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Use of a Simple Clinical Tool for Airway Assessment to Predict Adverse Pregnancy Outcomes

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Abstract

Objective—Obstructive sleep apnea (OSA) is a risk factor for adverse perinatal outcomes. We aimed to test the hypothesis that maternal Mallampati class (MC), as a marker for OSA, is associated with adverse perinatal outcomes.

Study Design—We performed a retrospective secondary analysis of a prospective cohort of term births (≥ 37 weeks). Fetal anomalies and aneuploidy were excluded. Primary outcome was small for gestational age (SGA). Secondary outcomes included preeclampsia, neonatal cord arterial blood gas pH < 7.10 and pH < 7.05, base excess < -8 mEq/L and < -12 mEq/L. Outcomes

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were compared between mothers with low MC airways and high MC airways using logistic regression.

Results—1,823 women met inclusion criteria. No significant differences were found in the risk of SGA (aOR 0.9 95% CI 0.6-1.2), preeclampsia (aOR 1.2 95% CI 0.8-1.9) or neonatal acidemia (aOR 0.8 95% CI 0.3-2.0), between high and low MC.

Conclusion—High MC is not associated with adverse perinatal outcomes.

Keywords

Mallampati; obstructive sleep apnea; OSA; small for gestational age; sleep disordered breathing

Introduction

Intermittent hypoxia and hypercapnia secondary to obstructive sleep apnea (OSA) is strongly associated with pathologic changes in the cardiac, pulmonary, metabolic and inflammatory systems in non-pregnant adults.¹⁻⁵ It has been postulated that OSA may also be associated with adverse maternal and fetal outcomes in pregnancy through mechanisms such as sympathetic activation, endothelial damage and fetal hypoxia triggered by maternal hypoxia and hypercapnia.⁶ The body of evidence to support this hypothesis is quickly mounting.⁵⁻⁹ Pregnancies complicated by OSA have been associated with increased risk for preeclampsia, cesarean section, indicated pre-term birth, neonatal intensive care unit admission, SGA and low birthweight.⁷⁻⁹

Overnight polysomnography is the gold-standard diagnostic test for OSA. An apnea-hypopnea index is determined from the overnight polysomnography and used to classify disease severity.¹⁰ However, the test is inconvenient and expensive,^{11,12} thus, there is crucial need for less burdensome screening tools both for clinical and investigative purposes. In 1985, Mallampati et al. published a simple, non-invasive, clinical tool to predict difficult intubation.¹³ The original three tier system described by Mallampati in 1985 was modified by Samsoon et al. in 1987 to include a fourth tier for the completely occluded posterior pharynx where the soft palate alone is visible with complete obliteration of the faucial pillars.¹⁴ This four tier system remains the current method used by anesthesiologists for risk stratification at the time of intubation.¹⁵ The four tier Mallampati classification system assigns a score of I-IV, with IV representing the most occluded oropharynx and, therefore, the most difficult airway for intubation. (Table 1) A recent meta-analysis by Friedman et al. assessed the diagnostic value of clinical airway assessment for obstructive sleep apnea and found that the Mallampati classification correlates with OSA severity as determined by the apnea-hypopnea index.¹⁵ Thus, the MC may be a simple clinical tool used in place of formal OSA testing to identify at-risk women. The specific aim of this study was to investigate the hypothesis that maternal MC, as a clinical marker of airway obstruction and OSA, is associated with adverse perinatal outcomes.

Methods

This was a retrospective secondary analysis of prospectively collected data nested within a larger, on-going prospective cohort study of consecutive term births investigating the

association between electronic fetal monitoring patterns and neonatal outcomes. We included the first 2000 women enrolled in the larger study in our evaluation of MC and perinatal outcomes. Women were excluded if they did not labor, carried a fetus with known structural or chromosome anomalies, multiple gestations, or were ≥ 37 weeks gestation. The study was approved by the Washington University School of Medicine Human Research Protection Office.

Detailed data extraction from the medical record was performed by formally trained obstetric research nurses including maternal sociodemographic information, obstetric and medical history. Gestational age was determined by last menstrual period and confirmed by first or second trimester ultrasound. If gestational age by ultrasound was noted to be discrepant from last menstrual period, then dating occurred by ultrasound if the dating discrepancy was greater than 7 days in the first trimester and greater than 10 days in the second trimester. If last menstrual period was unknown, gestational age was determined by ultrasound.

Every woman admitted to labor and delivery is assigned a MC by the attending anesthesiologist and this information was obtained from the medical record. Additionally, information regarding labor type (spontaneous, augmented or induced), mode of delivery and the use of regional anesthesia and neonatal cord blood gas analysis collected at the time of delivery were also extracted from the medical record.

Women with MC I or II airways were compared to those with MC III or IV airways. The decision to group women into two groups in this manner was based on previous work published by Nuckton et al. demonstrating the prevalence of OSA is similar among class III and class IV airways.¹⁶ The primary outcome was small for gestational age (SGA). SGA was defined as birthweight $< 10^{\text{th}}$ percentile and $< 5^{\text{th}}$ percentile according to the Alexander reference.¹⁷ Secondary outcomes were preeclampsia, neonatal cord arterial blood gas (ABG) pH < 7.05 and pH < 7.10 , base excess (BE) < -12 mEq/L and BE < -8 mEq/L. Preeclampsia was defined according to the guidelines of the International Society of the Study of Hypertension in Pregnancy.¹⁸

We compared baseline characteristics of mothers in the two groups. Continuous variables were compared using Student's t test or Mann-Whitney U test and categorical variables were compared using the χ^2 or Fisher's exact test, as appropriate. Normality was tested using the Shapiro-Francia test. Rates and odds ratios (ORs) with 95% confidence intervals (CIs) were calculated for primary and secondary outcomes. Multivariable logistic regression was used to estimate risk by adjusting for relevant confounding factors. Variables for logistic regression were chosen based on the univariate analyses and risk factors that have been identified in the literature. The numbers of variables in each model were reduced using backward step-wise elimination. The Wald test, or likelihood ratio test, was utilized to assess differences between hierarchical models. The Hosmer-Lemeshow goodness-of-fit test was used to assess the final models.¹⁹ Tests were two-tailed, and $P < 0.05$ was considered significant. We performed a subgroup analysis comparing outcomes in women with MC I airways to women with MC IV airways.

Based on an estimated 20% prevalence of MC III or IV in our patient population, we estimated that 1800 women will provide 80% power to detect a 40% (1.4 fold) difference in SGA < 10th percentile between women with MC III or IV and those with MC I or II airways (based on a 2-tailed test and alpha of 0.05). All statistical analyses were performed using STATA 10 special edition (College Station, TX).

Results

Of 1,823 women meeting inclusion criteria, 348 (19%) were classified as MCI, 1,069 (58.6%) as MC II, 376 (20.6%) as MC III and 30 (1.6%) as MC IV. Table 2 compares baseline demographic and clinical characteristics of the study population. Compared to women with MC I or II airways, women with MC III or IV airways were similar in age, parity and gestational age at delivery. Diabetes, as well as smoking and alcohol use did not differ between groups. There was no difference in mode of delivery in the previous or current pregnancy or difference in labor type between women with MC I or II airways compared to women with MC III or IV airways. Women with MC III or IV airways were more likely to be black and have a greater body mass index (BMI) compared to women with patent airways.

There were no significant differences in primary or secondary outcomes between women with MC I or II airways compared to MC III or IV airways (Table 3). There was no difference in the rate of SGA < 10th percentile (11.8% compared to 13.3%, aOR 0.9 95% CI 0.6-1.2), or SGA < 5th percentile (6.1% compared to 6.9%, aOR 1.2 95% CI 0.8-1.9) among women with MC III or IV and those with MC I or II airways. The risk of preeclampsia was also not significantly different between the MC I or II and the MC III or IV airway groups (10.6% compared to 13.1%, aOR 1.1 95% CI 0.8-1.6). Of neonates in the MC I or II airway group, 1.7% had a cord ABG of < 7.10 compared to 2.0% of neonates in the MC III or IV airway group, the difference remained insignificant after adjustment for confounding variables (adjusted OR (aOR) 0.8, 95% CI 0.3-2.0). The risk of neonatal ABG pH < 7.05 was also not significantly different between groups (aOR 0.7, 95% CI 0.1-3.2). Further, there was no difference in BE < -8 mEq/L or BE < -12 mEq/L between mothers with MC I or II airways and mothers with MC III or IV airways (3.4% compared to 3.7% aOR 0.9, 95% CI 0.5-1.6 and 0.9% compared to 0.7% aOR 0.8, 95% CI 0.2-2.8, respectively). The secondary analysis comparing extremes of airway score is represented in Table 4. The 348 women (19%) with MC I airways were compared to the 30 women (1.6%) with MC IV airways. The risk of SGA <10% and <5%, preeclampsia, and neonatal umbilical cord pH < 7.10, BE < -8 mEq/L, BE < -12 mEq/L for women with MC IV airways compared to MC I airways were not significantly different (Table 4).

Discussion

We found no association between maternal MC and adverse perinatal outcomes at term. Comparing mothers with MC I or II airways to mothers with MC III or IV airways, there was no difference in risk of having an SGA neonate. Further, mothers with MC III or IV airways were no more likely to have a neonate with acidemia or to develop preeclampsia when compared to mothers with patent airways. Comparison of the most occluded airways

(MC IV) to the least occluded (MC I) in our secondary analysis also demonstrated no difference in risks of adverse perinatal outcomes.

Substantial evidence now exists associating OSA with adverse perinatal outcomes. As such, identification of simple clinical tools to substitute for formal OSA testing in pregnancy is of great interest. In 2012, Chen et al. performed a case-control study of 4,746 women from the Taiwan National Health Insurance Research Data Set.⁸ Cases were selected based on diagnosis of OSA by overnight polysomnography within 1 year of delivery and controls were matched for maternal age. After adjusting for several confounding variables including but not limited to BMI, diabetes and gestational hypertension the adjusted ORs for SGA, low birth weight, preterm birth, cesarean section, and preeclampsia were all significantly greater in the OSA group compared to the non-OSA group. The authors also found increased risks of gestational diabetes and gestational hypertension among women with OSA.⁸ More recently, Louis et al. demonstrated similar findings in a prospective observational study of 175 obese pregnant women.⁹ These women with singleton pregnancies underwent screening for OSA using a home polysomnography device with confirmatory diagnosis of screen positive individuals using in-laboratory overnight polysomnography. Logistic regression with adjustment for age, race and BMI demonstrated increased odds of cesarean section, preeclampsia, neonatal intensive care unit admission and neonatal hyperbilirubinemia in the OSA group. Additionally, the incidence of OSA among obese individuals was found to be 15.4% and women with OSA were significantly heavier than women without OSA (BMI 46.8 ± 12.2 compared to 38.1 ± 7.5 , $p=0.002$).⁹

The need for effective tools to diagnose and screen for OSA in pregnancy is important both for future investigation and clinical utility; however, finding practical tools has remained challenging. The use of overnight polysomnography for investigative purposes is limited by the financial burden of the test and inconvenience for the patient. Further, screening for OSA presents a diagnostic dilemma in pregnancy due to physiologic changes including airway edema, weight gain, and hormonal effects leading to increased snoring and sleep disordered breathing.^{20,21} Our choice of the Mallampati Classification system as a marker of OSA was twofold. First, it is a simple, objective, clinical assessment, which makes it an ideal candidate tool for OSA screening. Second, there is supporting evidence in the non-pregnant population for the utility of Mallampati Classification for OSA screening.^{16,22,23} In a prospective trial published in 2006, Nuckton et al. demonstrated the odds of having OSA increased by greater than 2-fold for every 1 point increase in MC (OR 2.5 95% CI 1.2-5.0 $p=0.01$).¹⁶ Additionally, the severity of OSA as indicated by increasing apnea-hypopnea index on polysomnography also increased as MC increased. The authors concluded the Mallampati scoring system is a practical tool for investigative and clinical purposes.¹⁶ Most recently, Friedman et al. published a meta-analysis evaluating the diagnostic value of airway assessment for OSA. When the authors evaluated studies specific for Mallampati classification, they found that MC positively correlated with OSA severity by the apnea-hypopnea index.¹⁵

A major strength of our study is the novel approach of using an objective anatomic marker of OSA, MC, to study risk of adverse pregnancy outcomes. We were able to evaluate our research question using prospectively collected data, limiting observer bias in the

assignment of MC. Furthermore, we were able to accrue a relatively large sample size, which gave us the ability to evaluate rare outcomes such as neonatal acidemia at term and adjust for important confounders. Finally, our sample size afforded us the opportunity to evaluate our data in a stratified analysis comparing MC I airways to class IV airways to investigate the presence of an association between extremes of airway anatomy that may not have been detected in the primary analysis.

By design, we did not perform formal diagnosis of OSA; however, we chose to investigate MC in direct association with adverse perinatal outcomes, making our study clinically relevant with the intent that Mallampati screening might replace the need for OSA testing. Our study was limited in our ability to evaluate rare outcomes because, despite our large sample size, extremely obstructed airways were rare, accounting for only 1.6% of the study population. Additionally, it is important to highlight that MC was assigned by an anesthesiologist for clinical use, as opposed to assignment by a research nurse or trained study assistant for investigational purposes. Although our method of assignment of MC was unable to provide construct validity, the board certified anesthesiologists responsible for assignment of MC are trained outside the scope of the study to critically evaluate the airway. Further, because MC was assigned for general clinical use, without knowledge of study participation, we would expect misclassification to be random and tend to bias our results towards the null hypothesis or no difference.

One reason for our negative findings may be that MC has been reported to increase throughout pregnancy and during labor.^{24,25} Therefore, women who were classified as MC III or IV may have progressed through pregnancy as MC I or II and not exhibit the effects one would anticipate of a class III or IV airway. This could be remedied in a future study by assigning MC earlier in the pregnancy, ideally in the first trimester. Further, because our study was limited to patients who underwent labor, our findings may be biased toward the null hypothesis as patients who undergo cesarean without labor may be those at highest risk of adverse pregnancy outcomes. In addition, MC alone may be a poor predictor of OSA in pregnancy. The best way to answer this question would be to perform a prospective study to investigate the ability of MC to predict OSA by using overnight polysomnography as the diagnostic “gold standard”; however, recruitment is likely to be difficult given the nature of the “gold standard”. Finally, combining anatomic airway assessment such as MC with a qualitative questionnaire may provide better prediction of OSA in pregnancy than either tool alone.

In conclusion, we found no association between maternal MC and adverse pregnancy outcomes. Due to the rarity of class III and IV airways, larger studies are necessary to fully evaluate the potential role of the Mallampati classification system in predicting adverse pregnancy outcome. Finally, as the evidence associating OSA and adverse perinatal outcomes mounts and the number of OSA affected individuals grows in parallel to the rising obesity epidemic, so does the need to find a practical tool to screen for OSA in pregnancy.

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Abbreviations used

OSA	obstructive sleep apnea
MC	Mallampati class
SGA	small for gestational age
ABG	cord arterial blood gas
BE	base excess
OR	odds ratio
CI	confidence interval

Table 1Mallampati classification system¹³

Mallampati Class	Description of airway
I	Complete visualization of uvula, tonsils and palatal arches
II	Complete visualization of uvula, partly visible tonsils and palatal arches
III	Uvula, tonsils, and palatal arches hidden, soft and hard palate visible
IV	Only hard palate visible

Table 2

Maternal demographic and clinical characteristics

	MC* I, II (n=1417)	MC* III, IV (n=406)	P
Maternal age (years)	25.2 ± 6.1	25.7 ± 6.1	0.17
BMI (kg/m ²)	31.6 ± 7.1	34.6 ± 8.2	<0.01
Gravidity	2.6 ± 1.8	2.7 ± 1.8	0.40
Prior vaginal delivery	710 (50.1)	221 (54.4)	0.12
Nulliparous	639 (45.1)	167 (41.1)	0.16
Prior cesarean section	123 (8.7)	31 (7.6)	0.50
Gestational age (weeks)	38.9 ± 1.2	39.0 ± 1.2	0.16
Black race	902 (63.7)	296 (72.9)	<0.01
Labor			
Induction	612 (43.2)	200 (49.3)	0.09
Augmentation	439 (31.0)	109 (26.9)	
Spontaneous	366 (25.8)	97 (23.9)	
Regional anesthesia	1312 (92.6)	376 (92.6)	0.99
Mode of delivery			
Vaginal, spontaneous	1073 (75.7)	307 (75.6)	0.90
Vaginal, operative	95 (6.7)	25 (6.2)	
Cesarean	249 (17.6)	74 (18.2)	
Tobacco use	213 (15.0)	68 (16.8)	0.40
Alcohol use	22 (1.6)	9 (2.2)	0.38
Diabetes Mellitus	18 (1.3)	6 (1.5)	0.81
Gestational diabetes	38 (2.7)	13 (3.2)	0.61

* Mallampati class

Table 3

Association Between Adverse Perinatal Outcomes and Airway Class

	MC* I, II (n=1417)	MC* III, IV (n=406)	RR (95% CI)	Adjusted OR (95% CI)
SGA <10%	189 (13.3)	48 (11.8)	0.9 (0.7-1.2)	0.9 (0.6-1.2) [‡]
SGA <5%	86 (6.1)	28 (6.9)	1.1 (0.8-1.6)	1.2 (0.8-1.9) [‡]
Preeclampsia	150 (10.6)	53 (13.1)	1.2 (0.9-1.5)	1.1 (0.8-1.6) [‡]
Acidemia <7.10	24 (1.7)	8 (2.0)	1.1 (0.6-2.1)	0.8 (0.3-2.0) [‡]
Acidemia <7.05	9 (0.6)	3 (0.7)	1.1 (0.4-3.0)	0.7 (0.1-3.2) [‡]
Base Excess <-8 mEq/L	48 (3.4)	15 (3.7)	1.1 (0.7-1.7)	0.9 (0.5-1.6) [‡]
Base Excess <-12 mEq/L	12 (0.9)	3 (0.7)	0.9 (0.3-2.5)	0.8 (0.2-2.8) [‡]

* Mallampati class

[‡] Adjusted for black race, BMI ≥ 30 kg/m², and labor type (spontaneous, augmented or induced)[‡] Adjusted for black race and BMI ≥ 30 kg/m²

Table 4Association between adverse outcomes and extremes of airway class^{*}

	MC [†] I %(n=348)	MC [†] IV %(n=30)	RR (95% CI)
IUGR <10%	48 (13.8)	0 (0)	--
IUGR <5%	23 (6.6)	0 (0)	--
Preeclampsia	38 (10.9)	3 (10.0)	0.9 (0.3-2.9)
Acidemia <7.10	5 (1.4)	1 (3.3)	2.1 (0.3-13.2)
Acidemia <7.05	0 (0)	1 (3.3)	--
Base Excess <-8 mEq/L	11 (3.2)	3 (10.0)	2.9 (0.9-8.4)
Base Excess <-12 mEq/L	2 (0.6)	1 (3.3)	4.3 (0.8-22.1)

^{*} All models adjusted for BMI ≥30[†] Mallampati class