:** estudio de cohortes retrospectivo en 1765 pacientes con diabetes gestacional, según los criterios de los National Diabetes Data Groups (NDDG). Evaluamos: IMC antes del embarazo, aumento de peso materno total (MWG), aumento de peso durante el tercer trimestre, semana gestacional de inicio del tratamiento y modalidad de tratamiento (dieta o dieta más insulina). El peso al nacer se ajustó por edad gestacional y género: SGA (percentil de  $\leq 10$ ) y grande para la edad gestacional (LGA) (percentil de  $> 90$ ).

**Resultados:** el porcentaje de recién nacidos con peso percentil de  $\leq 10$  fue del 14,8%. La dieta y el momento de inicio del tratamiento se relacionaron con el aumento de peso materno en el tercer trimestre. Por cada 1 kcal/kg de variación en la dieta (aumento o disminución) se produjo una variación de aumento del peso materno de 0,03 (0,001-0,06) kg ( $p < 0,01$ ). Por cada semana antes de inicio del tratamiento, la madre dejó de ganar  $0,13 \pm [(-0,15) - (-0,11)]$  kg en el tercer trimestre ( $p < 0,01$ ). El SGA se relacionó con un aumento de peso materno más bajo en el total de la gestación: 7,0 (IQR 3,0-10,4) kg vs. 8,4 (IQR 5,0-11,6) kg ( $p < 0,01$ ), y en el tercer trimestre: 0,3 (IQR -0,9-1,5) kg vs. 0,9 (IQR -0,3-2,2) kg ( $p < 0,01$ ).

**Conclusión:** el tratamiento dietético para la diabetes gestacional puede conducir a un menor aumento de peso materno y a su vez inducir un impacto en el peso neonatal.

#### Palabras clave:

Diabetes gestacional.  
Peso neonatal.  
Gestación. Ganancia de peso. Edad gestacional.

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## INTRODUCTION

GD is defined as the diabetes diagnosed in the second or third trimester of pregnancy that is not clearly either type 1 or type 2 diabetes (1).

GD has been associated with many adverse maternal and newborn outcomes, such as large for gestational age (LGA) (2-5). Many systematic reviews and meta-analyses have found that the treatment of GD reduces LGA births (6-8). It is not a questionable result, but several of these studies have shown an increase in the percentage of small for gestational age (SGA) infants (9-12). Thus, it seems that in some cases the treatment of GD had unexpected results.

One of the most important factors in neonatal weight is maternal weight gain during gestation, and this is related to the maternal diet. In fact, nutrition therapy is the cornerstone of treatment in GD management, and its modifications are used to achieve optimal glycemic control. However, the optimal diet (energy content, macronutrient distribution...) remains open to question. The Institute of Medicine (IOM) has published the most widely adopted guidelines for weight gain during pregnancy (13), but it is unclear to what extent they can be influenced by changes in diet and to what extent they in turn influence birth weight. There are several studies that have been carried out on pregnant patients without GD in which there is evidence of an increase in SGA in relation to diet restriction (14). However, there is no evidence of this fact in patients with gestational diabetes.

We conducted this study to evaluate gestational diabetes treatment effects in a large group of gestations complicated with GD, and to identify any weight adverse effects on the newborns.

## METHODS

The present retrospective study was conducted using data from our database that contains the records of all gestations complicated with diabetes. We selected women with a singleton pregnancy who had been diagnosed with gestational diabetes using the National Diabetes Data Group (NDDG) criteria from 1994 through to 2014.

At weeks 24–28 of gestation, women with no previous history of diabetes were assessed by means of the O'Sullivan test, after a 12 h fast. If they had any known risk factors for gestational diabetes, a screening test was performed in the first gestational trimester. When plasma glucose levels 1 h after glucose load were  $\geq 140$  mg/dL, a further 100 g OGTT was performed, and new glucose levels were measured while fasting, at 1, 2, and 3 hour intervals after intake. GD was diagnosed according to NDDG criteria: two or more glucose levels above the following: 105 mg/dL basal, 190 mg/dL in 1 h, 165 mg/dL in 2 h, and 145 mg/dL in 3h. The exclusion criteria were patients with other types of diabetes than GD, multiple gestation, delivery < 20 weeks, and incomplete follow-up to delivery.

The treatment consisted of physical activity and a balanced diet (50-53% carbohydrates, 18-21% protein, and 28-32% fat). Considering pre-pregnancy BMI, 45-50 kcal/kg was prescribed

in women with low weight (BMI < 18.5 kg/m<sup>2</sup>), 35-40 kcal/kg in women with normal weight (BMI 18.5-24.9 kg/m<sup>2</sup>), 30 kcal/kg in women who were overweight (BMI 25-29.9 kg/m<sup>2</sup>), and 20 kcal/kg in women with obesity (BMI  $\geq 30$  kg/m<sup>2</sup>). These recommendations are consistent with the 2009 IOM guidelines (13).

Therapy with insulin was commenced if optimal glycemic targets were not reached (fasting blood glucose level  $\geq 95$  mg/dL and/or 1h postprandial blood glucose level  $\geq 140$  mg/dL). Patients had to register blood glucose controls, and describe the previous intake if glucose levels were out of the target range.

We assessed:

1. *Maternal data*: age, pre-pregnancy weight (kg) (based on self-reported or first-registered weight at the beginning of gestation) and BMI (kg/m<sup>2</sup>), weight gain during the third trimester and the total gestational period, the gestational week of starting treatment, and treatment modality (diet or diet plus insulin).

Concerning diet, we assessed kcal per pre-gestational weight (kcal/kg) and the total diet prescribed (kcal). Delivery was categorized as eutocic or caesarean delivery.

2. *Neonate data*: gender, neonatal weight adjusted by gestational age and gender, and total neonatal weight (g).

Birth weights were categorized into small for gestational age ( $\leq 10^{\text{th}}$  percentile) and large for gestational age ( $> 90^{\text{th}}$  percentile) by reference to Carrascosa et al. (15).

## STATISTICAL ANALYSIS

Qualitative variables are presented with their frequency distribution. Quantitative variables are summarized as mean and standard deviation (SD) or median and interquartile range (p25-p75) in case of asymmetry.

The association is made between qualitative variables with the Chi<sup>2</sup> test or Fisher exact test, in the event that more than 25% of the expected quantity of fewer than 5 were evaluated.

The behavior of quantitative variables was analyzed for each of the independent variables categorized using Student's t-test (in comparisons of a variable with two categories) and/or ANOVA. By using this technique, mean differences due to the individual, or the main effect of each factor and/or the effect of their interactions, were evaluated. The significance level was corrected retrospectively (compared to peers) with the Bonferroni test. If there were asymmetry differences with the non-parametric test, the Mann-Whitney test or median (where appropriate) was evaluated.  $p < 0.05$  was considered statistically significant.

The software package used for the analysis was the SPSS for Windows.

## RESULTS

A total of 1,765 patients were included in the study with a mean age of  $32.5 \pm 4.3$  years (mean  $\pm$  SD). Table I shows the maternal characteristics.

**Table I. Maternal characteristics**

|   |              |
|---|--------------|
| Age (years)                                     | 32.5 ± 4.3   |
| Pregestational weight (kg)                      | 68.4 ± 14.7  |
| Height (m)                                      | 1.59 ± 0.06  |
| Pregestational BMI (kg/m <sup>2</sup> )         | 26.9 ± 5.4   |
| Week of gestation at treatment initiation       | 29.2 ± 5.9   |
| Diet (kcal)                                     | 2050 ± 164.8 |
| Diet, kcal/kg                                   | 31.4 ± 7.5   |
| Insulin (%) (n = 354)                           | 20.1         |
| Weight, start of 3 <sup>rd</sup> trimester (kg) | 75.6 ± 14.1  |
| Weight, end of 3 <sup>rd</sup> trimester (kg)   | 76.6 ± 13.9  |
| Total weight gain (kg)                          | 8.2 ± 5.3    |
| 3 <sup>rd</sup> trimester weight gain (kg)      | 1.0 ± 2.3    |

The mean pre-gestational BMI (mean ± SD) was 26.9 ± 5.4 kg/m<sup>2</sup>, distributed as follows: low maternal weight 1.1%, normal weight 38.9%, overweight 32.6%, and obese 21.6%.

The intervention was started at 29.2 ± 5.9 weeks of gestation. The number of kilocalories prescribed in the initial diet was 2,050 ± 164.8 kcal, with 31.4 ± 7.5 kcal/kg of pre-gestational weight.

According to the pre-gestational BMI, mean kcal/kg was: low weight: 49.1 ± 7.7 kcal/kg, normal weight: 37.2 ± 5.1 kcal/kg, overweight: 29.9 ± 3.7 kcal/kg, and obese: 22.3 ± 3.5 kcal/kg.

The mean total maternal weight gain during gestation was 8.2 ± 5.3 kg; during the third trimester it was 1.0 ± 2.3 kg.

During pregnancy, 20.1% of the women needed insulin therapy.

Concerning neonatal data, mean neonatal birth weight was 3,204.4 ± 531.6 g. In all, 14.8% of births were SGA (weight percentile ≤ 10), 18.4% were in a low weight percentile (p10-25), and 10.1% were LGA (weight percentile > 90). Mean gestational week at birth was 38.8 ± 2.1, and there were 27.1% cesarean interventions.

Concerning neonatal birth weight and preconception and pregnancy-associated factors, we observed the association with maternal pre-gestational BMI as follows: 3,033.4, 3,137.9, 3,238.9 and 3,306.9 g in patients with low weight, normal weight, overweight and obesity, with significant differences between groups (p < 0.05).

For SGA neonates, maternal weight gain was less than for non-SGA neonates, both in pregnancy as a whole (7.0 vs. 8.4 kg, p < 0.01) and during the third trimester: (0.3 vs. 0.9 kg, p < 0.01) (Table II).

Pre-gestational maternal BMI was lower in women who gave birth to SGA children than in the rest of women (25.9 ± 5.4 kg/m<sup>2</sup> vs. 27.1 ± 5.4 kg/m<sup>2</sup>, p < 0.01). Maternal weight at the beginning of the third trimester and at the end of pregnancy was also lower (70.9 ± 13.6 vs. 76.5 ± 14.0 kg, and 71.1 ± 13.5 vs. 77.5 ±

**Table II. Differences between neonatal percentile weight groups (≤ 10 and > 10 percentile) and maternal weight gain**

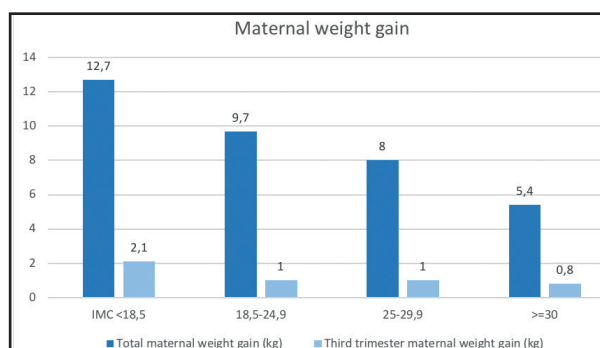
|   | Neonatal weight p ≤ 10 (n = 251; 14.8%) | Neonatal weight p > 10 (n = 1443; 85.2%) | (p)      |
|---|---|--|----------|
| Maternal total weight gain (kg)             | 7 (3-10.4)                              | 8.4 (5-11.8)                             | p < 0.01 |
| Maternal 3 <sup>rd</sup> T weight gain (kg) | 0.3 (-0.9-1.5)                          | 0.9 (-0.3-2.2)                           | p < 0.01 |

13.8 kg, respectively; p < 0.01). Fewer mothers with SGA children used insulin (10.0% vs. 16.1%, p < 0.01).

Treatment with insulin was associated with higher neonatal birth weight (3,267.6 vs. 3,188 g, p = 0.01) and birth by cesarean section (3,740.8 ± 500.1 vs. 3,116.5 ± 477.0, p < 0.01), possibly because increased fetal weight is one of the factors that encourage the use of insulin.

Related to maternal weight gain, we observed that, as regards the maternal pre-gestational BMI, there were significant differences between all groups in terms of median maternal weight gain over the entire pregnancy: low weight 12.3 kg (IQR: 11.1-15.1), normal weight 9.6 kg (IQR: 7.0-12.2), overweight 7.8 kg (IQR: 4.7-10.9), and obesity 5.0 kg (IQR: 1.5-9.1) (p < 0.01).

In third-trimester weight gain alone, differences were revealed between low-weight patients and the other groups (p < 0.01): low weight 2.1 kg (IQR: 1.5-2.8), normal weight 1.0 kg (IQR: -0.2-1.1), overweight 1.0 kg (IQR: -0.4-2.3), and obese patients 0.8 kg (IQR: -0.8-2.2) (Fig. 1).



**Figure 1.** Maternal weight gain according to maternal pregestational BMI during the total and third trimester gestation.

We observed a relationship between use of insulin and higher weight gain in the third trimester (1.5 kg (0.1-3.3) vs. 0.6 kg (-0.5-1.8),  $p < 0.01$ ), with no significant differences in total weight gain for the total gestational period.

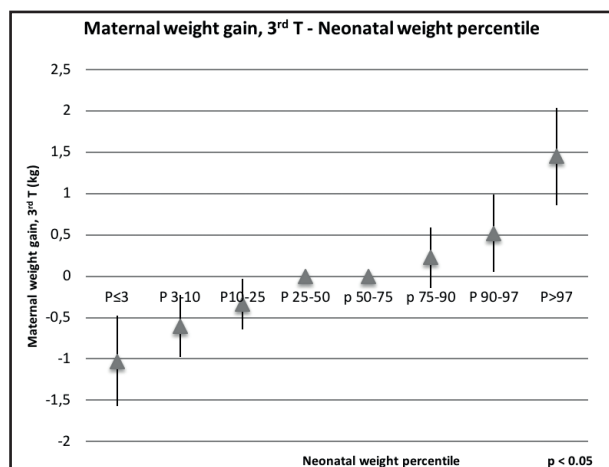
In the multivariate analysis of weight gain during the third trimester of gestation we found a relationship between diet and weight gain, with a variation of 0.03 kg per each 1 kcal/kg variation in the prescribed diet ( $p < 0.05$ ).

We also observed that for each week earlier that patient monitoring was started, patients failed to gain 0.13 kg during the third trimester ( $p < 0.01$ ).

Maternal weight gain during the third trimester was linked to the neonatal birth weight percentile, so that in the lowest weight percentile groups ( $p < 3$ , p3-10, p10-25) there was a difference in maternal weight gain of -1.03, -0.61, and -0.34 kg (respectively,  $p < 0.05$ ) compared to the weight gain seen in the mothers who gave birth to children in weight percentiles considered to be within the normal range (p25-75) (Fig. 2).

## DISCUSSION

Uncontrolled GD has a direct consequence on the fetus and increases the risk of macrosomia. Treatment leads to a decrease in birth weight (16). Today this is closer to 10% and thus similar to the general population. Some studies have evidenced a reduction in overall fetal weight linked to decreased macrosomia, which are valued as a positive effect of treatment as it lowers the incidence of complications in childbirth. On the other hand, there is the possibility of an increased risk of SGA infants that could be related to treatment (17,18). The increased incidence of SGA leads to more risk at birth (19) and is associated with late complications related to impaired motor development in children and increased cardiovascular risk in adults.



**Figure 2.**

Difference of maternal weight gain averages in the third trimester for each percentile of neonate weight relative to reference (percentile 26-75), adjusted for maternal BMI, kcal/kg of the prescribed diet, and gestational week at the intervention.

The prevalence of SGA (percentile  $\leq 10$ ) in our study was 14.8%; in the general population it is, or should be, 10%. This represents an increase of 48% above expectations. However, the prevalence of macrosomia is 10.1%, similar to the general population (15).

In recent studies, the prevalence of SGA in patients with GD reaches 16% or even 20% in some subgroups of patients. These are studies whose main objective was to evaluate fetal macrosomia, so they do not give an explanation of the findings on SGA (20-22).

Among the factors directly related to neonatal weight we included maternal weight progression and pre-pregnancy BMI.

1. It is known that maternal weight evolution plays a decisive role in fetal growth. In the absence of specific recommendations for gestational diabetes, IOM for general pregnant population recommendations are followed that take into account the pre-pregnancy BMI. The pre-pregnancy BMI in our sample was 26.9 kg/m<sup>2</sup>, with a total of 54.2% of patients who have a weight above normal.

The therapeutic intervention in our sample was at the beginning of the third trimester. Recent guidelines propose a caloric increase in the third trimester, so the prescription diet in our patients was consistent with these recommendations (13). Despite this, maternal weight gain during the third trimester was lower than the recommended level, coinciding with the start of the prescribed treatment.

There is little scientific evidence about this issue because most studies are directed towards good glycemic control to avoid the risk of macrosomia, not taking into account data on adverse effects such as weight gain or SGA (23).

Sugiyama et al. (24) found in their observational study of 1,615 patients with GD that the average weight gain during total pregnancy was just over 6 kg, and the prevalence of SGA was 16%. These results are consistent with those found in our work – that poor weight gain and a high percentage of SGA are linked. Surprisingly, they do not explain the possible reasons for the increase in the percentage of SGA.

Most studies analyze maternal weight gain in total gestation, but Barnes et al. (25) specify the progression of maternal weight during the third trimester. This was designed aiming to establish predictors of LGA ( $p > 90$ ) and SGA ( $p \leq 10$ ) in a population of 1,695 patients with GD. The mean weight gain in this work was 12.3 kg, but since the start of follow-up (average 28.1 weeks of gestation) average weight gain was only 1.7 kg. It is low compared with that recommended by the IOM and consistent with the evidence in our study. On analyzing the subgroup of women with BMI  $< 20$  and weight gain  $< 10$  kg during pregnancy, the percentage of SGA was 17%. In this work, as in our study, it is shown that low maternal weight progression conditions result in inadequate fetal weight gain, thereby increasing the incidence of children with SGA.

Stotland et al. (26), in their retrospective study on 20,465 non-diabetic patients, showed that a lower maternal weight gain of 7 kg is associated with an increased risk of children with SGA (OR 1.66, 95% CI 1.44-1.92). Other studies

among the Thai population (27), conducted on non-diabetic pregnant patients with normal pre-pregnancy weight, found that those who did not reach the goals of weight gain (less than 10 kg in total gestation) were at increased risk of SGA in the newborn. Several studies evidenced that patients with low weight gain are more likely to have children with SGA ( $p < 10$ ), which is consistent with the findings in our study population with gestational diabetes (11,28-32).

2. On the other hand, our work also shows a relationship between birth weight and maternal pre-pregnancy BMI in patients with gestational diabetes. Women with a lower BMI have children with lower weights, and a higher prevalence of SGA is found.

A meta-analysis conducted among pregnant women without gestational diabetes concluded that low pre-pregnancy maternal weight (defined by a BMI of less than 20 kg/m<sup>2</sup>) is related to newborns with SGA (33).

Black et al. (34) analyzed the impact of maternal pre-pregnancy BMI and maternal weight gain on fetal growth in a sample of women with and without a diagnosis of gestational diabetes (IADPSG criteria). They concluded that the prevalence of macrosomia is higher in women with higher pre-pregnancy BMI and greater weight gain during pregnancy, both in patients with and without gestational diabetes. It is true that this prevalence is higher in those with gestational diabetes.

Until now, the treatment of GD has been based on maternal glycemic control with diet and physical activity. In our work, among 1,765 patients with gestational diabetes, we show that maternal weight and BMI before pregnancy are very important on low fetal weight, specifically on the incidence of SGA.

These aspects have not been analyzed in other studies as primary objectives, and for this reason we consider the findings derived from our work to be important.

Although the number of patients included in our study is high, we assume that the fact that it is a retrospective study is a limitation, and therefore new work is necessary to support these conclusions.

Despite this, we consider that the results of our study are applicable to clinical practice. For the therapeutic control of type 2 diabetes mellitus a shift in focus is happening. It is moving from a glyco-centric vision to an adipocentric vision. It is not only hyperglycemia causing injury and requiring control as a single target, but also excess weight, and therefore adipose tissue that is central to its development. In view of the results we can apply this approach to patients with gestational diabetes. Thus, not all patients who meet some biochemical diagnostic criteria for gestational diabetes are at the same risk of macrosomia, or benefit from the same therapeutic strategy, and therefore would not be subjected to the same intensity of glycemic control. We consider that if one of the goals of gestational diabetes treatment is to prevent fetal macrosomia and obstetrical adverse events, the intensity of treatment should be determined not only by blood sugar levels but also by maternal pre-pregnancy BMI and weight progression during pregnancy.

Analyzing the potential benefits and risks of treatment for GD, some experts have argued that since the treatment is based on nutritional modifications and blood glucose monitoring exclusively in most cases, it poses little risk of harm to the mother and the fetus because it is a minimally invasive intervention. However, the diagnosis of GD increases the number of prenatal visits and work absences for the mother, besides the inconvenience and costs associated with regular monitoring of blood glucose. The literature suggests that not all patients diagnosed with GD have the same risk of macrosomia and do not therefore receive the same benefit from the prescribed treatment. If the patient has pre-pregnancy obesity, we can induce more limited weight gain (as recommended by the IOM guidelines), aimed at reducing the likelihood of fetal macrosomia, which may be appropriate only in this group of patients. However, those with low pre-pregnancy BMI and/or less weight gain during pregnancy will be those who obtain little benefit from treatment because their risk of macrosomia is already low. They may even experience a detrimental effect by increasing the percentage of SGA (35,36).

In our study, we found that the prevalence of macrosomia in patients with GD is similar to the general population, so we understand that treatment met the goal of reducing the risk of macrosomia effectively. However, maternal weight gain was very scarce and the prevalence of infants with SGA ( $p \leq 10$ ) was higher than expected. This has to do with a therapeutic intervention that, despite being consistent with the recommendations of the IOM, and being considered as "minimally invasive" measures, can have negative consequences. For this reason, and as a consequence of our study, we suggest modulating the intensity of treatment depending not only on glycemic control but also taking into account other factors related to fetal weight such as pre-pregnancy maternal BMI and maternal weight gain.

In conclusion, the dietary treatment for gestational diabetes leads to lower maternal weight gain and has an impact on neonatal weight being a risk factor for the development of SGA neonates. So we should consider other factors besides glycemic control when it comes to intensifying dietary treatment.

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