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A prospective study to evaluate the role of mean arterial pressure in predicting preeclampsia

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Abstract

Background: Diagnosing a woman's condition as "mild preeclampsia" is not helpful because it is a progressive disease, progressing at different rates in different women and appropriate care requires frequent re-evaluation for good clinical outcome.

Aims and Objective: To assess the role of Mean arterial pressure (MAP) at 11-14 weeks of gestation in prediction of preeclampsia.

Materials and Methods: Seventy eight pregnant women with 11- 14 weeks of gestation were studied at the Department of Obstetrics and Gynaecology, King George Medical University, Lucknow from September 2014 to August 2015. The women who developed preeclampsia and related complications at the end of pregnancy were grouped under GROUP 1 (n=29) and the women who did not develop any of these complications were kept under GROUP 2 (n=49). After routine antenatal examination, MAP was measured in each patient. A detailed questionnaire was filled which included demographic factors, obstetric history, past and family history, blood group and body mass index.

Results: The MAP multiple of the median (MoM) of the women who developed preeclampsia and related complications was 1.01 ± 0.08 and that of women who did not develop complications was 0.97 ± 0.10 ($p = 0.073$). The mean MAP MoM of the women who developed early onset and late onset preeclampsia was 1.07 ± 0.05 and 1.04 ± 0.14 respectively. When compared to women who did not develop complications, the value was statistically significant for early onset preeclampsia ($p = 0.029$) but not for late onset preeclampsia ($p = 0.299$). The mean MAP MoM of the women who developed IUGR was 1.0058 ± 0.0806 . The comparison of this to women who did not develop complications was not statistically significant ($p = 0.2087$). The area under the curve of the receiver operating curve (ROC) of MAP MoM was 0.618 ($p = 0.032$).

Conclusion: MAP was found to be a significant tool for predicting preeclampsia in women at 11-14 weeks of gestation.

Keywords: Preeclampsia, mean arterial pressure, pregnant women

Introduction

As pathophysiological changes in preeclampsia are believed to occur in early pregnancy before the clinical manifestation of the disease are seen, there has been a quest for the methods to predict it early so as to prevent its development or at least prevent adverse outcomes^[1].

Most guidelines on prenatal care recommend that the risk of developing preeclampsia be assessed based on maternal characteristics such as maternal age, ethnicity, body mass index (BMI), nulliparity, family or personal history of preeclampsia and chronic diseases, but these characteristics have equivocal predictive values^[2].

Various modalities have been explored like mean arterial pressure, uterine arterial pressure and serum markers like PAPP-A, placental growth factor etc. which have shown promising results^[3,4].

In present study we assessed the role of mean arterial pressure (MAP) at 11-14 weeks of gestation in prediction of preeclampsia

Material and Methods

Present prospective study included 78 pregnant women at the Department of Obstetrics and Gynaecology, King George Medical University, Lucknow over a period of one year from September 2014 to August 2015.

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Pregnant women were enrolled in the antenatal OPD at 11-14 weeks of gestation and followed till delivery.

All pregnant women with singleton pregnancy who attended the antenatal OPD at 11-14 weeks of gestation and were willing for follow up were enrolled after taking informed consent. Women who did not give consent, with multifetal pregnancy and women with essential hypertension were excluded from the present study.

The women who developed preeclampsia and related complications (gestational hypertension, small for gestational age (SGA) babies, abruption placentae or intrauterine fetal demise) at the end of pregnancy were grouped under GROUP 1 and the women who did not develop any of these complications were kept under GROUP 2. Out of the 78 women, 29 women (37.18%) developed the above mentioned complications and constituted Group 1 and the other 49 (62.82%) were Group 2.

A detailed questionnaire was filled which included demographic factors, obstetric history, past and family history, blood group and body mass index.

After routine antenatal examination mean arterial pressure was measured which is defined as the average arterial pressure during a single cardiac cycle.

MAP was calculated by the formula: $MAP = DBP + 1 (SBP - DBP)/3$

The values of MAP was matched for age of women, gestational age, height, weight, racial origin, type of conception, obstetric history, past history, family history of preeclampsia and history of smoking and converted to multiples of median (MoMs) using the Fetal Medicine Foundation algorithm for prediction of preeclampsia.⁵

The women enrolled in the study were then kept under follow up for the subsequent development of preeclampsia, gestational hypertension, small for gestational age (SGA) babies, placental abruption and other maternal or fetal complications. The gestational age of development of these complications was recorded along with their severity.

At delivery gestational age at delivery, type of delivery, indication of Caesarean section and baby details (weight, sex, APGAR score, NNU admission) were recorded.

The data was analyzed using SPSS (Statistical Package for Social Sciences) Version 20.0 statistical analysis software. For the categorical data in the study, relative risk was calculated and chi square test was applied. For the MAP and Mean PI, mean \pm SD was calculated and these were analyzed by student t test. 95% confidence interval was calculated for all the parameters.

Results

Out of the 29 women who developed complications, 9 (11.54%) developed pre-eclampsia. Of these, 6 (7.69%) developed early onset preeclampsia (before 34 weeks of gestation) and 3 (3.85%) developed late onset preeclampsia (after 34 weeks of gestation). A total 25 (32.05%) women

had IUGR babies. Of these, 5 women (6.41%) had early onset preeclampsia with IUGR and none of the women with late onset preeclampsia were associated with IUGR.

There was one case of abruption placentae at 35 weeks of gestation in a woman with early onset preeclampsia and IUGR and one antepartum intrauterine death occurred in a woman with early onset severe pre-eclampsia with severe IUGR at 29 weeks 4 day of gestation.

The mean age of the women who developed complications was 26.48 years and in those who did not it was 25.61 years ($p = 0.33$). The mean BMI of Group 1 was 24.36 kg/m^2 and of those in Group 2 was 24.59 kg/m^2 ($p = 0.803$).

Primipara were those women for whom it was the first pregnancy that led to delivery of a viable fetus (≥ 24 weeks of gestation) and multipara were those women who had delivered a viable fetus (≥ 24 weeks of gestation) before this pregnancy. The relative risk of developing complication in a primipara was 0.65 (95% CI- 0.36-1.15) and that in a multipara was 1.54 (95% CI = 0.869 – 2.728) ($p = 0.2$).

Relative risk of developing complications in the present pregnancy if there was a previously complicated pregnancy (in multiparous women) was 1.54 (95% CI – 0.588-3.098) ($p = 0.88$)

The 32 multiparous women enrolled in the study were evaluated for presence of complications in the previous pregnancy which included preeclampsia, eclampsia, IUGR, abruption placentae, antepartum IUD. The relative risk of developing complications in the present pregnancy in these was 1.36 (95% CI = 0.588-3.098) ($p = 0.88$).

Eighteen of the enrolled women had a family history of hypertension and/or diabetes mellitus. Out of these, 7 developed complications and 11 did not develop complications. The relative risk of development of preeclampsia and related complications in women who have one or both parents affected by hypertension or diabetes compared to women with no such family history was 1.06 (95% CI = 0.54 – 2.068) ($p = 0.915$). Only one woman enrolled had family history of preeclampsia in her elder sister. She developed non- severe preeclampsia at 28 weeks of pregnancy.

Amongst the women enrolled, 31 (39.74%) had B positive blood group, 25(32.05%) had O positive blood group, 12 (15.38%) had A positive blood group, 4 (5.12%) had AB positive blood group and 5 women (6.4%) had a RH negative blood group (3 were O negative and one each of A negative and B negative). The relative risk of developing preeclampsia and related complications according to the blood groups were 0.8(95% CI = 0.682 – 2.192) for B positive blood group ($p = 0.623$), 1.3 (95% CI = 0.431 – 1.479) for O positive ($p = 0.679$), 0.88 (95% CI = 0.373 – 2.075) for A positive ($p = 0.98$), 1.37(95% CI = 0.492 – 3.82) for AB positive ($p = 0.99$), 1.08 for Rh negative pregnancy ($p = 0.731$, 95% CI = 0.355 – 3.296). No blood group was statistically significantly associated with development of complications.

Table 1: Comparison of mean MAP MoM in different groups

Variable	Group 1					Group 2 N=49
	All (n=29)	PE (n=9)	IUGR (n=25)	EO-PE (n=6)	LO-PE (n=3)	
Mean MAP	1.01 \pm 0.08	1.06 \pm 0.09	1.00 \pm 0.08	1.07 \pm 0.05	1.04 \pm 0.14	0.97 \pm 0.10
P value	0.073	0.019	0.208	0.029	0.299	
CI (95%)	0.98 – 1.05	0.01 – 1.15	0.01 – 0.07	0.009 – 0.178	-0.58 – 0.18	

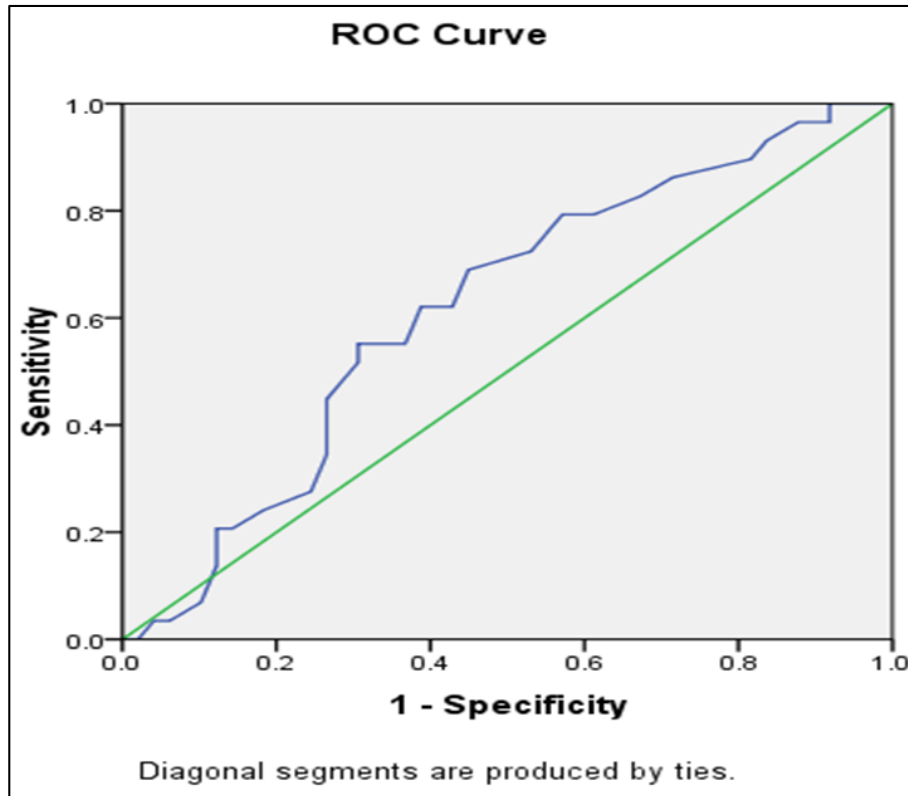


Fig 1: Receiver Operating Characteristic (ROC) curve for MAP MoM

Area under curve (AUC) = 0.618, (95% CI = 0.492 – 0.745),
Cut off for MAP MoM= 1.025

Discussion

Reason for choosing time frame of 11-14 weeks was that it is at this gestation an integrated study combining data from maternal characteristics and history with findings of sonographic and biochemical tests can be carried out. This can define the patient-specific risk for a wide spectrum of pregnancy complications, including aneuploidies, fetal defects, miscarriage and fetal death, preterm delivery, fetal growth restriction and macrosomia, gestational diabetes, hypothyroidism and preeclampsia (37-43) [6-9]. mean arterial pressure was calculated from the blood pressure of both arms. The values of MAP was converted to their respective multiples of median (MoMs) which was matched for age of women, gestational age, height, weight, racial origin, type of conception, obstetric history, past history, family history of preeclampsia and history of smoking using the Fetal Medicine Foundation algorithm for prediction of preeclampsia.

It was found that 29 (37.18%) of these enrolled women developed preeclampsia and related complications (intrauterine growth restriction, abruption placentae and intrauterine death). Preeclampsia developed in 9 (11.5%) women. This is comparable to the world average of 5-10% but is higher than the Indian average of 4.60% (as per WHO 2014). This discrepancy is there because this is a tertiary care centre and thus high risk cases get referred here from peripheral areas. Of these, 6 (7.69% of total) developed early onset preeclampsia i.e before 34 weeks of gestation and 3 (3.85% of total) developed late onset preeclampsia i.e after 34 weeks of gestation. Incidence of IUGR in India is 39.71% and in this study it was found to be 32.05% (developed in 25 women). Of these, 5 women had early

onset preeclampsia with IUGR and none of the women with late onset preeclampsia were associated with IUGR.

Demographic factors of the women in the two groups were compared and the women who developed the above mentioned complications were found to be demographically similar to women who did not develop complications. Various studies have found that women with antiphospholipid antibodies, a history of pre-eclampsia, preexisting diabetes, multiple pregnancy, family history, nulliparity, a raised BMI before pregnancy or at booking, maternal age > 40, renal disease and hypertension all increased the risk of a woman developing pre-eclampsia [10-12]. These results were not reciprocated in this study because only a limited number of women were enrolled. Additionally they were recruited from the general population and did not have preexisting high risk factors like chronic hypertension and multifetal pregnancy.

Mean arterial pressure (MAP) is the average arterial pressure during a single cardiac cycle. It is related to the maternal vascular adaptation. This was taken as a parameter for this study as studies show that when blood pressure is measured in the first or second trimester of pregnancy, the mean arterial pressure is a better predictor for pre-eclampsia than systolic blood pressure, diastolic blood pressure, or an increase of blood pressure [13].

The mean MAP (mean arterial pressure) MoM of the women who developed preeclampsia and related complications was 1.01 ± 0.08 and that of women who did not develop complications was 0.97 ± 0.10 . The comparison of these was not statistically significant ($p = 0.073$).

The area under the curve (AUC) of the receiver operating curve (ROC) of MAP MoM was 0.618 (95% CI = 0.492 – 0.745) for a cut off of 1.025. The sensitivity of MAP MoM at 11-14 weeks of pregnancy for predicting complications is 55.2% and specificity is 69.4%. This is statistically significant ($p = 0.032$).

A study done at Kings College Hospital, London, ¹⁴ found that the mean MoM MAP at 11⁺⁰ to 13⁺⁶ weeks in those developing preeclampsia did not change significantly with gestation at delivery, and, therefore, measurement of MAP is equally effective in screening for early and late disease. The area under the receiver operating characteristic curve (AROC) for the detection of PE MAP alone (AROC: 0.734; P = 0.001).

A nested case control study done in Netherlands by Kuc *et al.* ^[15] in 2013 found first-trimester MAP to be one of the most important predictors of preeclampsia. In case of EO-PE, MAP was significantly higher (cut-off - 1.04 MAP MoM, p = 0.0001) compared to controls. Also higher MAP MoM were found in LO-PE pregnancies (MAP -1.05 MoM, p= 0.0001).MAP was again found to be statistically significantly increased in the EO-PE SGA group as compared to controls (1.04 MoM, p = 0.01) but not for SGA alone.

Conclusion

In present study it was found that MAP MoM was statistically significant for predicting preeclampsia as a whole (with or without SGA) but not statistically significant for development of SGA babies.

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