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Neonatal Morbidity in the Offspring of Obese Women Without Hypertension or Diabetes

Brock Elliot Polnaszek, MD, Nandini Raghuraman, MD, MS, Julia D. Lopez, PhD, MPH, Antonina L. Frolova, MD, Victoria Wesevich, BS, Methodius G. Tuuli, MD, MPH, Alison G. Cahill, MD, MSCI

Washington University in Saint Louis School of Medicine, Department of Obstetrics and Gynecology, Division of Maternal Fetal Medicine. Saint Louis, MO, United States of America

Abstract

Objective—To compare the independent risk of neonatal morbidity between the offspring of obese and non-obese women without hypertension or diabetes.

Methods—This is a secondary analysis of a prospective single-center cohort study of singleton deliveries at or beyond 37 weeks of gestation from 2010–2014. Women with diabetes (pre-gestational or gestational) and hypertensive disorders were excluded. The primary outcomes were: 1) a composite neonatal morbidity including death, mechanical ventilation, respiratory distress, meconium aspiration, suspected sepsis, confirmed, sepsis, hypoxic ischemic encephalopathy, therapeutic hypothermia, or seizures and 2) a composite of neonatal neurologic morbidity including hypoxic ischemic encephalopathy, therapeutic hypothermia, or seizures. The primary outcomes were compared between the offspring of obese (BMI ≥ 30 kg/m²) and non-obese women. Adjusted odds ratios were estimated using multivariable logistic regression.

Results—Of 6,458 women without diabetes or hypertensive disorders, 3,311 (51%) were obese. After adjusting for race, neonates of obese patients were at significantly increased risk for the composite neonatal morbidity (9.2% vs 7.2%, (adjusted odds ratio [aOR] 1.39, 95% CI 1.15–1.67) and neurologic neonatal morbidity (0.7% vs 0.3%, aOR 2.84, 95% CI 1.22–6.65). Specifically, neonates of obese patients were more likely to have hypoxic ischemic encephalopathy (0.5% vs 0.2%, aOR 2.80, 95% CI 1.02–7.68), hypothermia treatment (0.6% vs 0.2%, aOR 2.92 95% CI 1.17–7.30], and suspected sepsis (7.6% vs 5.8%, aOR 1.45, 95% CI 1.18–1.79).

Conclusion—In patients who labor, maternal obesity is an independent risk factor for significant neonatal morbidity, even in the absence of hypertensive disorders or diabetes.

Corresponding Author: Brock E. Polnaszek, MD, Department of Obstetrics and Gynecology, Washington University in Saint Louis School of Medicine, 901 Forest Park Avenue. St. Louis, MO 63108. Phone: 314-565-3765; brock.polnaszek@wustl.edu.

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Introduction

Among reproductive age women, 58.5% are defined as overweight or obese, making obesity the most common health care condition for this population^{1,2}. Despite increasing prevalence, obesity's implications for pregnancy and neonatal outcomes are often underappreciated and unrecognized^{3,4}. Pregnancy in this population of women is considered high risk, mostly due to associated comorbidities such as hypertension and diabetes and their obstetric sequelae^{5,6}.

Obese mothers have poor health outcomes including an increased risk for cardiac dysfunction⁷ and venous thrombosis, as well as higher rates of cesarean sections, failed trial of labor, endometritis, and wound complications^{8,9}. Furthermore, obesity and the comorbidities of hypertension and diabetes have long been associated with adverse neonatal outcomes including congenital anomalies¹⁰, fetal growth restriction, stillbirth¹¹, and death¹¹. However, current literature suggests that between 65–85% of obese mothers do not have diabetes or hypertension at the time of delivery^{3,12,13}.

Few studies have examined the independent risk of maternal obesity on neonatal outcomes in the absence of these comorbidities. Current data suggests that obesity may be as important, if not more important, than hypertension and diabetes as a potentially modifiable risk factor for clinicians to target in order to improve neonatal outcomes^{14,15}. As obesity alone has been found to be a pro-inflammatory, pathophysiologic state^{16,17}, it is possible that this condition may alter the intrauterine environment of the developing fetus leading to adverse neonatal outcomes^{3,18}. We tested the hypothesis that obese women without hypertension or diabetes who labor are at increased risk of delivering neonate who suffer morbidity.

Materials and Methods

This is a secondary analysis of a prospective cohort study of singleton deliveries at 37 weeks or beyond from 2010–2014 from one large academic hospital¹⁹. The Washington University School of Medicine Human Research Protection Office approved of this study prior to enrollment (identification number 201102438). Women with diabetes (pre-gestational or gestational), hypertension (chronic, gestational, pre-eclampsia, and eclampsia), scheduled cesarean deliveries, and fetal anomalies were excluded from our cohort.

The primary outcomes were: 1) a composite of neonatal morbidity including death, mechanical ventilation, respiratory distress, meconium aspiration, suspected sepsis, confirmed sepsis, hypoxic ischemic encephalopathy, therapeutic hypothermia, or seizures and 2) a composite of neonatal neurologic morbidity including hypoxic ischemic encephalopathy, therapeutic hypothermia, or seizures. Information regarding maternal demographics, antenatal care, labor outcomes, and neonatal diagnoses were collected from medical records by trained research staff. Hypoxic ischemic encephalopathy was defined using American College of Obstetricians and Gynecologists (ACOG) and American Academy of Pediatrics (AAP) definitions^{20,21}. Therapeutic hypothermia was based on institution specific indications for treatment (i.e. pH <7.1, base deficit >12 mmol/L, Apgar <5 at 10 minutes, acute perinatal event and/or neurologic exam demonstrating moderate to

severe encephalopathy). Suspected sepsis was defined by pediatrician documentation of such in the medical records. At our institution, neonates with suspected sepsis are managed by AAP guidelines²⁰.

The primary outcomes were compared between the offspring of patients with and without obesity (BMI ≥ 30 kg/m²) using standard World Health Organization obesity definitions at the time of delivery⁴. Baseline clinical characteristics between women with obesity and without obesity were compared using the χ^2 or Fisher exact test for categorical variables and Mann-Whitney U test or Student t test for continuous variables, as appropriate. Odds ratios (OR) and 95% confidence intervals (CI) were calculated for each of the outcomes of interests. Multivariable logistic regression was used to adjust for pertinent confounding variables, which were identified by those variables that had at least a 10% effect size on the OR or were clinically relevant as confounders. Normality of distribution of the continuous variables was evaluated using the Kolmogorov-Smirnov test. Final models were tested with the Hosmer-Lemeshow goodness of fit test. We assessed the trend in neonatal morbidity across increasing classes of obesity using the Cochran Armitage test. We also performed additional analyses of neonatal outcomes among those with suspected sepsis. All patients from the primary study who met inclusion criteria were included; no a priori sample size estimation was performed. All analyses were performed using SAS software, Version 9.4 for Windows (Cary, North Carolina).

Results

A total of 8,580 patients were enrolled in the primary study¹⁹. Of these 1,585 were excluded for hypertension, 246 for diabetes, 135 with both hypertension and diabetes, 98 due to missing BMI data, and 58 who developed pre-eclampsia with severe features in labor and received magnesium, leaving a final cohort of 6,458 women. Of these, 3,147 (49%) were non-obese and 3,311 (51%) were obese. Of the women with obesity, 1,736 were class I, 947 were class II, and 628 were class III (Figure 1).

Several clinical characteristics differed between obese and non-obese pregnant women in our cohort. Women with obesity were older, more likely to be African American, deliver at a greater gestational age, have a prior cesarean section and undergo a cesarean delivery for the index pregnancy. Obese mothers were also more likely to be induced, have a prolonged 1st stage of labor, prostaglandin, foley bulb, and oxytocin during labor. Women with obesity also delivered neonates with a higher birthweight and lower mean umbilical artery pH. Rates of chorioamnionitis, maternal fever at delivery, and post-partum fever were higher in women with obesity, but these differences were not statistically significant (Table 1).

Of the neonates, 532 (8%) had the composite neonatal morbidity and 31 (0.5%) had the composite neurologic morbidity. After adjusting for race, neonates of women with obesity had increased odds of overall morbidity (9.2% vs 7.2% adjusted odds ratio [aOR] 1.39, 95% CI 1.15–1.67) and neurologic morbidity (0.7% vs 0.3%, aOR 2.84, 95% CI 1.22–6.65). Among the individual components of the composites, neonatal morbidity was largely driven by higher rates of suspected sepsis (7.6% vs 5.8%, aOR 1.45, 95% CI 1.18–1.79). Neonatal neurologic morbidity was driven by higher rates of hypoxic ischemic encephalopathy (0.5%

vs 0.2%, aOR 2.80, 95% CI 1.02–7.68) and hypothermia treatment (0.6% vs 0.2%, aOR 2.92 95% CI 1.17–7.30). (Table 2). A supplementary analysis of neonates born to women with diabetes or hypertension comorbidities who were obese compared to non-obese demonstrated no differences in neonatal morbidity (Table 3).

When stratified by degree of obesity, there was an increasing trend of the composite neonatal morbidity (7.2% for non-obese, 8.6% for class I obesity, and 10.4% for class II obesity, and 9.2% for class III obesity, p -trend=0.01) and the composite neurologic morbidity (0.3% for non-obese, 0.8% for class I obesity, 0.5% for class II obesity, and 0.8% for class III obesity, p -trend=0.04) with increasing class of obesity.

In the analysis of laboratory and clinical data among neonates with suspected sepsis, neonates born to obese mothers had lower absolute neutrophil counts at <6 hours and >12 hours. Neonates born to obese mothers had similar durations of antibiotic treatment (59.7 [interquartile range 59.3, 149.8] hours vs 59.7 [57.8, 72.1] hours, p =0.16). Obese mothers who delivered neonates with suspected sepsis were more likely to screen positive for group beta streptococcus on vaginal cultures. Maternal risk factors for sepsis as well as other clinical neonatal data did not differ between groups. The rate of bacterial proven sepsis in neonates with suspected sepsis was not significantly different with 3 (1.2%) neonates born to women with obesity and 4 (2.1%) neonates of non-obese women (Table 4).

Discussion

The results of this cohort study suggest that the offspring of women with obesity have a small but statistically significantly increased risk of neonatal and neurologic neonatal morbidity compared to the offspring of women without obesity. There is an obesity class-dependent trend towards increased neonatal morbidity. Specifically, neonates of women with obesity were more likely to have hypoxic ischemic encephalopathy, hypothermia treatment, and suspected sepsis. Among neonates with suspected sepsis, there were objective data that differed between women with and without obesity. However, bacterial culture proven sepsis was not different between groups.

The association between maternal obesity and neonatal morbidity is biologically plausible. Obesity is a pro-inflammatory, pathophysiologic disease state which alters the environment for the developing fetus^{16,17,22}. Maternal fat deposition results in inflammation leading to oxidative stress and endothelial dysfunction in maternal and placental tissues^{3,18}. Schmatz et. al propose that unique clinical manifestations are seen in neonates as the result of the pro-inflammatory state of obesity²². This has been validated at the molecular and tissue level using animal models^{17,22}. Teo et. al found that maternal obesity exacerbated the severity of damage caused by hypoxic-ischemic brain injury with evidence of enhanced pro-inflammatory mediators (e.g. TNF-alpha) and tissue-specific damage in the brain¹⁷. Furthermore, this pro-inflammatory state may be further exacerbated by the additive effects of maternal obesity on labor such as prolonged first stage of labor^{4,9,23}. Our findings suggest that neonates born to women with obesity may have clinical manifestations of this pro-inflammatory state.

Neonatal neurologic morbidity was an important but infrequent outcome in our study. The association between maternal obesity and neonatal neurologic morbidity has been previously demonstrated^{24–26}. A recently published nationwide cohort study of 3,029 children in Sweden demonstrated that maternal obesity was associated with an increased rate of cerebral palsy in term neonates²⁵. Additionally, our results are further in agreement with the growing body of literature which suggest that maternal obesity is as important for neonatal outcomes as obesity-related comorbidities^{14,15,27}. In a retrospective study of 24,505 singleton pregnancies in Denmark, Kristensen et. al showed that increased pre-pregnancy weight doubled the risk of stillbirth and neonatal death after excluding hypertensive disorders or diabetes¹⁴. Furthermore, in a retrospective study of 109,488 singleton deliveries, Rastogi et. al used ICD-9 data to demonstrate increased neonatal morbidity including sepsis (aOR 1.91 95% CI 1.45, 2.50) in neonates born to women with obesity, both with and without disease comorbidities²⁷. Our study is unique in that it includes a sample of women with obesity without comorbidities in a country with a wide prevalence of obesity and uses validated data abstracted from medical records.

The strengths of our study include the large sample size which allowed us to detect rare neonatal outcomes, prospective design, and unique cohort with the ability to use BMI in a class-dependent manner.

Our study has potential limitations to consider. Neonatal morbidity was largely driven by suspected sepsis. Although this was not culture proven sepsis, suspected sepsis is a clinically relevant outcome. The AAP guidelines for the management of suspected or early bacterial proven sepsis includes objective laboratory and clinical data pediatricians use to triage and risk stratify the neonate²⁰. Our analysis demonstrated differences in these variables between women with and without obesity. Our study suggests that maternal obesity alone may be an important factor for clinicians to consider when interpreting AAP data. Additionally, the rate of culture-proven sepsis was not different between groups but was consistent with the rate of 0.77 to 1 per 1,000 reported in the literature^{28–30}. Moreover, even after adjusting for race and the higher rate of maternal group B streptococci (GBS) culture in women with obesity, suspected sepsis remained statistically significant aOR 1.46 (1.17–1.82). Abnormal labor processes which may clinically impact neonatal morbidity including higher rate of induction, prolonged first stage of labor, and difference in mode of delivery in women with obesity were not significant covariates in our analysis.

The use of a composite morbidity may be considered a limitation. However, this was necessary for adequate statistical power to perform detailed analyses in what are otherwise infrequent individual outcomes.

In conclusion, obesity is an independent risk factor for neonatal morbidity, in the absence of maternal hypertension or diabetes. The results of this study highlight specific neonatal outcomes with long term sequelae that physicians should consider when counseling women with obesity. Future studies are needed to corroborate and identify strategies to reduce neonatal morbidity with the increasing number of women with obesity.

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Figure 1.
Participant study flowchart. BMI, body mass index.

Table 1

Clinical characteristics among pregnant women with obesity (BMI ≥ 30) and without obesity (N= 6,458) *

Clinical Characteristics	Obese (n= 3311)	Non-Obese (n= 3147)	P†
Maternal Age, years	25.7 ± 5.6	25.2 ± 5.9	<0.01
Maternal age ≥ 35, years	257 (7.8)	240 (7.6)	0.84
Race			<0.01
African American	2288 (69.1)	1813 (57.6)	
Caucasian	683 (20.6)	825 (26.2)	
Latina	246 (7.4)	238 (7.6)	
Gestational age at delivery, weeks	39.1 ± 1.2	38.9 ± 1.1	<0.01
Body mass index, kg/m ²	36.2 ± 5.8	26.2 ± 2.5	<0.01
Nulliparous	1252 (37.8)	1420 (45.1)	<0.01
Prior cesarean	344 (10.4)	231 (7.3)	<0.01
Prostaglandin	519 (15.7)	390 (12.4)	<0.01
Foley bulb	326 (9.9)	244 (7.8)	<0.01
Oxytocin	2227 (67.3)	1870 (59.4)	<0.01
Labor Type			
Induction	1325 (40.0)	1069 (34.0)	<0.01
Spontaneous and/or Augmented	1986 (60.0)	2078 (66.0)	
1 st Stage of Labor >95 th tile	159 (4.8)	106 (3.4)	<0.01
Mode of delivery			<0.01
Vaginal	2546 (76.9)	2605 (82.8)	
Operative vaginal	154 (4.7)	172 (5.4)	
Cesarean	611 (18.5)	370 (11.8)	
Maternal fever			
At delivery	168 (5.1)	129 (4.1)	0.06
Post-partum	131 (3.9)	97 (3.1)	0.06
Infant gender			0.63
Female	1591 (48.1)	1550 (49.2)	
Male	1719 (51.9)	1596 (50.8)	
Arterial pH	7.2 ± 0.07	7.3 ± 0.06	<0.01
Acidemia pH ≤ 7.10	55 (1.7%)	51 (1.6%)	0.90
Elevated lactate ≥ 4	819 (24.7)	811 (25.8)	0.32
Birthweight, grams	3331 ± 454	3173 ± 442	<0.01
Birthweight > 4000, grams	242 (7.2)	102 (3.2)	<0.01
Chorioamnionitis	162 (4.9)	122 (3.9)	0.16

Data are presented as number (percent) or mean ± standard deviation

* Excludes women with diabetes (pre-gestational and gestational), hypertension (chronic, gestational, pre-eclampsia and eclampsia), and intra-partum magnesium

† P values based on Student t test and X²

Table 2

Associations of neonatal outcomes among pregnant women with obesity (BMI ≥ 30) and without obesity (N= 6,458)*

Neonatal Outcomes	Total	Obese (n= 3311)	Non-Obese (n= 3147)	cOR [†] (95% CI)	aOR [‡] (95% CI)
Composite neonatal morbidity [§]	532 (8.2)	305 (9.2)	227 (7.2)	1.31 (1.09–1.56)	1.39 (1.15–1.67)
Neonatal death	3 (0.05)	2 (0.1)	1 (0.03)	1.90 (0.17–20.98)	1.84 (0.16–20.48)
Respiratory distress	228 (3.5)	118 (3.6)	100 (3.2)	1.13 (0.86–1.48)	1.12 (0.85–1.49)
Mechanical ventilation	30 (0.5)	15 (0.5)	15 (0.4)	0.95 (0.46–1.95)	0.87 (0.41–1.83)
Meconium aspiration syndrome	16 (0.2)	11 (0.3)	5 (0.2)	2.09 (0.73–6.04)	1.73 (0.59–5.10)
Suspected sepsis	437 (6.8)	253 (7.6)	184 (5.8)	1.33 (1.19–1.62)	1.45 (1.18–1.79)
Confirmed sepsis	7 (0.1)	3 (0.1)	4 (0.1)	0.71 (0.16–3.19)	0.61 (0.14–2.73)
Hypoxic-ischemic encephalopathy	21 (0.3)	16 (0.5)	5 (0.2)	3.05 (1.17–8.34)	2.80 (1.02–7.68)
Therapeutic hypothermia	27 (0.4)	20 (0.6)	7 (0.2)	2.73 (1.15–6.46)	2.92 (1.17–7.30)
Seizures	14 (0.2)	10 (0.3)	4 (0.2)	2.38 (0.75–7.60)	2.65 (0.73–9.64)
Composite neurologic morbidity ^{//}	31 (0.5)	23 (0.7)	8 (0.3)	2.74 (1.23–6.15)	2.84 (1.22–6.65)
Hypoxic-ischemic encephalopathy	21 (0.3)	16 (0.5)	5 (0.2)	3.05 (1.17–8.34)	2.80 (1.02–7.68)
Therapeutic hypothermia	27 (0.4)	20 (0.6)	7 (0.2)	2.73 (1.15–6.46)	2.92 (1.17–7.30)
Seizures	14 (0.2)	10 (0.3)	4 (0.2)	2.38 (0.75–7.60)	2.65 (0.73–9.64)

Data are presented as number (percent)

* Excludes women with diabetes (pre-gestational and gestational), hypertension (chronic, gestational, pre-eclampsia and eclampsia), and intra-partum magnesium

[†] Crude odds ratios[‡] Adjusted for race[§] Includes death, mechanical ventilation, respiratory distress, meconium aspiration syndrome, seizures, suspected sepsis, confirmed sepsis, hypoxic-ischemic encephalopathy, therapeutic hypothermia, and seizures^{//} Includes hypoxic-ischemic encephalopathy, therapeutic hypothermia, seizures

Table 3

Associations of neonatal outcomes among pregnant women with diabetes or hypertension with obesity (BMI 30) and without obesity (N=2,024) *

	Obese (n= 1416)	Non-Obese (n= 608)	cOR (95% CI)	aOR [†] (95% CI)
Composite neonatal morbidity [‡]	162 (11.4)	57 (9.4)	1.25 (0.91–1.72)	1.04 (0.75–1.44)
Neonatal death	1 (0.1)	0 (0.0)	--	--
Respiratory distress	83 (5.9)	27 (4.4)	1.34 (0.86–2.09)	1.10 (0.69–1.73)
Mechanical ventilation	16 (1.1)	3 (0.5)	2.30 (0.67–7.94)	2.12 (0.61–7.40)
Meconium aspiration syndrome	6 (0.4)	0 (0.0)	--	--
Suspected sepsis	132 (9.3)	46 (7.6)	1.26 (0.89–1.78)	1.05 (0.73–1.50)
Hypoxic-ischemic encephalopathy	13 (0.9)	1 (0.2)	5.62 (0.73–43.09)	4.00 (0.51–31.08)
Therapeutic hypothermia	13 (0.9)	2 (0.3)	2.81 (0.63–12.48)	2.26 (0.50–10.19)
Seizures	4 (0.3)	0 (0.0)	--	--
Composite neurologic morbidity [§]	17 (1.2)	2 (0.3)	3.68 (0.85–15.99)	2.78 (0.63–12.25)
Hypoxic-ischemic encephalopathy	13 (0.9)	1 (0.2)	5.62 (0.73–43.09)	4.00 (0.51–31.08)
Therapeutic hypothermia	13 (0.9)	2 (0.3)	2.81 (0.63–12.48)	2.26 (0.50–10.19)
Seizures	4 (0.3)	0 (0.0)	--	--

Data are n (%)

* Includes women with diabetes (pre-gestational and gestational), hypertension (chronic, gestational, pre-eclampsia and eclampsia), and intra-partum magnesium

[‡] Includes death, mechanical ventilation, respiratory distress, meconium aspiration syndrome, seizures, suspected sepsis, hypoxic-ischemic encephalopathy, therapeutic hypothermia, and seizures

[§] Includes hypoxic-ischemic encephalopathy, therapeutic hypothermia, seizures

[†] Adjusted for mode of delivery

Table 4

Maternal and neonatal characteristics used by the American Academy of Pediatrics' guidelines for suspected or bacterial proven neonatal sepsis among pregnant women with obesity (BMI ≥ 30) and without obesity (N=444) *

Laboratory and Clinical Data	Obese(n= 256)	Non-Obese(n= 188)	p†
Maternal and neonatal risk factors for suspected and/or bacterial proven sepsis			
Gestational Age	39.2 ± 1.2	39.0 ± 1.2	0.11
Induction	105 (41.2)	75 (39.9)	0.82
Rupture of Membranes > 18 hours	179 (69.9)	123 (65.4)	0.36
Maternal WBC			
Leukopenia <3.8 k/cumm	0 (0.0)	0 (0.0)	--
Leukocytosis >30 k/cumm	1 (0.4)	2 (1.1)	0.58
Chorioamnionitis	129 (50.4)	94 (47.8)	0.57
Maximum Temperature (Celsius)	38.1 ± 0.75	38.2 ± 0.80	0.40
Antibiotics Given during labor	194 (75.8)	133 (70.7)	0.23
Length of Antibiotics, hours	0.0 (0.0, 16.2)	0.0 (0.0, 15.5)	0.41
Maternal Vaginal Group B Streptococci Culture	75 (29.3)	35 (18.6)	0.03
White blood cell count			
< 6 hours	15.5 ± 5.9	16.8 ± 6.1	0.03
6 to 12 hours	16.6 ± 7.7	21.6 ± 10.8	0.09
>12 hours	15.3 ± 5.5	17.02 ± 5.4	0.02
Absolute neutrophil count			
< 6 hours	8379 (5616, 11160)	9834 (6527, 13680)	<0.01
6 to 12 hours	11552 (5704, 16236)	14096 (7540, 21594)	0.44
>12 hours	8178 (5618, 12052)	10335 (7692, 13452)	0.01
Immature to mature neutrophil ratio			
< 6 hours	0.07 (0.04, 0.10)	0.05 (0.03, 0.10)	0.07
6 to 12 hours	0.08 (0.03, 0.17)	0.08 (0.01, 0.18)	0.72
>12hours	0.08 (0.04, 0.13)	0.04 (0.02, 0.09)	0.02
Neonatal C-reactive protein			
< 6 hours	4.0 (3.0, 14.0)	4.0 (3.0, 27.5)	0.94
6 to 12 hours	16.5 (9.5, 29.5)	10.0 (3.0, 14.7)	0.29
>12hours	22.4 (8.0, 33.0)	21.0 (11.5, 33.2)	0.95
Maximum Temperature (Celsius)	36.9 ± 8.5	36.0 ± 14.0	0.40
Minimum Temperature (Celsius)	21.1 ± 4.5	20.8 ± 4.5	0.54
Bacterial Culture Positive	3 (1.2)	4 (2.1)	0.47
Antibiotics Given	241 (94.1)	177 (94.2)	0.99
Length Antibiotic, hours	59.7 (59.3, 149.8)	59.7 (57.8, 72.1)	0.16
Lumbar Puncture	43 (16.8)	28 (14.9)	0.61
Cerebral Spinal Fluid Culture	2 (0.8)	0 (0.0)	0.52
Length of Neonatal Intensive Care or Specialty-Care Unit Stay, days	3.0 (2.0, 7.0)	3 (2.0, 6.0)	0.20

Data are presented as number (percent), mean $\pm$ standard deviation, or median (interquartile range)

* Excludes women with diabetes (pre-gestational and gestational), hypertension (chronic, gestational, pre-eclampsia and eclampsia), and intra-partum magnesium

[†] Chi-square, Fisher's exact test, Student's t-test or Mann Whitney

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