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Perinatal Outcomes in Hispanic and Non-Hispanic White Women With Mild Gestational Diabetes

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Abstract

OBJECTIVE—To compare perinatal outcomes between self-identified Hispanic and non-Hispanic white women with mild gestational diabetes mellitus (GDM) or glucose intolerance.

METHODS—In a secondary analysis of a mild GDM treatment trial, we compared perinatal outcomes by race and ethnicity for 767 women with glucose intolerance (abnormal 50g 1-hour screen, normal 100g 3-hour oral glucose tolerance test [OGTT]), 371 women with mild GDM assigned to usual prenatal care, and 397 women with mild GDM assigned to treatment. Outcomes included: composite adverse perinatal outcome (neonatal death, hypoglycemia, hyperbilirubinemia, hyperinsulinemia; stillbirth; birth trauma), gestational age at delivery, birthweight, and hypertensive disorders of pregnancy. Adjusted regression models included: 100g 3-hour OGTT results; parity; gestational age, body mass index, maternal age at enrollment; and current tobacco use.

RESULTS—The sample of 1535 women was 68.3% Hispanic and 31.7% non-Hispanic White. Among women with glucose intolerance, Hispanic women had more frequent composite outcome (37% vs. 27%, aOR 1.62 95%CI 1.10, 2.37), with more neonatal elevated C-cord peptide (19% vs. 13%, aOR 1.79 95%CI 1.04, 3.08) and neonatal hypoglycemia (21% vs. 13%, aOR 2.04 95%CI 1.18, 3.53). Among women with untreated mild GDM, outcomes were similar by race/ethnicity. Among Hispanic women with treated mild GDM, composite outcome was similar to non-Hispanic

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White women (35% vs. 25%, aOR 1.62 95% CI 0.92, 2.86), but Hispanic neonates had more frequent hyperinsulinemia (21% vs. 10%, aOR 2.96 95%CI 1.33, 6.60).

CONCLUSION—Individual components of some neonatal outcomes were more frequent in Hispanic neonates, but most perinatal outcomes were similar between Hispanic and non-Hispanic ethnic groups.

Introduction

Gestational diabetes (GDM) complicates 4–7% of all pregnancies, and its prevalence continues to rise in parallel with the evolving obesity epidemic.(1) Hispanic women in the United States have a higher prevalence of GDM compared with Caucasian or African-American women.(2–5) Across racial/ethnic groups, hyperglycemia increases risks of adverse perinatal outcomes, including large for gestational age (LGA), shoulder dystocia, cesarean delivery, and hypertensive disorders of pregnancy.(6, 7) Strict glycemic control decreases risks of these outcomes.(8)

Whether the risk of adverse outcomes associated with mild GDM differs by race/ethnicity, however, is less clear. Among those without GDM, Hispanic women experience lower neonatal mortality, are less likely to deliver low birthweight infants,(9, 10) and have had a lower reported prevalence of pre-eclampsia, compared with Caucasians and African-Americans.(11) While some data suggest adverse outcome risk may also vary by race/ethnicity among women with GDM,(5, 12–14) findings are not consistent. . Thus, whether differences are specific to GDM or related to race/ethnicity independent of hyperglycemia is uncertain.

To address this uncertainty, we analyzed a large cohort of women with glucose intolerance, and untreated and treated mild GDM, but not overt diabetes of pregnancy, and measured differences in perinatal outcomes by race/ethnicity.

Materials and Methods

We performed a secondary analysis of the *Eunice Kennedy Shriver* National Institute of Child Health and Human Development (NICHD) Maternal-Fetal Medicine Units (MFMU) Network multi-site randomized clinical trial for the treatment of mild GDM(15) and compared perinatal outcomes by self-reported race/ethnicity. We compared women self-reported as Hispanic (including Mexican-American, Central/South American, or Caribbean) with women self-reported as White (and not Mexican-American, Central/South American, or Caribbean). A total of eight women self-reported their race/ethnicity as Hispanic as well as: African-American or Native American/Alaskan; these eight women were included in the Hispanic group.

Women were eligible if they had a 1-hour 50-g glucose load test result between 135 and 200 mg/dL between 24 0/7 weeks and 30 6/7 weeks' gestation. They were excluded if they had pre-gestational diabetes; an abnormal glucose screening test prior to 24 weeks' gestation; a history of GDM, stillbirth, multi-fetal gestation, asthma, or chronic hypertension; if taking corticosteroids; or if imminent preterm delivery was anticipated. The original study sample and randomization process has been further described previously.(15) In initial randomization, eligible women with an elevated 50g 1-hr glucose load result underwent a 100g 3-hr OGTT, and these results diagnosed mild GDM. Women were randomized to treatment vs. no treatment, matched for race/ethnicity and body mass index (BMI < vs. > 27 mg/kg^2). Women with normal OGTT results were enrolled as the observational cohort. The primary composite adverse perinatal outcome for the original study was occurrence of one or more of the following: perinatal mortality (stillbirth or perinatal death), hypoglycemia,

hyperbilirubinemia, elevated cord blood C-peptide level, or birth trauma.(15) The sample size for the current study was constrained by the sample size of the original trial. The original study was approved by the institutional review board at each participating center, and all participating women provided written informed consent.

A total of 1889 women were enrolled in the original study, including 1535 who were either Hispanic and non-Hispanic White. These 1535 women were classified into one of three groups in the parent study: 1) 767 women with glucose intolerance with an elevated 50g 1-hour screening test (≥135, but <200 mg/dL) but normal 3-hour oral glucose tolerance test (OGTT), matched to the randomized patients by clinical center, race/ethnicity, and body mass index (BMI); 2) 371 women with mild GDM as diagnosed by a fasting glucose <95 mg/dL but two or more 100g 3-hour oral glucose tolerance test (OGTT) results at or above established thresholds(16) who were randomized to no treatment; and 3) 397 women also diagnosed with mild GDM by the same criteria as above but who were randomized to treatment. Women were randomly assigned to no treatment versus treatment (nutritional counseling and diet therapy, with insulin if required) by simple urn method(17) as previously described.(15)

We report overall and individual components of the composite outcome: hyperbilirubinemia, elevated cord blood C-peptide, and hypoglycemia. Perinatal mortality and birth trauma are not included in individual analyses due to small numbers for each.(15) We also evaluated gestational age at delivery (weeks' gestation, preterm birth <37 weeks), birthweight (grams, small for gestational age, large for gestational age, macrosomia >4000g), either gestational hypertension or pre-eclampsia, and neonatal intensive care unit (NICU) admission. Identification of large for gestational age used growth standards that accounted for race and Hispanic ethnicity, as in the original study.(18) Study personnel abstracted pertinent data for enrolled women and their newborns at time of hospital discharge.

We compared baseline demographic characteristics and perinatal outcomes by Hispanic versus non-Hispanic White race/ethnicity. We used Student's t-test for continuous variables and chi-square or Fisher's exact test for categorical variables. For each perinatal outcome, we performed multiple linear or logistic regression analysis for continuous and categorical outcomes, respectively. All adjusted models included: fasting, 1, 2, and 3-hour results of 100 g 3-hour OGTT; parity; gestational age at enrollment; pre-pregnancy body mass index; maternal age at enrollment; and tobacco use in current pregnancy.

Statistical analyses were conducted with SAS software (SAS Institute, Cary, NC). A nominal two-sided P value less than 0.05 was considered to indicate statistical significance and no adjustments were made for multiple comparisons.

Results

Our sample of 1535 women was 68.3% Hispanic and 31.7% non-Hispanic White. Race/ethnicity was similar (p=.90) among the three cohorts. Hispanic and non-Hispanic White women in the treated mild GDM cohort were equally likely to receive insulin (1.2% vs. 2.3%, p=0.09).

Demographic characteristics by race/ethnicity are shown in Table 1. Results of GDM screening (50g 1-hour glucose load) and diagnostic (100g 3-hour oral OGTT) tests are shown in Table 2. For all three cohorts, 50 g 1-hour oral glucose load results were similar by ethnicity. In the glucose intolerant cohort, Hispanic women had higher 100g 3-hour OGTT results at 1 hour (156.3 vs. 151.2 mg/dL, p=0.006) and 3 hours (111.6 vs. 105.3 mg/dL, p=0.0002). In the untreated mild GDM cohort, Hispanic women had higher 100g 3-hour OGTT results at 3 hours (136.7 vs. 128.6 mg/dL, p=0.02). In the treated mild GDM cohort,

Hispanic women had greater 100g 3-hour OGTT results at time of fasting (86.9 vs. 85.5 mg/dL, p=0.04) and 100g 3-hour OGTT results at 3 hours (140.3 vs. 133.3 mg/dL, p=0.02).

Results for women in the glucose intolerant cohort are shown in Table 3. Hispanic women had more frequent composite perinatal outcome. The observed increased risk of composite perinatal outcomes among Hispanic neonates is driven by their increased risk of hyperinsulinemia and hypoglycemia. Gestational age at delivery was greater among Hispanic women, but mean gestational age was greater than 39 weeks for both groups. Other secondary perinatal outcomes were similar.

Results for women with untreated mild GDM are shown in Table 3. Non-Hispanic White and Hispanic women had similar prevalence of the composite perinatal outcome and each of the three reported outcome components is similar between groups. As in the glucose intolerant cohort, Hispanic women with untreated mild GDM delivered at a greater gestational age, but this difference was less than four days. Other secondary outcomes were similar between groups.

Results for women in the treated mild GDM cohort are shown in Table 3. Composite neonatal outcome prevalence did not differ between groups. The 10% greater prevalence of the composite outcome, equal to the prevalence difference in the glucose intolerant cohort, is driven by the increased risk of elevated cord C-peptide among Hispanic women. As in the other two cohorts, Hispanic women with untreated mild GDM delivered at a greater gestational age, but this difference was less than four days. Other secondary outcomes were equally likely between groups.

Mexican-American women comprised 75% of all women classified as Hispanic. Significant differences in the composite outcome reported among all Hispanic women, compared with non-Hispanic White women, remained significant when only Mexican-American women were compared to non-Hispanic White women. Outcomes among the smaller subsets of Central or South American women and Caribbean women, also classified as Hispanic, compared with non-Hispanic White women, were not different.

Discussion

Among women with glucose intolerance and with treated or untreated mild GDM, Hispanic neonates were more likely to experience adverse neonatal outcomes, although not in a uniform pattern, and maternal outcomes did not differ by race/ethnicity. Our data do not support racial/ethnic tailored diagnostic thresholds, as suggested in reports of women with overt GDM.

Our findings differ from other published data that illustrate the Hispanic Paradox, a term to describe the better-than-expected outcomes among a socioeconomically disadvantaged racial/ethnic group, more likely to live in poverty than their non-Hispanic White counterparts, and more similar to African-American than non-Hispanic populations.(19) While GDM prevalence is higher among Hispanic women, other important outcomes such as low birthweight remain lower.(20–22) Others(3, 11, 14, 23) have reported on the differences in hyperglycemia-associated adverse outcomes among infants of Hispanic women with GDM, when compared with other races/ethnicities.

Results have been inconsistent. One report found that, when compared with Caucasian women, Hispanic women were more likely to achieve glycemic control with diet alone in one large cohort.(14) Data from another large cohort reported that Hispanic women were more likely to require medical management of their GDM.(23) Hispanic women in the latter

cohort had less frequent hypertensive disorders of pregnancy and fewer preterm births, but more frequent shoulder dystocia, when compared with Caucasian women.

Retrospective analyses without a non-GDM group or groups with varying degrees of hyperglycemia have limitations. Inconsistent differences in adverse outcomes by race/ethnicity may be specific to GDM pathophysiology or mechanisms of insulin resistance, as has been proposed. A recent analysis of statewide birth certificate data suggested the effect size of racial/ethnic differences in outcomes among GDM are not large enough to support racial/ethnic specific GDM diagnosis and treatment thresholds.(24) Our results support this conclusion, and our analysis was able to overcome some limitations of these prior studies.

A primary strength of our study was our ability to evaluate racial/ethnic differences among women with mild hyperglycemia. These women are at increased risk of adverse outcomes but not diagnosed or treated by current diagnostic thresholds. The randomization of women with mild GDM in the original trial allowed us to evaluate racial/ethnic differences in women who are treated and not treated. A cohort of women with glucose intolerance but no GDM allowed us to compare racial/ethnic differences even among women with hyperglycemia below the most inclusive thresholds warranting treatment. As only women in the treated mild GDM cohort were eligible for insulin and the overall proportion of women receiving insulin was low and similar by race/ethnicity, treatment type did not likely bias our results. Finally, a large sample size and rigorously collected prospective data allowed us to examine several relative perinatal outcomes and consider multiple potential confounders in adjusted analyses.

This secondary analysis also has limitations. While our study population was over two-thirds Hispanic, it remains a heterogenous group, and data on women's culture of origin are limited. Length of time in the United States and acculturation, measured by English proficiency, may minimize the paradoxical improved health seen in Hispanic women.(9, 22) Our data did not include measures of acculturation, so we were unable to evaluate this potential confounder. While our reported statistical differences only persist when Mexican women are compared with non-Hispanic White women, this is likely due to small numbers of women from other countries. Without adequate numbers to power such a sub-analysis, these data are exploratory only. Larger numbers of South or Central American or Caribbean women would be required to evaluate these differences.

In addition, the primary trial was not powered to the outcomes used in this secondary analysis, and lack of statistical significance may represent a beta error. For example, the difference in primary composite outcome is significant by race/ethnicity in the glucose intolerant groups. However, while the prevalence of this outcome is mirrored among Hispanic and non-Hispanic White women in the treated mild GDM cohort, this is not statistically significant.

Despite these limitations, this secondary analysis of a large, prospective, randomized controlled trial of mild GDM demonstrates that most perinatal outcomes were similar between Hispanic and non-Hispanic ethnic groups. Only individual components of neonatal outcomes were more frequent in Hispanic neonates.

Growing support to implement more inclusive GDM diagnostic criteria will further increase GDM prevalence. A substantial proportion of women will still have some degree of hyperglycemia but not be diagnosed and treated for GDM. The overall high prevalence of adverse outcomes among these women without overt GDM, however, highlights an area for research. Additional efforts may target at-risk women with hyperglycemia, but not overt GDM, for intervention and treatment, regardless of race/ethnicity. Our findings suggest that diagnostic criteria tailored to race/ethnicity may not be warranted, at least not among women

with mild GDM or glucose intolerance. If tailored treatment to optimize glycemic control among Hispanic women are pursued, we would suggest using specific neonatal diagnoses – such as C-cord peptide or neonatal hypoglycemia - as study outcomes.

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Table 1

Demographic Characteristics Comparing Hispanic and Non-Hispanic White Race and Ethnicity for Each of Three Study Cohorts

	Glucose Intolerant (n=767)			Mild Untreated Gestational Diabetes Mellitus (n=371)			Mild Treated Gestational Diabetes Mellitus (n=397)		
	Hispanic 520 (68)	Non-Hispanic White 247 (32)	*P*	Hispanic 255 (69)	Non-Hispanic White 116 (31)	*P*	Hispanic 274 (69)	Non-Hispanic White 123 (31)	*P*
Maternal age at enrollment (yrs)	27.6 ± 5.4	28.0 ± 5.4	0.29	29.5 ± 5.6	28.5 ± 5.0	0.08	29.5 ± 5.7	29.2 ± 5.9	0.67
Primigravida	136 (26.2)	107 (43.3)	<.001	75 (29.4)	48 (41.4)	0.02	55 (20.1)	55 (44.7)	<0.001
Smoking	16 (3.1)	48 (19.4)	<0.001	4 (1.6)	17 (15.0)	<0.001	4 (1.5)	22 (17.9)	<0.001
Body mass index at entry	30.1 ± 4.5	29.5 ± 5.3	0.11	30.2 ± 4.3	30.6 ± 6.2	0.51	30.3 ± 4.4	29.7 ± 5.5	0.29
Gestational age at enrollment (wks)	28.4 ± 1.5	29.2 ± 1.3	<0.001	28.6 ± 1.5	29.5 ± 1.3	<0.001	28.5 ± 1.5	29.1 ± 1.4	<0.001

Data are n (%) or mean ± standard deviation unless otherwise specified.

Table 2

Gestational Diabetes Mellitus Screening and Diagnostic Test Results Comparing Hispanic With Non-Hispanic White Race and Ethnicity for Each of Three Study Cohorts

	Glucose Intolerant (n=767)			Mild Untreated Gestational Diabetes Mellitus (n=371)			Mild Treated Gestational Diabetes Mellitus (n=397)		
	Hispanic 520 (68)	Non-Hispanic White 247 (32)	*P*	Hispanic 255 (69)	Non-Hispanic White 116 (31)	*P*	Hispanic 274 (69)	Non-Hispanic White 123 (31)	*P*
50g 1-hr oral glucose load (mg/dL)	152.6 ± 13.1	153.1 ± 13.3	0.57	160.6 ± 15.5	159.5 ± 15.9	0.51	159.0 ± 15.1	157.1 ± 14.3	0.25
100g 3-hr oral glucose tolerance test (mg/dL):									
Fasting	84.7 ± 5.8	85.0 ± 5.8	0.50	86.3 ± 5.8	86.3 ± 5.6	0.90	86.9 ± 5.6	85.5 ± 6.1	0.04
1 hr	156.3 ± 23.4	151.2 ± 26.0	0.006	193.8 ± 18.3	192.1 ± 21.9	0.46	192.1 ± 23.8	189.2 ± 19.1	0.20
2 hr	130.1 ± 22.0	130.5 ± 21.6	0.82	172.5 ± 21.1	172.6 ± 16.4	0.94	172.7 ± 22.6	174.8 ± 20.2	0.38
3 hr	111.6 ± 21.0	105.3 ± 23.2	0.0002	136.7 ± 29.2	128.6 ± 32.2	0.02	140.3 ± 28.3	133.3 ± 27.4	0.02

Data are n (%) or mean ± standard deviation unless otherwise specified.

Table 3

Prevalence and Adjusted Odds of Each Outcome Between Hispanic Compared With Non-Hispanic White Women With Glucose Intolerance (n=767), Mild Untreated Gestational Diabetes Mellitus (GDM) (n=371), and Mild Treated GDM (n=397)

	Glucose Intolerant (n=767)			**Mild Untreated Gestational Diabetes Mellitus (n=371)**			**Mild Treated Gestational Diabetes Mellitus (n=397)**		
	n (%) or Mean ± SD		**Adjusted Odds Ratio (95% CI) or β Coefficient ± SE (*P*)***	**n (%) or Mean ± SD**		**Adjusted Odds Ratio (95% CI) or β Coefficient ± SE (*P*)***	**n (%) or Mean ± SD**		**Adjusted Odds Ratio (95% CI) or β Coefficient ± SE (*P*)***
	Hispanic	**Non-Hispanic White**		**Hispanic**	**Non-Hispanic White**		**Hispanic**	**Non-Hispanic White**	
Primary composite outcome (n=731)†	184 (37)	65 (27)	**1.62 (1.10, 2.37)**	94 (38)	38 (34)	1.16 (0.69, 1.93)	92 (35)	30 (25)	1.62 (0.92, 2.86)
Hyperbilirubinemia	*47 (10)*	*24 (10)*	*0.88 (0.49, 1.57)*	31 (13)	12 (11)	1.11 (0.51, 2.40)	27 (11)	11 (9)	1.55 (0.63, 3.84)
Elevated cord C-peptide	*85 (19)*	*25 (13)*	***1.79 (1.04, 3.08)***	55 (24)	21 (20)	1.29 (0.69, 2.44)	50 (21)	11 (10)	**2.96 (1.33, 6.60)**
Hypoglycemia	*84 (21)*	*25 (13)*	***2.04 (1.18, 3.53)***	30 (15)	13 (14)	0.98 (0.44, 2.18)	34 (16)	15 (15)	0.75 (0.35, 1.59)
Gestational age at delivery (weeks)	39.4±1.6	39.1±1.5	**0.39±0.14 (0.005)**	39.2±1.6	38.7± 1.9	**0.48±0.21(0.02)**	39.2± 1.8	38.7± 1.7	**0.50±0.22(0.02)**
Preterm birth before 37 weeks	35 (7)	14 (6)	1.58 (0.75, 3.36)	23 (9)	14 (12)	0.61 (0.28, 1.33)	24 (9)	14 (11)	0.67 (0.30, 1.52)
Birthweight (g)	3431±499	3344±510	31.7±41.9 (0.45)	3478±543	3388±630	34.0±69.1(0.62)	3339±520	3287±484	20.1±61.0(0.74)
SGA (less than 10th percentile)	25 (5)	24 (10)	0.89 (0.44, 1.81)	13 (5)	9 (8)	1.21 (0.41, 3.58)	20 (7)	10 (8)	0.91 (0.37, 2.19)
LGA (greater than 90th percentile)	63 (12)	22 (9)	1.19 (0.67, 2.11)	38 (15)	16 (14)	0.94 (0.47, 1.86)	22 (8)	6 (5)	1.17 (0.42, 3.29)
Macrosomia greater than 4000g	62 (12)	23 (9)	1.12 (0.63, 1.98)	40 (16)	17 (15)	1.01 (0.52, 1.96)	20 (7)	5 (4)	1.56 (0.50, 4.86)
Gestational hypertension or preeclampsia	38 (7)	27 (11)	0.73 (0.41, 1.30)	37 (15)	13 (11)	1.71 (0.78, 3.71)	23 (8)	11 (9)	1.53 (0.58, 4.05)
NICU admission	30 (6)	19 (8)	0.97 (0.48, 1.94)	21 (8)	13 (11)	0.63 (0.28, 1.41)	20 (7)	8 (7)	1.07 (0.41, 2.81)

SD, standard deviation; CI, confidence interval; SE, standard error; SGA, small for gestational age; LGA, large for gestational age; NICU, neonatal intensive care unit.

*$P<.05$ significant; variables included in models: fasting, 1, 2, and 3 hour results of 100 g 3-hour oral glucose tolerance test (OGTT), parity, gestational age at enrollment, body mass index at enrollment, maternal age at enrollment, tobacco use in current pregnancy.

† Primary composite outcome: stillbirth, neonatal death, hypoglycemia, hyperbilirubinemia, neonatal elevated cord C-peptide, birth trauma; individual components analyzed and shown below composite except for stillbirth, neonatal death, birth trauma not analyzed due to small numbers.