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First-trimester mean arterial blood pressure and the risk of preeclampsia: The Great Obstetrical Syndromes (GOS) study

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ABSTRACT

Objective: To estimate the predictive value of first-trimester mean arterial pressure (MAP) for the hypertensive disorders of pregnancy (HDP).

Study methods.: We performed a prospective cohort study of nulliparous women recruited at $11^{0/7}$–$13^{6/7}$ weeks. MAP was calculated from blood pressure measured on both arms simultaneously using an automated device taking a series of recordings until blood pressure stability was reached. MAP was reported as multiples of the median adjusted for gestational age. Participants were followed for development of gestational hypertension (GH), preeclampsia (PE), preterm PE (< 37 weeks) and early-onset (EO) PE (< 34 weeks). Receiver operating characteristic curves and the area under the curve (AUC) were used to estimate the predictive values of MAP. Multivariate logistic regressions were used to develop predictive models combining MAP and maternal characteristics.

Results: We obtained complete follow-up in 4700 (99%) out of 4749 eligible participants. GH without PE was observed in 250 (5.3%) participants, and PE in 241 (5.1%), including 33 (0.7%) preterm PE and 10 (0.2%) EO-PE. First-trimester MAP was associated with GH (AUC: 0.77; 95%CI: 0.74–0.80); term PE (0.73; 95%CI: 0.70–0.76), preterm PE (0.80; 95%CI: 0.73–0.87) and EO-PE (0.79; 95%CI: 0.62–0.96). At a 10% false-positive rate, first-trimester MAP could have predicted 39% of GH, 34% of term PE, 48% of preterm PE and 60% of EO-PE. The addition of maternal characteristics improved the predictive values (to 40%, 37%, 55% and 70%, respectively).

Conclusion: First-trimester MAP is a strong predictor of GH and PE in nulliparous women.

1. Introduction

Preeclampsia (PE) is a significant cause of perinatal and maternal morbidity and mortality, which affects 2–8% of pregnancies [1]. PE is an hypertensive disease of pregnancy (HDP) that occurs after 20 weeks of gestation in previously normotensive women and is typically associated with significant proteinuria and adverse outcomes [2,3]. PE has been subdivided into term and preterm PE (after or before 37 weeks of gestation) and into late- and early-onset PE (after or before 34 weeks) [4,5]. Preterm and early-onset PE are associated with incomplete transformation of uterine spiral arteries, commonly reported as deep placentation disorders, and with high incidence of fetal growth restriction and perinatal morbidity [2,6–8].

Recent meta-analyses suggested that PE, but mainly the preterm and severe forms of PE, could be prevented with low-dose aspirin started before 16 weeks of gestation [9–11]. The ASPRE trial confirmed these hypotheses: the rate of preterm PE was decreased by 62% (from 4.3% to 1.6%, p = 0.004) in women taking 150 mg of aspirin at bedtime daily compared to placebo [12]. Those evidences enhance the interest for the early prediction of preterm PE.

Several maternal characteristics are associated with greater risk of developing PE (personal or family history of PE, nulliparity, multifetal gestation, diabetes, obesity, etc.) but the prediction of PE based on maternal characteristics alone remains difficult [13–15]. National societies are recommending low-dose aspirin from early pregnancy to nulliparous women based on the presence of one additional risk factors or more, but the current literature suggests that such approach is associated with a low predictive values (< 35%) or high false positive rates (> 64%) [16,17]. Some authors reported that the combination of maternal history, biophysical markers, biochemical markers and

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ultrasound markers could potentially predict most cases of preterm PE [18,19]. The algorithm used to identify women at high-risk for preterm PE in ASPRE included the combination of maternal characteristics (age, body mass index, family history), mean arterial blood pressure (MAP) obtained from an automated device, uterine artery pulsatility index and biochemical markers (placental growth factor and pregnancy-associated plasma protein A) in a mathematical model with an expected predictive value of 77% for a false-positive rate of 10% [20]. However, others have challenged these observations [21]. MAP is an important biophysical marker of those predictive models with inconsistent performance, which can be due to the variations in methods of measurement [22,23]. A case-cohort study suggested that MAP taken by an automated device, as recommended for the ASPRE trial, is better to identify women at high-risk of PE than MAP measured using a manual device [24]. It became urgent to evaluate prospectively the predictive role of MAP using an automated device.

In this study, we aimed to estimate the value of first-trimester MAP, alone or in combination with other maternal characteristics, for the prediction of the hypertensive disorders of pregnancy, including GH, PE, preterm PE and early-onset PE.

2. Material and methods

We conducted a prospective cohort study composed of nulliparous women with singleton pregnancies attending their $11^{0/7}$–$13^{6/7}$ weeks ultrasound between March 2011 and December 2014 in two academic centers of Quebec City in Canada. Eligible women were at least 18 years old and at $11^{0/7}$–$13^{6/7}$ weeks of gestation. Gestational age was determined by the fetal crown-rump length (CRL). Only pregnancies without major fetal abnormalities were included. We excluded women who underwent pregnancy interruption. After obtaining written informed consent, we asked each participant to complete a questionnaire on maternal characteristics and medical history. Maternal weight and height were measured to calculate the body mass index. Participants were followed until delivery and medical charts were reviewed. The ethic committee of the CHU de Québec–Université Laval (CHUL) approved the study.

Trained research nurses took maternal blood pressure with validated automated devices for pregnant women (Microlife, BIOS Diagnostics 33603) that were calibrated at regular intervals during this study. Women were allowed to rest for 15 min before the measurement. During the examination, participants were in a sitting position with their arms supported at the level of their heart. Blood pressure was measured with an adult cuff at the right size for the patient on both arms simultaneously. Automated devices were taking a series of recordings (minimum of 2) until the difference between two consecutive readings was least than 10 mmHg in systolic or 6 mmHg in diastolic blood pressure to obtain the best validity of the measurement as recommended by previous studies, a single measurement being associated to be a common error in blood pressure measurement [25,26]. The MAP was the average of both arms' computation of the addition of the systolic blood pressure and the double of diastolic blood pressure, divided by three. Measures of blood pressure taken in the research setting were not revealed to the participants or to their healthcare providers.

Outcomes included all HDP including PE as the primary outcome. A standard definition based on the American College of Obstetricians and Gynaecologists (ACOG) and the Society of Obstetricians and Gynaecologists of Canada (SOGC) guidelines was used [27,28]. The diagnosis of PE was based on the presence of gestational hypertension (GH) occurring at/or after 20 weeks' gestation in previously normotensive women with the presence of significant proteinuria and/or one or more adverse conditions and/or one or more severe complications. GH is defined as a systolic blood pressure of 140 mmHg or more and/or a diastolic blood pressure of 90 mmHg or more on at least two occasions 4 h apart. Proteinuria is defined as ≥0.3 g/d in a complete 24-h urine collection or ≥30 mg/Mmol urinary creatinine in a spot (random) urine sample or ≥1+ proteinuria on a urinary dipstick. Adverse conditions and severe complications include headache and visual symptoms, epigastric pain, fetal death, intra-uterine growth restriction, placental abruption, elevated liver enzyme, thrombocytopenia, and severe hypertension (> 160 mmHg of systolic blood pressure or > 105 mmHg of diastolic blood pressure). We used the gestational age at delivery to divide cases of PE into term (≥37 weeks), preterm (< 37 weeks) and early-onset (< 34 weeks) PE. A maternal-fetal medicine subspecialist blinded to all other research data reviewed all cases with an adverse pregnancy outcome to confirm the diagnosis.

We computed the multiples of the median (MoM) of MAP adjusted for gestational age. A graphical display of the relationship between CRL and MAP was produced. Receiver operating characteristic (ROC) curves analyses were performed. We estimated the screening performance of MAP for HDP by calculating the area under the curve (AUC). Logistic regression was used to develop predictive models including maternal characteristics (age, body mass index, ethnicity, smoking, method of conception, chronic diseases [diabetes, hypertensive disorder, renal disease, rheumatoid arthritis and inflammatory diseases]) for term, preterm and early-onset PE. Two-tailed P-values < 0.05 were used to describe a statistically significant association for all analyses. The statistical software packages SAS (Version 9.3, SAS Institute Inc. Cary, NC, USA) was used for all data analyses.

3. Results

We recruited 5005 nulliparous women among which 4749 were eligible. Exclusions were based on age < 18 years old (n = 2), multiple gestations (n = 29), medical termination of pregnancy (n = 46) and baseline visit outside the targeted gestational age window (n = 179). Maternal characteristics are shown in Table 1. We lost 49 participants to follow-up (1%). Therefore, 4700 (99%) participants remained for the analyses. Out of them, 491 (10.3%) developed an HDP, including 250 (5.3%) GH without PE; 241 (5.1%) PE; 33 (0.7%) preterm PE and 10 (0.2%) early-onset PE.

Table 1
Characteristics of the study population.

	No GH or PE	GH	PE	p-value
Maternal age (years)	28.9 ± 4.1	28.3 ± 4.4	29.3 ± 3.9	0.009
Baseline gestational age (weeks)	13.0 ± 0.6	13.0 ± 0.7	13.0 ± 0.6	0.54
Baseline crown-rump length (mm)	67 ± 9	67 ± 9	67 ± 8	0.58
Baseline maternal body mass index (kg/m^2)	24.7 ± 4.7	28.3 ± 4.4	29.3 ± 3.9	< 0.0001
Gestational age at delivery (weeks)	39.5 ± 2.0	39.4 ± 1.5	38.3 ± 2.5	< 0.0001
Ethnicity				0.72
Caucasian	4038 (96.0%)	245 (97.2%)	232 (96.3%)	
Afro-American	57 (1.4%)	1 (0.4%)	1 (0.4%)	
Asian	33 (0.8%)	2 (0.8%)	2 (0.8%)	
First Nations, mixed or others	69 (1.6%)	4 (1.6%)	6 (2.5%)	
Missing	10 (0.2%)	0	0	
Smoking	306 (7.4%)	17 (6.8%)	13 (5.5%)	0.27
History of chronic disease				
Diabetes	8 (0.2%)	0	1 (0.4%)	0.58
Hypertensive disorder	13 (0.3%)	4 (1.6%)	6 (2.5%)	< 0.0001
Renal disease	92 (2.2%)	7 (2.8%)	5 (2.1%)	0.88
Rheumatoid arthritis or other inflammatory diseases	69 (1.6%)	2 (0.8%)	1 (0.4%)	0.07

Proportions are reported as n (%) and continuous variables as mean ± standard deviation.

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