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Adverse Birth Outcomes and Maternal *Neisseria gonorrhoeae* Infection: a Population-Based Cohort Study in Washington State

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Abstract

Background—*Neisseria gonorrhoeae* (gonorrhea) remains an important cause of reproductive and obstetric complications. There has been limited population-based research to evaluate the association between maternal gonorrhea and adverse birth outcomes.

Methods—A population-based retrospective cohort study was conducted of women with singleton pregnancies in Washington State from 2003–2014 using linked birth certificate and birth hospitalization discharge data. The exposed cohort consisted of women with gonorrhea diagnosed during pregnancy. The unexposed group, defined as pregnant women without gonorrhea, was selected by frequency-matching by birth year in a 4:1 ratio. Logistic regression was used to determine crude and adjusted odds ratios (OR) for the association of maternal gonorrhea and adverse birth outcomes.

Results—Women with gonorrhea during pregnancy (N=819) were more likely to be younger, Black, single, less educated, multiparous, and smokers compared to women without gonorrhea (N=3276). Maternal gonorrhea was significantly associated with a 40% increased odds (adjusted OR 1.4, 95% confidence interval (CI) 1.0–1.8) of low birth weight (LBW) infants compared to women without gonorrhea when adjusted for marital and smoking status. Maternal gonorrhea was associated with a 60% increased odds (OR 1.6, 95% CI 1.3–2.0) of small for gestational age (SGA) infants compared to women without gonorrhea.

Conclusions—This analysis showed that pregnant women with gonorrhea were more likely to have LBW infants, consistent with prior literature, and provided new evidence that maternal gonorrhea is associated with SGA infants. These findings support increased public health efforts to prevent, identify, and treat gonorrhea infection during pregnancy.

Short summary

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Maternal *Neisseria gonorrhoeae* infection was associated with low birth weight infants and small for gestational age infants in a population-based cohort study in Washington State.

Keywords

Gonorrhea; birth outcomes; maternal infection

Introduction

Sexually transmitted diseases (STDs) remain an important cause of reproductive and obstetric complications and can have severe adverse effects on pregnant women and infants. *Neisseria gonorrhoeae* (gonorrhea) is the second most prevalent STD in the United States and the burden of disease is highest among women 15 to 24 years of age.[1] In Washington State in 2014, the incidence of gonorrhea infection in women ages 15–24 was 273 cases per 100,000 women and this has been rising in the past five years.[1] Gonorrhea is often undiagnosed due to the high prevalence of asymptomatic infections and, therefore, relies on screening for detection.[2] If left untreated, gonorrhea can be associated with complications in women primarily due to the sequela of pelvic inflammatory disease, including ectopic pregnancies.[3] Multiple studies have shown that maternal gonorrhea is associated with preterm birth.[4–7] Additional adverse outcomes in pregnancy associated with maternal gonorrhea include chorioamnionitis, intrauterine growth restriction (IUGR), and premature rupture of membranes (PROM).[5] Furthermore, perinatal transmission of gonorrhea to the offspring can result in infant blindness, joint infections, and bloodstream infections.[3]

The Centers for Disease Control and Prevention (CDC) recommends screening for gonorrhea at the first prenatal visit in all pregnant women less than 25 years of age and older women at increased risk for gonorrhea.[3] The CDC only recommends rescreening women for gonorrhea in the third trimester if they are considered to be at continued high risk.[3] Screening during pregnancy is important to prevent the adverse effects of gonorrhea, however strong evidence for the CDC guidelines for gonorrhea screening in pregnancy is lacking.

There has been limited population-based research to evaluate the association between maternal gonorrhea infection and adverse birth outcomes. In addition, studies may have been hampered by small sample sizes. Older studies may have utilized less sensitive, culture-based testing to detect gonorrhea. Since 2002, nucleic acid amplification testing (NAAT) for gonorrhea has become more widely implemented in screening programs, which may have improved the detection of gonorrhea.[8] Further population-based cohort studies are needed to better characterize the association between maternal gonorrhea and adverse birth outcomes to better guide pregnancy screening recommendations and decrease maternal and infant morbidity.

This retrospective population-based cohort study used linked Washington State birth certificate and birth hospitalization discharge data from 2003 to 2014 to characterize the association between gonorrhea infection during pregnancy and adverse birth outcomes including low birth weight (LBW), preterm delivery, small for gestational age (SGA),

chorioamnionitis, prolonged PROM, and infant neonatal intensive care unit (NICU) admission.

Materials and Methods

Study Design & Data Sources

We conducted a population-based retrospective cohort study of women with singleton pregnancies in Washington State from 2003–2014. Our primary data source was Washington State birth certificate records. Birth certificate data are recorded shortly after delivery and submitted electronically to the Washington State Department of Health. Data captured by the birth certificate are obtained from the mother, the medical record, and/or health care providers. For this analysis, birth certificate data were linked to Comprehensive Hospital Abstract Report System (CHARS), which includes maternal discharge and infant birth hospitalization data. CHARS records provided information based on International Classification of Diseases, Ninth Revision (ICD-9) diagnosis codes. We identified specific codes that were used in our analysis through the Center for Medicare and Medicaid Services ICD-9 Code Lookup.[9] Because all data were de-identified, the Washington State Institutional Review Board considered this research to be exempt from review.

Exposure

The exposed cohort was defined as pregnant women with gonorrhea infection. A total of 819 mothers were included; 800 mothers were identified as having the check box for maternal gonorrhea “present and/or treated during pregnancy” selected on their infant’s birth certificate and an additional 19 mothers were identified as having ICD-9 codes 098.0–098.8 in CHARS. All those identified by birth certificate were also identified by ICD-9 code. Only the first birth with gonorrhea was included for mothers with more than one pregnancy with gonorrhea. The unexposed group, defined as mothers without gonorrhea, was frequency-matched by birth year in a 4:1 ratio from the remainder of the population of women with singleton pregnancies in Washington State from 2003–2014 who had birth hospitalization data in CHARS (N=3276).

Outcomes

Primary outcomes evaluated were LBW, preterm delivery, and SGA. Secondary outcomes were prolonged PROM, chorioamnionitis, and infant NICU admission. LBW was defined as a weight less than 2500 grams, preterm as delivery at less than 37 weeks gestation, and SGA as weight below the 10th percentile for gestational age. Gestational age was based on standard ultrasound measurements and was documented on the birth certificate. Birth weight and gestational age were recorded on the birth certificate and SGA was calculated from the birth weight and gestational age variables. High birth weight (greater than 4500 grams), post-term (greater than 43 weeks gestation), and large for gestational age infants (weight greater than 90th percentile for gestational age, LGA) were excluded from relevant analyses.[10] We excluded these infants because we were interested in comparing low birth weight, gestational age, and size for gestational age to those infants falling within the normal range. The variables prolonged PROM, chorioamnionitis, and NICU admission are check boxes marked on the birth certificate. Prolonged PROM is defined as premature rupture of

membranes for greater than or equal to 12 hours prior to labor. We supplemented diagnoses with data from CHARS using the ICD-9 codes 658.1 and 761.1 for prolonged PROM and 658.4 and 762.7 for chorioamnionitis. The ICD-9 code 761.1 identified PROM affecting the fetus or newborn including artificial rupture of membranes (ROM), which is dually coded with 658.3. We would not expect artificial ROM to be related to gonorrhea thus those coded with both 761.1 and 658.3 were not included as prolonged PROM diagnoses.

Covariates

We investigated the following descriptive maternal covariates from birth certificate data: age (≤ 25, 26–34, ≥35 years), race/ethnicity (White, Black, Hispanic, Asian, Native American, Hawaiian/Pacific Islander), marital status (married, single), maternal education (≤12 years, >12 years), parity (nulliparous, multiparous), maternal cigarettes smoking status (non-smoker, smoker), maternal diabetes (established or gestational diabetes, no diabetes), use of Women, Infants, and Children (WIC) federal assistance program (yes, no), and prior preterm births (yes, no). The Adequacy of Prenatal Care Utilization (Kotelchuck) Index, which categorizes the adequacy of prenatal care using gestational age and number of prenatal care visits (inadequate, intermediate, adequate, intensive), was also calculated from birth certificate data. Further description and validation of this index are described elsewhere.[11] Using both birth certificate data and CHARS, we also investigated the following maternal covariates: insurance status (insured, government-sponsored, uninsured, other) and presence of other maternal infections including chlamydia (ICD-9 099.5), genital herpes (ICD-9 054.9, 054.1), syphilis (ICD-9 097.9, 090.9), group B streptococcus (ICD-9 041.02, V02.51), hepatitis B (ICD-9 070.2, 070.3), and hepatitis C (ICD-9 070.51, 070.54, 070.70). The aforementioned maternal infections are also indicated through checkboxes marked on the birth certificates. We designated insured as private insurance and Civilian Health and Medical Program of the Uniformed Services (CHAMPUS)/Tricare, government-sponsored as Medicaid, Indian Health Service, and other government insurance, uninsured as self-pay and charity care, and other as other, unknown, or missing.

Statistical analyses

Stata 14.1 software was used for all statistical analyses.[12] We first estimated unadjusted odds ratios (ORs) for the association between maternal gonorrhea infection and each of our six outcomes using univariable logistic regression. Multivariable logistic regression models were then created specific for each outcome. Potential effect modifiers were evaluated by assessing stratum-specific estimates for meaningful differences using the Breslow-Day test for homogeneity.[13] If effect modification was present, an interaction term was included in the logistic regression model. We evaluated potential confounders using bivariable logistic regression. Variables that changed the unadjusted OR by greater than 10% were included in the multivariable logistic regression model. Records with missing data on variables for the association under investigation were excluded from the relevant analyses. Effect modifiers and confounders were selected based on prior studies, biologic plausibility for effect modification, and known associations with exposure and outcome for confounders.[14–22] We evaluated maternal age, diabetes, and prior preterm births for effect modification in our analyses of the association between maternal gonorrhea and LBW, preterm delivery, and SGA. We investigated diabetes and smoking as effect modifiers for chorioamnionitis and

evaluated prior preterm birth as an effect modifier for NICU admission. Maternal age, race/ethnicity, marital status, education, WIC, insurance, Kotelchuck index, smoking status, and other infections including chlamydia, genital herpes, syphilis, group B streptococcus, hepatitis B, and hepatitis C were assessed as potential confounders for all outcomes. Additionally, we conducted restricted analyses comparing the six adverse outcomes among the exposed and unexposed within sub-groups of possible confounders including age (≤25 >25), education (≤12 years, >12 years), and any maternal STD (combined chlamydia, herpes, syphilis, hepatitis B, hepatitis C; none). We calculated univariate and adjusted OR in each of these sub-groups for the association between maternal gonorrhea and each outcome. Any previously identified confounders by the 10% rule for the association between gonorrhea and each outcome were adjusted for in multivariable analyses.

We examined patterns of missingness in the data and used Hot Deck single imputation to impute values that were similar to the available values in the data matrix to evaluate the impact of missingness on our associations. We performed sensitivity analyses with varying categorizations of the variables age, insurance, and race. We also performed sensitivity analyses for our primary outcomes that included the previously excluded high weight infants in the LBW analysis, post-term infants in the preterm analysis, and LGA infants in the SGA analysis.

Results

Women with gonorrhea during pregnancy (N=819) were younger and more frequently Black, single, less educated, multiparous, and smokers during pregnancy compared to women without gonorrhea (N=3276, Table 1). Maternal chlamydia and genital herpes were more common in women with gonorrhea infection compared to women without gonorrhea. Additionally, women with gonorrhea during pregnancy more frequently received assistance from WIC, had inadequate prenatal care by Kotelchuck index, and had government-sponsored insurance compared to women without gonorrhea infections. Women with gonorrhea had a higher prevalence of maternal co-morbidities including diabetes, syphilis, hepatitis B, and hepatitis C.

Women with gonorrhea during pregnancy had a 40% increased odds of having an LBW infant (adjusted OR [aOR] 1.4, 95% confidence interval (CI) 1.0–1.8) compared to women without gonorrhea, when adjusted for marital status and smoking status (Table 2). Similarly, maternal gonorrhea infection was associated with a 60% increased odds (OR 1.6, 95% CI 1.3–2.0) of SGA infants compared to women without gonorrhea infection. There were no confounders found for the association between SGA and maternal gonorrhea. Gonorrhea infection during pregnancy was not significantly associated with preterm delivery. Maternal age, maternal diabetes, and prior preterm births did not modify the association observed between maternal gonorrhea and the primary outcomes (LBW, preterm delivery, and SGA).

There were no significant associations between maternal gonorrhea infection and our secondary outcomes of chorioamnionitis, prolonged PROM, or infant NICU admission (Table 3). The latter association was adjusted for maternal chlamydia infection and smoking status. Maternal diabetes and cigarette smoking during pregnancy did not modify the

association observed between maternal gonorrhea and chorioamnionitis. Prior preterm birth did not modify the association observed between maternal gonorrhea and NICU admission. Restricted analyses within each outcome by age, education, and any other maternal STD did not substantially change the risk estimates. Maternal gonorrhea infection was associated with SGA infants in mothers without any other STD (OR 1.5, 95% CI 1.1–2.1). Our missingness and sensitivity analyses did not substantially change the estimates of the associations.

Discussion

This analysis showed that after adjusting for confounders, women with gonorrhea were more likely to have LBW and SGA infants. Our analysis of preterm delivery and our secondary analyses of chorioamnionitis, prolonged PROM, and infant NICU admission showed no significant associations with maternal gonorrhea.

Our study joins a prior study, which identified an association between maternal gonorrhea infection and LBW.[23] This is biologically plausible as gonorrhea can cross the placenta and can result in intrauterine growth restriction (IUGR) and subsequently, LBW.[24] To our knowledge, there have been no prior published studies identifying a link between maternal gonorrhea and SGA infants. A retrospective population-based cohort study in Australia found maternal gonorrhea infection to be associated with preterm birth, but there was no association with SGA.[7] Our analysis showed a significant association between maternal gonorrhea and SGA, which we suspect may also be explained by maternal gonorrhea infection and IUGR, which in turn, could lead to SGA infants.[5,25] Our study, as compared to this prior study, included more women with gonorrhea infection and this may explain why we were able to detect the association between gonorrhea and SGA infants. In subanalyses restricted to mothers without any other STD, maternal gonorrhea was still associated with SGA infants, further supporting maternal gonorrhea as a risk factor for SGA independent of other STDs.

Several prior studies have shown an association between maternal gonorrhea infection and preterm delivery, however this association was not shown in our analysis.[4–7] The biological mechanism of preterm birth associated with maternal gonorrhea infection has been attributed to maternal cytokine response and increased fetal corticotropin-releasing hormone in response to infection.[26] It is possible that the association between gonorrhea infection and preterm delivery may be dependent on timing and the trimesters in which the infection was acquired, diagnosed and treated, for which we do not have data.

Prior literature has shown an association between gonorrhea infection during pregnancy and both chorioamnionitis and prolonged PROM, however this was not demonstrated in our analyses.[5] We also did not find a strong association between gonorrhea infection during pregnancy and infant NICU admissions, despite adequate power. It is possible that the lack of association with these secondary outcomes may have been due to residual confounding that attenuated our measured association. To our knowledge, there has been no prior research investigating the association between maternal gonorrhea infection and infant NICU admissions.

Strengths of our analyses include the large, population-based sample size and biologic plausibility of our findings. However, our findings should be considered in light of several limitations. First, we do not know which trimester the maternal gonorrhea infection was acquired and diagnosed. A prior study found that the association with maternal gonorrhea infection and preterm birth was strengthened if gonorrhea infection was diagnosed in the first trimester of pregnancy.[6] Adverse outcomes associated with infection in the first trimester are mechanistically plausible as the first trimester is the key period in which vital organs develop. Because we did not know the trimester of infection, we likely included infections from all three trimesters, which we would expect to attenuate the associations we found. Additionally, we do not have documentation of treatment of gonorrhea or the time to treatment. Based on standard medical practice, we would expect all documented gonorrhea infections to have been treated soon after identification, particularly in pregnancy, as this is a time of frequent medical follow-up if mothers are engaged in prenatal care. Presumptive antibiotic treatment of gonorrhea in pregnancy has been previously shown to decrease adverse birth outcomes, including low birth weight.[27] If all mothers had been promptly treated and we still found an association between gonorrhea and both LBW and SGA infants, this would support prioritizing gonorrhea prevention efforts. Furthermore, the number of gonococcal infections each mother had during each individual pregnancy was not known. This information would be important to evaluate for the potential impact of having multiple infections in pregnancy compared to a single infection. Our analysis was also limited by the fact that births less than 20 weeks gestation are not recorded on birth certificates. Many early deliveries are associated with infection, and we were not able to evaluate the role of maternal gonorrhea in these cases. The use of birth certificate data and CHARS is also limiting given the potential biases that can occur from under-ascertainment of exposures and outcomes, misreporting of information on birth certificates, and miscoding of ICD-9 diagnoses for the birth hospitalization. We investigated the impact of these limitations by performing missingness and sensitivity analyses, which did not change the estimates of our associations. Lastly, as with all observational studies, we must consider the influence of residual confounding on our findings.

The CDC recommends screening for gonorrhea at the first prenatal visit in all pregnant women less than 25 years of age and older women at increased risk for gonorrhea.[3] The CDC only recommends rescreening women for gonorrhea in the third trimester if they are considered to be at continued high risk.[3] In support of these guidelines, we found that maternal gonorrhea was more common in younger women although maternal age did not modify the association observed between maternal gonorrhea and adverse birth outcomes including LBW, preterm delivery, and SGA. Furthermore, several additional characteristics differed between women with and without gonorrhea infection in pregnancy such as race, marital status, proxies of low socioeconomic status, and the presence of other STDs. Clinician ascertainment of whether or not women remain at high risk in third trimester is subjective and prone to error. With the growing body of evidence that gonorrhea is associated with adverse birth outcomes, the potential benefits of screening all women for gonorrhea in the first and third trimester should be reconsidered. Further studies that delineate the effects of maternal gonorrhea infection by trimester as well as cost effectiveness analyses could help to further shape these guidelines.

In addition to assuring treatment of pregnant women with gonorrhea infection, it is also important to treat all of their sex partners in a timely fashion in an effort to prevent reinfection. This can be done by a patient referral strategy, in which the infected woman recommends her partners be evaluated for medical treatment. Expedited partner therapy (EPT) is an alternative strategy, in which patients deliver oral antibiotic treatment for an STD to their sex partners, and has been found to be a successful intervention.[28] Unfortunately, gonorrhea resistance has increased in recent years, and oral antibiotic therapy is no longer considered first line treatment for gonorrhea.[29] Nevertheless, the CDC recommends that if a sex partner of a pregnant woman with gonorrhea cannot be linked to evaluation and treatment in a timely fashion, expedited partner therapy (EPT) with oral antibiotic therapy could be considered, as not treating partners is more harmful than is the use of EPT for gonorrhea.[28] Yet another strategy for treating partners is concurrent patient-partner treatment, in which the pregnant women and her partner are treated at the same medical visit, as this has been shown to be more effective than a traditional patient referral treatment strategy.[30] The treatment of sex partners of pregnant women with gonorrhea infection remains a challenging public health concern and should be investigated in future studies.

In conclusion, our study found that women with gonorrhea were more likely to have LBW and SGA infants. Our findings support increased public health efforts to prevent, identify, and treat gonorrhea infection during pregnancy in order to decrease adverse birth outcomes.

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Table 1

Characteristics of women with and without *Neisseria gonorrhoeae* (gonorrhea) delivering singletons in Washington, 2003–2014

	Maternal gonorrhea infection			
	Yes (N=819)		No (N=3276)	
	n	%	n	%
Maternal age (years)				
≤25	544	66.4	1237	37.8
26–34	232	28.3	1545	47.2
≥35	43	5.3	492	15.0
Maternal Race/Ethnicity				
White	529	64.9	2291	70.9
Black	128	15.9	153	4.7
Hispanic	65	8.1	356	11.0
Asian	27	3.4	341	10.6
Native American	44	5.5	79	2.4
Hawaiian/Pacific Islander	10	1.3	13	0.4
Maternal Education (years)				
≤ 12	571	70.5	1387	42.8
Multiparous *	439	54.1	1224	37.9
Unmarried	618	76.3	1231	37.7
Prior Preterm Births	40	4.9	63	1.9
Cigarette smoking during pregnancy ◊	272	33.8	314	9.6
Maternal Diabetes	41	5.0	208	9.6
Maternal Chlamydia	391	47.7	43	1.3
Maternal Genital Herpes	124	15.1	116	3.5
Maternal Syphilis	12	1.5	0	0.0
Maternal Group B Streptococcus	231	28.2	671	20.5
Maternal Hepatitis B	9	1.1	12	0.4
Maternal Hepatitis C	15	1.8	5	0.2
Women, Infants and Children (WIC) **	547	71.6	1211	40.8
Kotelchuck 2-Factor Index §				
Inadequate	198	27.2	441	15.5
Intermediate	141	19.4	585	20.5
Adequate	252	34.6	1290	45.2
Intensive	137	18.8	538	18.9
Insurance Type				
Uninsured	10	1.3	37	1.2
Government-sponsored	602	75.8	1464	45.7
Insured	181	22.8	1703	53.1

	Maternal gonorrhea infection			
	Yes (N=819)		No (N=3276)	
	n	%	n	%
Other	1	0.1	1	0.0

N=sample size

[a]Numbers may not add up to totals because of missing data; all variables have <5% missing unless indicated.

[*]5.2% missing data;

[**]16.2% missing data;

[§]23.0% missing data

[◊]Defined as any cigarette smoking during pregnancy

Table 2

Birth outcomes following maternal *Neisseria gonorrhoeae* (gonorrhea) infection during pregnancy, Washington State, 2003–2014

	Gonorrhea N=819		No Gonorrhea N=3276					
Outcome	**n**	**%**	**n**	**%**	**Odds Ratio**	**95% CI**	**Adjusted Odds Ratio**	**95% CI**
Low birth weight[a,e]	80	9.9	186	5.8	1.8	1.4–2.4	1.4[b]	1.0–1.8
Preterm infant[c,f]	93	11.4	260	7.9	1.5	1.2–1.9	1.1[b]	0.9–1.5
Small for gestational age[d]	119	15.7	316	10.5	1.6	1.3–2.0	-	-

N=sample size; CI=Confidence Interval

[a] Defined as <2500 grams

[b] Adjusted for marital status and smoking status

[c] Defined as <37 weeks

[d] Defined as <10% of weight by gestational age

[e] The low birth weight rate for singleton births in Washington State from 2005–2014 ranged from 4.6–5.0%[31]

[f] The preterm birth rate for singleton births in Washington State from 2005–2014 ranged from 9.5–10.7%[31]

Table 3

Birth outcomes following maternal *Neisseria gonorrhoeae* (gonorrhea) infection during pregnancy, Washington State, 2003–2014

	Gonorrhea N=819		No Gonorrhea N=3276			
Outcome	**n**	**%**	**n**	**%**	**Odds Ratio**	**95% CI**
Chorioamnionitis[a]	31	3.8	150	4.6	0.8	0.6–1.2
Prolonged premature rupture of membranes[b]	90	11.0	326	10.0	1.1	0.9–1.4
Neonatal intensive care unit transfer[c]	98	12.3	235	7.3	1.2[d]	0.9–1.7

N=sample size; CI=Confidence Interval

[a] Intra-amniotic infection

[b] Greater than or equal to 12 hours prior to labor

[c] During birth hospitalization

[d] Adjusted for maternal Chlamydia infection and smoking status