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Am J Obstet Gynecol. Author manuscript; available in PMC 2016 October 01.

Published in final edited form as:

Am J Obstet Gynecol. 2015 October ; 213(4): 563.e1–563.e5. doi:10.1016/j.ajog.2015.07.030.

Evaluating Gestational Weight Gain Recommendations in Pregestational Diabetes

Anne M. Siegel, MD[1], **Alan Tita, MD, PhD**[1], **Joseph R. Biggio, MD**[1], and **Lorie M. Harper, MD, MSCI**[1]

[1]Division of Maternal Fetal Medicine. Department of Obstetrics and Gynecology, Center for Women's Reproductive Health at the University of Alabama at Birmingham, Birmingham, AL

Abstract

Objectives—The Institute of Medicine (IOM) does not provide recommendations for gestational weight gain (GWG) specific to women with pregestational diabetes. We aimed to assess the impact of GWG outside the IOM recommendations on perinatal outcomes.

Study Design—We performed a retrospective cohort study of all singletons with pregestational diabetes from 2008–2013. Women were classified as GWG within, less than, or greater than IOM recommendations for body mass index per week of pregnancy. Maternal outcomes examined were cesarean delivery (CD), preeclampsia, and percentage of visits with glycemic control (>50% blood sugars at goal). Neonatal outcomes were birth weight, small for gestational age (SGA, <10th percentile), large for gestational age (LGA, >90th percentile), macrosomia (>4000 g), preterm delivery (PTD, <37 weeks), and birth injury (shoulder dystocia, fracture, brachial plexus injury, cephalohematoma). Groups were compared using analysis of variance and chi-squared test, as appropriate. Backwards stepwise logistic regression was used to adjust for confounding factors.

Results—Of 340 subjects, 37 (10.9%) were within, 64 (18.8%) less than, and 239 (70.3%) greater than IOM recommendations. The incidence of CD, preeclampsia, glycemic control, PTD, and birth injury were not significantly different between GWG groups. The incidence of LGA and macrosomia increased as GWG category increased AOR 3.08, 95% CI 1.13–8.39 and AOR 4.02, 95% CI 1.16–13.9 respectively) without decreasing the incidence of SGA (AOR 0.34, 0.10–1.19 CI 95%). Increases in the risk in LGA and macrosomia were not explained by differences in glycemic control by GWG groups.

Conclusion—Women with pregestational DM should be counseled to gain within the IOM recommendations to avoid LGA and macrosomic newborns.

Condensation

Women with pregestational diabetes gaining above the IOM recommendations had an increased risk of LGA and macrosomia without an associated decrease in SGA; women gaining below the IOM recommendations had no increased risk of adverse outcomes.

Corresponding author: Anne M. Siegel, The University of Alabama at Birmingham, Department of Obstetrics and Gynecology, Address: 1700 6th Ave South Ste 10270, Birmingham, AL 35233, Phone: 205-975-0515 Fax: 205-975-4375 asiegel@uabmc.edu.

The authors report no conflict of interest

Findings presented at the 35th Annual Society of Maternal Fetal Medicine Pregnancy meeting in San Diego, CA February 2–7th at a poster session.

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Keywords

Gestational Weight Gain; Pregestational Diabetes; Pregnancy; Type 1 diabetes; type 2 diabetes

Introduction

Pregestational diabetes complicates 1% of all pregnancies in the United States.[1, 2] The number of pregnancies complicated by diabetes is increasing;[3] the age-adjusted rate of pregestational diabetes doubled between 1996 and 2010.[3] Furthermore, currently one half of pregnant women are overweight or obese[4] and obesity increases the lifetime risk of diabetes by as much as 74% for women.[5,6]

Pregestational diabetes increases the risk for preeclampsia, primary cesarean, fetal anomalies, macrosomia, preterm delivery, stillbirth, and growth restriction. Maternal glycemic control can reduce the risk of these complications. Given the association between obesity, weight gain, and insulin resistance,[7] gestational weight gain in this population may contribute to adverse maternal and neonatal outcomes.

The Institute of Medicine (IOM) has developed guidelines for gestational weight gain to target an ideal birth weight[8]. However, these guidelines were developed in a healthy population and no specific guidelines were created for special populations such as pregestational diabetics. Gestational weight gain is directly linked to birth weight, which is in turn linked to mode of delivery and neonatal outcomes. Therefore, it is essential to evaluate these guidelines as this patient population continues to expand.

Consequently, we aimed to evaluate the effect of gestational weight gain in pregestational diabetics outside the IOM guidelines on perinatal outcomes. We predicted a high proportion of women will gain more than IOM recommendations and that these women will have more LGA neonates with a higher risk of cesarean delivery, preeclampsia, PTD, and birth injury.

Materials and Methods

This was a retrospective cohort study of all singleton pregnancies at a tertiary care center complicated by pregestational diabetes from 2008–2013. The study period was determined by the years when the complete electronic medical record was available for reliable data collection. Institutional review board approval was obtained from the University of Alabama at Birmingham.

Subjects were identified by a diagnosis of pregestational diabetes in our searchable electronic medical records. Subjects who reported a diagnosis of diabetes prior to pregnancy were considered to have pregestational diabetes; women diagnosed with diabetes at any point during pregnancy (even at early gestational ages) were not included in this study. Trained chart abstractors completed standardized chart abstraction forms and the principal investigator reviewed greater than 3% of all abstracted charts. Data collected included maternal demographics, medical and obstetrical history, diabetes diagnosis and care, prenatal blood sugar logs, medication use, labor and delivery events, and neonatal outcomes.

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Chart abstractors reviewed each patient's blood sugar logs for each visit and determined the number of values recorded, the number of values above goal for each visit, and the number of blood sugars <60mg/dL. All women were managed under the supervision of Maternal-Fetal Medicine specialists. Per institutional protocol, patients met with a nutritional counselor and diabetic educator at their initial visit and as needed throughout their pregnancy. Also per institutional protocal, patients were seen every 1–2 weeks and adjustments were made to insulin regimen when either >50% of the fasting or 50% of the post prandial blood sugars were elevated. Patients with fewer than 3 fasting or 7 post prandial blood sugars recorded were assumed to have poor control. Subjects were excluded for incomplete BMI data, last weight measured >14 days before delivery, major maternal comorbidity unrelated to DM (e.g. systemic lupus erythematosus, maternal cardiac disease, HIV), late prenatal care (>26w at first prenatal visit), and any fetal anomalies.

Gestational weight gain per week of the second and third trimesters was calculated as: (last measured weight-prepregnancy weight)/(gestational age at delivery-13), assuming a 0.5–2 kg weight gain in the first trimester. Women were classified as GWG within, less than, or greater than the IOM recommendations for prepregnancy body mass index (BMI). Prepregnancy BMI was determined by patient report of prepregnancy weight which has been established as a validated method of obtaining biometric data[9].Per week of pregnancy in second and third trimesters, the IOM recommends underweight women (BMI <18.5 kg/m^2) gain 0.44–0.58 kg/week, normal weight women (BMI 18.5–24.9 kg/m^2) gain 0.35–0.50 kg/week, overweight women (BMI 25.0–29.9 kg/m^2) gain 0.23–0.33 kg/week and obese women (BMI >= 30 kg/m^2) gain 0.17–0.27 kg/week.

Maternal outcomes examined were mode of delivery, preeclampsia, and percentage of visits with glycemic control. To assess the impact of gestational weight gain on glycemic control, we assessed patients blood sugar logs. For each visit, a patient was considered to have glycemic control if ≥50% of blood sugars were at goal. In order to account for variations in the number of visits each patient has during pregnancy, we calculated the percentage of visits with glycemic control by dividing the number of visits with glycemic control by the total number of visits. Neonatal outcomes examined were birth weight, small for gestational age (SGA, <10th percentile on Alexander standard),[10] large for gestational age (LGA, >90th percentile on Alexander standard), macrosomia (>4000 g), preterm delivery (PTD, <37 weeks), gestational age at delivery, birth injury (defined as shoulder dystocia as documented by the delivery physician, fracture of the clavicle, humerus, or skull, brachial plexus injury, and cephalohematoma), and neonatal length of stay.

A secondary analysis was performed to measure the impact of gestational weight gain on the adverse maternal and neonatal outcomes stratified by type of diabetes (type 1 or type 2 diabetes) and by obesity. Type 1 and type 2 diabetics were determined by history in the medical record. Women exclusively treated with oral agents were determined to be type 2 diabetics. If the subjects' type was never documented, they were labeled as an unknown type and excluded from the secondary analysis.

Normal distribution of continuous variables was tested using the Kolmogorov-Smirnov test and by visually assessing the histograms. The characteristics and outcomes of subjects

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gaining within, less than, or greater than the IOM recommendations were compared using analysis of variance or Kruskall-Wallis test and chi-squared test for trend, as appropriate. Clinically relevant covariates for initial inclusion in multivariable statistical models were selected using results of the stratified analyses and factors were removed in a backward step-wise fashion based on significant changes in the exposure adjusted odds ratio or significant differences between hierarchical models using likelihood ratio test. Confounding factors considered include age, race, parity, and prior mode of delivery, hypertension and tobacco use. All analyses were completed using Stata SE, version 11.2 (College Station, TX).

Results

Of 597 women identified with pregestational diabetes, 340 were included in the analysis (62 excluded for major medical problems unrelated to diabetes, 51 excluded for congenital malformations, 9 excluded for late prenatal care, 30 missing prepregnancy BMI, and 105 with missing final weight or weight >14 days prior to delivery). Of these 340 women, 37 (10.9%) gained within the IOM recommendations, 64 (18.8%) less than IOM recommendations, and 239 (70.3%) more than the IOM recommendations. Maternal characteristics according to gestational weight gain group are shown in Table 1. Women in each gestational weight gain category were similar with regards to maternal age, race, insurance type, chronic hypertension, prior cesarean delivery, parity, White's classification, and type of medication regimen. Women gaining less than the IOM recommendations had a higher prepregnancy BMI compared to the within and more than the IOM recommendations groups (Table 1 p value 0.021).

Mode of delivery and glycemic control were not significantly different between GWG groups (Table 2). The risk of preeclampsia was not significantly different between GWG groups. As GWG category increased, the risks of LGA and macrosomia increased without a concomitant significant decrease in the incidence of SGA. These results remained statistically significant after controlling for chronic hypertension, type 1 versus type 2 diabetes and prepregnancy BMI (Table 3).

Stratifying the exposure groups by type of diabetes demonstrated a similar relationship between gestational weight gain category and perinatal outcomes. When type 2 diabetics were considered independently, significant increases in both LGA and macrosomia with weight gain greater than the IOM recommendations were seen ($p < 0.01$ for LGA and $p=0.02$ for macrosomia). Additionally, type 2 diabetics showed a trend towards an increased risk of CD with weight gain above IOM recommendations although this failed to reach statistical significance ($p=0.43$). Similarly, when type 1 diabetics were considered independently, there was an association with LGA ($p =0.02$) with weight gain above IOM recommendations. Obese patients were also found to have a statistically significant increased association with LGA ($p=0.04$) with GWG above IOM recommendations when considered independently

Comment

In this large cohort of pregestational diabetics, gestational weight gain above the IOM recommendations was associated with an increased risk of LGA, macrosomia, and mean

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birth weight. However, weight gain greater than the IOM recommendations was not associated with a significant decrease in the risk of SGA. The incidences of cesarean delivery, preeclampsia, and glycemic control were not significantly different between GWG groups. Interestingly, the increased risks of LGA and macrosomia were not explained by differences in glycemic control between groups.

Our data is supported by similar studies in the literature of pregnancies complicated by insulin resistance (type 2 and gestational diabetes), which also demonstrate an association between excessive GWG and LGA infants[11–14]. Additionally, our data supports previous work by Harper et al.[14] by demonstrating that gaining more than IOM recommendations is not protective for SGA infants.

Egan et al[15] investigated an association between GWG measured as a per week gain in the second and third trimesters and adverse perinatal outcomes in pregnancies complicated by diabetes mellitus with a prospective cohort study. They also found an increased risk of LGA and macrosomia. However, this study also included gestational diabetics and did not evaluate for adverse fetal and neonatal effects such as PTD, stillbirth, or birth injury.

Scifres et al[16] also evaluated the effects of excess gestational weight gain on birth weight and other pregnancy outcomes in women with type 1 DM only. Their study also found an association between excessive maternal weight gain and LGA infants consistent with our findings.

We recognize several limitations of our study. First, as women cannot be randomized to gain a certain amount of weight, studies examining weight gain in pregnancy are necessarily limited to observational data. Also given the study design, we were required to exclude many subjects' data and may have introduced a selection bias. However, analyses of subjects excluded from the analysis were not significantly different than those included in the analysis by age, race, prepregnancy body mass index, parity, or type of diabetes. Additionally, our study had a limited number of subjects with each type of diabetes to fully determine any differences in maternal and neonatal events. We also had few patients gaining within and below the recommended weight. A larger cohort of patients in these categories may have allowed further analysis to determine significant adverse pregnancy outcomes for patients with insufficient weight gain or if lower gestational weight gain is actually preferred in diabetic pregnancies. Finally, the vast majority of our patients were obese and overweight. Although we considered BMI-specific recommendations, our findings may not be generalizable to underweight and normal weight women.

The detailed chart abstraction of the patients in our cohort was one of the strengths of the study. Information regarding diagnosis of diabetes, medications, maternal weight, and glycemic control were reviewed as well as neonatal outcomes. Additionally, our study included the calculation for GWG category per week instead of weight gain as a whole. Given that longer gestations will inherently have greater weight gain, considering gestational weight gain per week controls for length of gestation, an important factor in this type of study.

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Our study demonstrates that gaining above the IOM recommendations increases the risk of LGA and macrosomia among women with pregestational diabetes without decreasing the risk of SGA. Women with pregestational diabetes should be counseled not to gain more than the IOM recommendations; further research is needed to determine if women with pregestational diabetes should gain less than the IOM recommendations.

Acknowledgments

Dr. Harper is supported by K12HD001258-13, PI WW Andrews, which partially supports this work.

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Table 1

Maternal Characteristics by Gestational Weight Gain Group

	Less than IOM Guidelines N=64	Within IOM guidelines N=37	Above IOM guidelines N=239	P value
Age (yrs)	29.8 ± 6.3	27.6 ± 6.6	29.6 ± 6.1	0.16
Nulliparous	28 (43.8)	13 (35.1)	92 (38.5)	0.65
Race				
White	14 (21.9)	10 (27.0)	81 (33.9)	0.26
Black	47 (73.4)	26 (70.3)	133 (55.6)	
Hispanic	3 (4.7)	1 (2.7)	20 (8.4)	
Government Insurance	43 (67.2)	25 (67.6)	162 (67.8)	0.88
Smoking	15 (23.4)	10 (27.0)	51 (21.3)	0.72
Prior C/S	13 (20.3)	11 (29.7)	68 (28.5)	0.40
cHTN	29 (45.3)	11 (29.7)	96 (40.2)	0.30
White Classification				
B	21 (32.8)	11 (29.7)	63 (26.4)	0.43
C	9 (14.1)	9 (24.3)	44 (18.4)	
D	31 (48.4)	12 (32.4)	107 (44.8)	
R, F, RF	3 (4.7)	5 (13.5)	25 (10.5)	
Diabetes Type				
Type 1	14 (21.9)	14 (37.8)	68 (28.5)	0.39
Type 2	48 (75.0)	22 (59.5)	167 (69.9)	
Unknown	2 (3.1)	1 (2.7)	2 (0.8)	
Type I DM Insulin Pump Use	5 (7.8)	4 (10.8)	24 (10.0)	0.84
Prepregnancy BMI (kg/m^2)	37.5 ± 9.8	33.8 ± 10.4	33.9 ± 9.0	0.021
Prepreg BMI category				
Underweight	1 (1.6)	0 (0)	1 (0.4)	0.07
Normal	5 (7.8)	10 (27.0)	28 (11.7)	
Overweight	10 (15.6)	7 (18.9)	54 (22.6)	
Obese	48 (75.0)	20 (54.1)	156 (65.3)	
Gestational weight gain (kg)	0.27 ± 5.9	7.2 ± 2.0	17.74 ± 9.6	<0.01

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	Less than IOM Guidelines N=64	**Within IOM guidelines N=37**	**Above IOM guidelines N=239**	**P value**
Oral Medication Used During Pregnancy (with or without insulin)[‡]	14 (21.9%)	14 (37.8%)	61 (25.5%)	0.20
Oral Medication Only (No insulin)[‡]	7 (11.3%)	5 (13.4%)	18 (7.8%)	0.39

-Values reported as absolute number of subjects in that GWG category within each parameter with a percent of patients in parentheses.

-Values also reported as mean ± standard deviation.

[‡]Oral medications used were either glyburide or metformin, approximately evenly divided.

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Table 2

Maternal and Neonatal Outcomes Associated with Gestational Weight Gain

	Less than IOM guidelines N=64	Within IOM guidelines N=37 (Reference Group)	Above IOM guidelines N=239	P value (trend)
Maternal				
Mode of Delivery				
Spontaneous	28 (43.8%)	19 (51.4%)	88 (36.8%)	
Operative Vaginal *	3 (4.7%)	0	10 (4.2%)	
Cesarean Delivery	33 (51.6)	18 (48.7)	141 (59.0)	0.33
Preeclampsia	15 (23.4)	12 (32.4)	73 (30.5)	0.49
Percent of visits with Blood Sugar Controlled (%)	31.8 +/− 28.9	28.9 +/− 26.8	29.7 +/− 26.7	0.92
Neonatal				
Birth weight (g)	2787 ± 990	3198 ± 734	3296 ± 1109	<0.01
SGA	5 (7.8)	4 (10.8)	13 (5.4)	0.41
LGA	5 (7.8)	6 (16.2)	72 (30.1)	<0.01
Macrosomia	4 (6.3)	4 (10.8)	56 (23.4)	<0.01
PTD	30 (46.9)	10 (27.0)	92 (38.5)	0.14
Gestational Age at Delivery (wks)	35.5 +/− 4.6	37.4 +/− 2.2	36.4 +/− 4.0	0.24
Birth Injury †	7/60 (11.7)	3/36 (8.3)	17/222 (7.7)	0.61
Neonatal Length of Stay (days)	9.1 +/− 14.5	4.9 +/− 7.7	7.6 +/− 12.1	0.27

--Values reported as absolute number in that GWG category with the associated outcome with percent of patient in parentheses or as mean ± standard deviation.

*Operative vaginal delivery includes vacuum- and forceps-assisted vaginal delivery. Of the 3 operative vaginal deliveries in the less than IOM guidelines, 1 was vacuum and 2 were forceps. In the 10 operative vaginal deliveries in the more than IOM guidelines, 4 were vacuum and 6 were forceps.

†Reported as n/total included (%). The total number included in the birth injury analysis is less than in the category due to stillbirths.

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Table 3

Adjusted Odds Ratio For Maternal and Neonatal Outcomes associated with Gestational Weight Gain

	AOR of outcome for weight gain less than IOM recommendations (95% CI)	**AOR of outcome for weight gain above IOM recommendations(95% CI)**
Maternal		
Cesarean Delivery	1.59 * (0.61–4.19)	1.72 * (0.77–3.82)
Preeclampsia	0.42 † (0.05–3.91)	1.70 † (0.34–8.43)
Neonatal		
SGA	0.51 † (0.10–2.61)	0.34 † (0.10–1.19)
LGA	0.47 † (0.11–1.96)	3.08 † (1.13–8.39)
Macrosomia	0.74 † (0.14–3.93)	4.02 † (1.16–13.9)
PTD	2.41 ‡ (0.91–6.37)	1.71 ‡ (0.76–3.83)

Weight Gain within IOM recommendations was the reference group to calculate the AOR

* Adjusted for prior cesarean, nulliparity, type of diabetes

† Adjusted for chronic hypertension, type of diabetes, prepregnancy BMI

‡ Adjusted for chronic hypertension, type of diabetes, and prior preterm delivery