Original Article

Flow-mediated Dilation of the Brachial Artery in Women with Hypertensive Disorders of Pregnancy

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Abstract

Background: Hypertensive disorder of pregnancy (HDP) comprise chronic hypertension, gestational hypertension, preeclampsia/eclampsia, and preeclampsia superimposed on chronic hypertension. HDP complicate up to 10% of pregnancies worldwide and carry significant risks of maternal and perinatal morbidity and mortality. The aim of this study was to evaluate the derangement and characteristics of brachial artery flow-mediated dilation (BAFMD) in women with HDP. **Methods:** The BAFMD of the right brachial artery of 80 women with HDP (pregnant HDP), 80 normotensive pregnant women (pregnant non-HDP), and 80 healthy nonpregnant women (nonpregnant controls) was evaluated with B-mode ultrasound. The age, blood pressure, body mass index (BMI), brachial artery diameter, and BAFMD of the participants were compared. $P \leq 0.05$ was statistically significant. **Results:** The pregnant HDP group had significantly lower mean BAFMD compared to pregnant non-HDP and nonpregnant controls (6.9% ± 2.53% vs. 8.32% ± 3.4% vs. 9.4% ± 2.68%; $P < 0.001$). There was no significant difference between the mean BAFMD of the pregnant HDP subgroups: preeclampsia (5.81% ± 1.7%) versus gestational hypertension (6.43% ± 3.02%); $P = 0.57$. BAFMD diminished with advancing gestational age in both the pregnant HDP and pregnant non-HDP groups. On regression analysis, BAFMD was a poor marker for HDP, while BMI was an independent predictor for HDP. **Conclusion:** Even though HDP were associated with significantly diminished BAFMD, it was not a good marker for HDP.

Keywords: Brachial artery, endothelial dysfunction, flow-mediated dilation, hypertensive disorders of pregnancy, ultrasound

Introduction

Hypertensive disorders of pregnancy (HDP) comprise chronic hypertension, gestational hypertension, preeclampsia/eclampsia, and preeclampsia superimposed on chronic hypertension.[1] HDP complicate up to 10% of pregnancies worldwide and carry significant risks of maternal and perinatal morbidity and mortality.[2-5] It also predisposes to other maternal cardiovascular, and sometimes cerebrovascular, complications later in life.[6] Women with HDP have higher risks of postpregnancy hypertension, ischemic heart disease, and stroke.

In a bid to accommodate the growing fetus, normal pregnancy is accompanied by changes in the maternal cardiovascular system. There is increased vasodilation with the endothelium playing a key role in regulating the vascular tone.[7] The maintenance of vascular tone involves multiple vasoconstrictors and vasodilators such as nitric oxide, prostaglandins, endothelium-derived relaxing factor, thromboxane A, and endothelin 1, of which nitric oxide is the major mediator of vasodilation.[8,9] Reduced nitric oxide level is associated with endothelial dysfunction which results in impaired arterial wall response.[7,8]

Endothelial dysfunction, a precursor of atherosclerosis, is one of the earliest consequences of systemic hypertension. It plays a major role in onset, progression, and clinical manifestation of atherosclerosis.[10] This is also applicable to pregnant patients with hypertensive disorders.[11] Although symptoms of preeclampsia resolve in a number of weeks after

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delivery, a growing body of literature suggests that maternal vascular dysfunction may persist for years, with increased risk of developing cardiovascular disease and systemic hypertension.[12]

The ultrasonographic brachial artery flow-mediated dilation (BAFMD) test is a standard examination of endothelial activity. Positive result is defined as an augmentation of brachial artery diameter (BAD) in reaction to hyperemia. The assessment of endothelial function through BAFMD represents a functional bioassay for endothelium-derived bioavailability of nitric oxide in humans.[13-15]

The noninvasive ultrasound-based BAFMD technique has been widely accepted as a means of evaluating vascular endothelial function, comfortably replacing other invasive options of assessing endothelial function. It is sensitive, reliable, relatively cheap, convenient, and safe for the patient. BAFMD involves the suprasystolic occlusion of blood flow to the arm, with consequent hyperemia and increased laminar shear forces parallel to the axis of the vessels. This initiates a cascade of events leading to the synthesis of nitric oxide by endothelial nitric oxide synthase. The nitric oxide will subsequently lead to vasodilation which can be assessed by ultrasound as percentage change in luminal diameter of the brachial artery.[14]

This study investigated the differences in BAFMD (as a surrogate marker of endothelial function) between pregnant HDP, pregnant non-HDP, and nongravid normotensive women in a sub-Saharan African population.

Materials and Methods

Study design and participants recruitment

This cross-sectional study was carried out at the Department of Radiology, Obafemi Awolowo University Teaching Hospitals Complex, Ile-Ife, Osun State, Nigeria, from November 2019 to December 2020. Approval was granted by the Research and Ethics Committee of our tertiary hospital (Approval Number: ERC/2019/04/12). Written informed consent was obtained from all the participants.

There were three study groups: 80 pregnant women with HDP (pregnant HDP), 80 pregnant women without HDP (pregnant non-HDP), and 80 volunteer age-matched healthy nonpregnant women. The pregnant women were recruited consecutively from the antenatal clinic of the hospital. The pregnant HDP group comprised pregnant women at gestational ages of 20–40 weeks with hypertension, i.e., blood pressure ≥140/90 mmHg, with or without proteinuria (i.e., 300 mg/24 h or > dipstick+). The pregnant non-HDP group comprised women with known last menstrual period (LMP) and uncomplicated pregnancies at 20–40 weeks of gestational age.

The exclusion criteria were diabetes mellitus, gestational diabetes, sickle cell disease, thyroid disease, previous history of coronary heart disease or stroke (including a history of angina, myocardial infarction, or abnormal stress test), dyslipidemia, HIV-positive status, history of systemic chemotherapy, radiation therapy or long-term systemic steroids use, smoking, alcohol intake, history of vasoactive drugs use, and strenuous exercise 8 h before the ultrasound evaluation.

Clinical and laboratory evaluation

Demographic and clinical data included age, LMP (to estimate the gestational age in pregnant women), and day of cycle in the nonpregnant women. Past medical history was obtained to exclude participants with exclusion criteria. Recent urinalysis result was documented for the pregnant HDP group. Their weight (kilogram) and height (meter) were measured using a mechanical physician weighing scale with attached stadiometer (Model ZT160, China). The body mass index (BMI) was then calculated for the participants using the formula BMI (kg/m^2) = $weight/height^2$.

The blood pressure of the participants was measured by one examiner after 15 min of rest. An appropriate bladder cuff of an analog mercury sphygmomanometer was applied circumferentially across 80% or more of the left arm. After inflation, the mercury column was deflated at a rate of 2–3 mmHg/s. The first and last audible sounds were taken as systolic and diastolic blood pressures, respectively, and their measurements were given to the nearest 2 mmHg.

Ultrasonographic brachial artery flow-mediated dilation

All the participants refrained from caffeine products and strenuous exercise before the procedure. They were evaluated between the hours of 7 am and 10 am (after a fast) using a General Electric ultrasound scanner equipped with a 7.5–12 MHz transducer and Doppler functionality (Versana Essential GE Medical Systems [China] Co., Ltd). All BAFMD ultrasound measurements were performed by one sonologist (a final-year radiology specialty senior registrar supervised by a radiology consultant/attending) using a previously published standard protocol in the literature. The participants were made to lie in the left lateral position for 10 min at room temperature before baseline measurements were obtained. The right arm was extended on a pillow at heart level. Acoustic gel was applied to the arm. The right brachial artery was visualized on the transverse plane superior to the cubital fossa – color Doppler was utilized to localize the artery where necessary.

Light pressure was applied on the transducer with the artery retaining its size and shape while the vein collapsed. Pulsed Doppler was further used to confirm the artery as having a pulsatile flow while the vein demonstrated monophasic flow. The transducer was rotated 90° at 5 cm above the cubital fossa to display the brachial artery in longitudinal section. This longitudinal still image in the distal third of the upper arm, above the elbow, was used to measure the brachial artery anteroposterior (AP) diameter from the near endothelial-luminal surface to the distal luminal surface (baseline diameter, D1). An indelible ink was used to mark the lower end of the transducer to ensure the consistency of the area measured.[16]

The forearm blood pressure cuff was placed immediately distal to the medial epicondyle (about 3 cm distal to the elbow joint) and inflated to 200 mmHg blood pressure for 5 min. Brachial artery AP diameter measurement was restarted seconds after cuff deflation and continued at least 3 min after deflation.[16,17] In all, at least four postdeflation measurements were taken from serial images frozen at 30 s, 45 s, 60 s, and 90 s.[10,15] The average diameter of the postserial measurements was obtained as the brachial artery peak diameter (postocclusion value, D2) at the same spot where the first measurement (D1) was taken before cuff occlusion. Once all the measurements had been taken, the cuff was removed and the coupling gel is cleaned off the participant's arm. The BAFMD was calculated using the following formula:[10,15]

$$\text{BAFMD} = \frac{\text{D2 - D1}}{\text{D1}} \times 100$$

Data and statistical analysis

The study data were analyzed using the IBM SPSS Statistics for Windows, version 20 (IBM Corp., Armonk, N.Y., USA). Data normality was tested with the Kolmogorov–Smirnov test. Continuous variables such as age, BAFMD, and estimated gestational age (EGA) were presented as mean ± standard deviation. The mean values of continuous data were compared with the independent samples *t*-test. Categorical variables were compared using Chi-square test, while analysis of variance was used to compare the BAFMD values across the study groups. Pearson's correlation was used to assess the correlation between the BAFMD and other parameters. The degree of correlation was graded as follows: r = 0–0.2: very low/negligible and probably meaningless correlation; $r \geq 0.2$–0.4: low correlation; $r \geq 0.4$–0.6: moderate correlation; $r \geq 0.6$–0.8: high correlation; and $r \geq 0.8$–1.0: excellent/very high correlation.[18] Binomial logistic regression was done to evaluate the degree of relationship between BAFMD, BAD, BMI, maternal age, and EGA as predictive markers of hypertension in pregnancy. $P \leq 0.05$ was considered statistically significant.

Table 1: Characteristics of the study population

Variables	Nonpregnant control (n=80), n (%)	Pregnant non-HDP (n=80), n (%)	Pregnant HDP (n=80), n (%)	P*
Maternal age (years), mean±SD	30.21±4.41	30.19±4.40	30.19±4.45	0.999
EGA (weeks), mean±SD	NA	29.84±5.16	31.84±4.16	0.798
Parity				
None	15 (18.8)	24 (30.0)	10 (12.8)	<0.0001
One	11 (13.8)	17 (21.2)	29 (37.2)	
Two	19 (23.8)	29 (36.2)	24 (30.8)	
Three	23 (28.8)	8 (10.0)	11 (16.7)	
Four	11 (13.8)	2 (2.5)	2 (2.6)	
Six	1 (1.2)	0	0	

*ANOVA. EGA: Estimated gestation age, HDP: Hypertensive disorders of pregnancy, ANOVA: Analysis of variance, SD: Standard deviation, NA: Not available

Table 2: Blood pressure and anthropometric parameters of the study groups

Variables	Nonpregnant control (n=80)	Pregnant non-HDP (n=80)	Pregnant HDP (n=80)	P*
SBP (mmHg)	114.72±7.25	120.25±5.28	150.03±7.29	<0.0001
DBP (mmHg)	69.63±10.69	74.39±7.30	89.57±11.36	<0.0001
Height (m)	1.61±0.07	1.63±0.06	1.61±0.05	0.073
Weight (kg)	68.00±9.38	75.58±7.87	85.59±6.39	<0.0001
BMI (kg/m^2)	26.24±4.14	28.49±3.18	32.94±2.25	<0.0001
BMI range, n (%)				
Normal	41 (51.2)	8 (10.0)	0	
Overweight	24 (30.0)	50 (62.5)	9 (11.2)	
Obese	15 (18.8)	22 (27.5)	71 (88.8)	

*ANOVA. BMI: Body mass index, DBP: Diastolic blood pressure, HDP: Hypertensive disorders of pregnancy, SBP: Systolic blood pressure, ANOVA: Analysis of variance

Results

A total of 240 participants were studied. The detailed demographic and clinical characteristics of the study participants are shown in Tables 1 and 2. Of the 80 participants in the pregnant HDP group, 14 (17.5%) had gestational hypertension, while 66 (82.5%) had preeclampsia. Fourteen (17.5%) of the participants with HDP tested negative for urinary protein, 42 (52.5%) had dipstick$^+$ (30 mg/dL of protein), while 24 (30%) had dipstick^{++} (100 mg/dL of protein) on dipstick urinalysis.

The BADs and BAFMD of the study population are shown in Table 3. There were no significant differences between the mean preinflation BAD of the study groups. Conversely, there were significant differences between the mean postinflation BAD (nonpregnant control > pregnant non-HDP > pregnant HDP). *Post hoc* analysis established a significant difference between the pregnant non-HDP and pregnant HDP groups [Table 3]. Furthermore, *post hoc* test yielded significant differences between the BAFMD of the pregnant non-HDP and pregnant HDP, as well as the nonpregnant control and pregnant non-HDP groups [Table 3]. There was no statistically significant difference in BAFMD between the subgroups of the pregnant HDP group (preeclampsia vs. gestational hypertension).

Figure 1 is a cluster bar chart which shows an inverse trend between BAFMD and advancing EGA in the pregnant participants. Table 4 shows the association between the EGA and various clinical parameters in the pregnant HDP group. Of all the clinical parameters, only BMI and preinflation BAD correlated significantly with EGA [Table 4]. In the pregnant HDP group, BAFMD showed varying degrees of correlation with EGA ($r = -0.43$; $P < 0.001$) and preinflation

BAD ($r = -0.33$; $P < 0.003$). In the pregnant non-HDP group, BAFMD correlated with EGA ($r = -0.41$; $P < 0.001$), preinflation BAD ($r = -0.37$; $P = 0.001$), and diastolic blood pressure ($r = -0.23$; $P = 0.04$). The BMI ($r = -0.41$; $P < 0.001$) and preinflation BAD ($r = -0.27$; $P = 0.02$) correlated with BAFMD in the nonpregnant controls [Table 5]. A scatterplot of BAFMD against EGA yielded a statistically significant moderately negative relationship in both the pregnant HDP ($r = -0.43$; $P < 0.001$) and pregnant non-HDP ($r = -0.41$; $P < 0.001$) groups; i.e., BAFMD decreased with advancing gestational age in the pregnant HDP group [Figure 2].

Two logistic regression models were tested to assess the utility of BAFMD, BAD, BMI, maternal age, and EGA as predictive markers of hypertension in pregnancy compared to pregnant non-HDP and nonpregnant controls. The first binomial logistic regression model was statistically significant for pregnant HDP versus nonpregnant controls ($\chi^2 = 88.53$ and $P < 0.001$), explained 56.7% (Nagelkerke R^2) of the variance in pregnant HDP patients, and correctly classified 84.4% of the cases [Table 6]. The second model was also statistically significant for pregnant HDP versus nonpregnant controls ($\chi^2 = 108.69$ and $P < 0.00$), explained 65.7% (Nagelkerke R^2) of the variance in pregnant HDP patients [Table 6], and correctly classified 88.1% of the cases [Table 7]. The result showed that BAFMD, BAD, maternal age, and EGA were not independently associated with hypertension in pregnancy (no statistically significant odds ratio). In contrast, BMI was independently associated with hypertension in pregnancy in both the first model (odds ratio [OR]: 1.84, 95% confidence interval [CI]: 1.52–2.24; $P < 0.001$) and the second model (OR: 1.68, 95% CI: 1.42–1.99; $P < 0.001$).

Table 3: Pre- and postinflation brachial artery diameter and brachial artery flow-mediated dilation across the study groups

	Preinflation BAD (mm)	Postinflation BAD (mm)	BAFMD (%)
NPC	3.78±0.20	4.13±0.22	9.40±2.68
PnHDP	3.80±0.21	4.11±0.21	8.32±3.40
PHDP	3.79±0.17	4.05±0.18	6.89±2.53
*P***	0.785	0.033	<0.0001
NPC	3.78±0.20	4.13±0.22	9.40±2.68
PnHDP	3.80±0.21	4.11±0.21	8.32±3.40
*P**	0.510	0.585	0.028
PnHDP	3.80±0.21	4.11±0.21	8.32±3.40
PHDP	3.79±0.17	4.05±0.18	6.89±2.53
*P**	0.770	0.047	0.003
NPC	3.78±0.20	4.13±0.22	9.40±2.68
PHDP	3.79±0.17	4.05±0.18	6.89±2.53
*P**	0.675	0.011	<0.0001
PE (*n*=66)	3.80±0.16	4.01±0.15	5.81±1.70
GH (*n*=14)	3.81±0.14	4.06±0.17	6.43±3.02
*P**	0.835	0.562	0.570

*Student's *t*-test, **ANOVA. ANOVA: Analysis of variance, BAD: Brachial artery diameter, BAFMD: Brachial artery flow-mediated dilation, GH: Gestational hypertension, NPC: Nonpregnant control, PnHDP: Pregnant nonhypertensive disease of pregnancy, PHDP: Pregnant hypertensive disease of pregnancy, PE: Preeclampsia

Discussion

The mean BAFMD of the pregnant HDP was significantly diminished compared to the pregnant non-HDP group. This

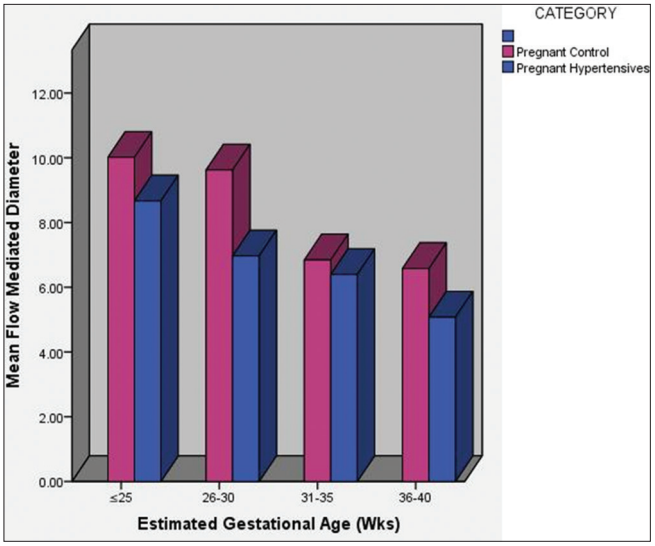

Figure 1: Cluster bar chart of BAFMD at different fetal gestational ages in the pregnant HDP and pregnant non-HDP groups. BAFMD: Brachial artery flow-mediated dilation, HDP: Hypertensive disorders of pregnancy

Table 4: Association between estimated gestational age and other parameters in the hypertensive disorders of pregnancy group

Variables	Gestational age (weeks), mean±SD				*F* ratio	*P**
	20–25 (*n*=15)	26–30 (*n*=30)	31–35 (*n*=20)	36–40 (*n*=15)		
SBP	147.18±5.48	148.73±7.05	148.76±6.72	157.64±7.14	1.486	0.225
DBP	90.06±5.37	89.91±4.56	86.44±17.69	95.27±6.47	1.179	0.323
BMI	32.61±2.34	33.04±2.53	32.79±2.11	33.58±1.98	6.756	<0.0001
Pre-BAD	3.73±0.21	3.84±0.14	3.79±0.16	3.82±0.19	6.179	0.001
Post-BAD	4.05±0.20	4.11±0.20	4.03±0.15	4.00±0.16	1.614	0.194
BAFMD	8.67±2.62	6.97±2.59	6.39±1.79	5.08±2.16	0.507	0.679

*ANOVA. BAFMD: Brachial artery flow-mediated dilation, BMI: Body mass index, SBP: Systolic blood pressure, DBP: Diastolic blood pressure, BAD: Brachial artery diameter, Pre-BAD: Preinflation BAD, Post-BAD: Postinflation BAD, SD: Standard deviation

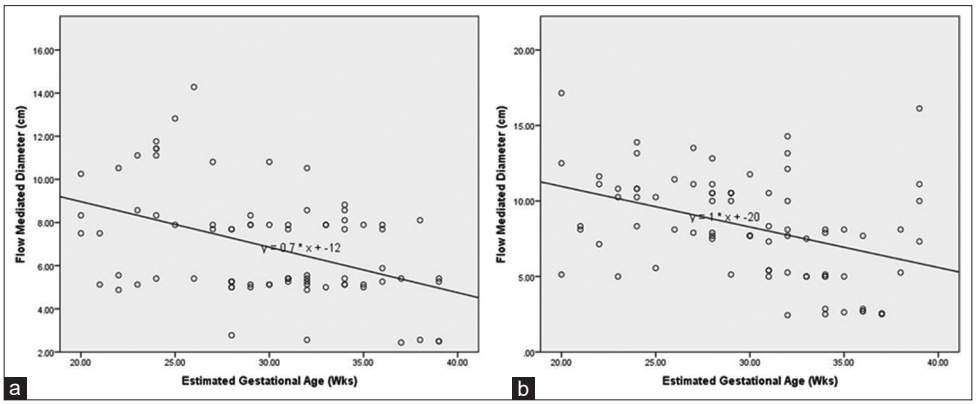

Figure 2: Scatterplot of correlation between BAFMD and EGA in the pregnant HDP (a) and pregnant non-HDP (b) groups. BAFMD: Brachial artery flow-mediated dilation, HDP: Hypertensive disorders of pregnancy, EGA: Estimated gestational age

Table 5: Correlation between brachial artery flow-mediated dilation and other variables in the study population

	PHDP		PnHDP		NPC	
	r	*P*	*r*	*P*	*r*	*P*
SBP	−0.215	0.064	−0.156	0.168	−0.202	0.082
DBP	−0.212	0.070	−0.228*	0.042	−0.175	0.134
BMI (kg/m^2)	−0.161	0.154	0.001	0.995	−0.413**	<0.0001
EGA	−0.431**	<0.0001	−0.407**	<0.0001	NA	NA
Pre-BAD	−0.333**	0.003	−0.368**	0.001	−0.268*	0.016
Post-BAD	0.196	0.082	0.210	0.061	0.189	0.134

*Correlation is significant at the 0.05 level (two-tailed), **Correlation is significant at the 0.01 level (two-tailed). BMI: Body mass index, EGA: Estimated gestational age, NPC: Nonpregnant control, PnHDP: Pregnant nonhypertensive disorders of pregnancy, PHDP: Pregnant hypertensive dilation of pregnancy, SBP: Systolic blood pressure, DBP: Diastolic blood pressure, BAD: Brachial artery diameter, Pre-BAD: Preinflation BAD, Post-BAD: Postinflation BAD, NA: Not applicable

Table 6: Binomial logistic regression for distinguishing the study groups

	Model I PHDP versus PnHDP		Model II PHDP versus NPC	
	OR (95% CI)	*P*	OR (95% CI)	*P*
BAFMD	0.19 (0.16–2.40)	0.203	0.25 (0.01–6.52)	0.403
Pre-BAD	0.00 (0.00–4.19)	0.248	0.00 (0.00–1.36)	0.434
Post-BAD	3.35 (0.00–8.32)	0.254	1.11 (0.00–6.34)	0.454
BMI	1.84 (1.52–2.24)	<0.0001	1.68 (1.42–1.99)	<0.0001
Age	0.97 (0.88–1.07)	0.547	0.92 (0.82–1.02)	0.108
EGA	0.92 (0.84–1.01)	0.087	NA	
Nagelkerke R^2	0.567		0.657	
χ^2	88.53	<0.0001	108.69	<0.0001

OR: Odds ratio, BAFMD: Brachial artery flow-mediated dilation, BMI: Body mass index, EGA: Estimated gestational age, NPC: Nonpregnant control, PnHDP: Pregnant nonhypertensive disorders of pregnancy, PHDP: Pregnant hypertensive disorders of pregnancy, BAD: Brachial artery diameter, Pre-BAD: Pre-inflation BAD, Post-BAD: Postinflation BAD, NA: Not applicable, CI: Confidence interval

finding is in tandem with the study of Liu *et al*.[19] who also recorded reduced BAFMD in pregnant HDP compared to pregnant non-HDP, reflecting reduced nitric oxide bioavailability and endothelial dysfunction. Similar findings were also noted by Yoshida *et al*.[15] and Guimarães *et al*.[20] Yoshida *et al*.[15] attributed this finding to the higher plasma levels of fibronectin in pregnant women with HDP compared to the pregnant non-HDP. For comparison, the BAFMD of women with gestational hypertension (6.43% ± 3.02%) and preeclampsia (5.1% ± 1.7%) in this study is similar to those reported by Quinton *et al.* (6.5% ± 4.1% and 5.3% ± 3.2%, respectively).[16] The mean BAFMD of the pregnant non-HDP was 8.32% ± 3.40%. This is similar to the value obtained by Savvidou *et al*. (8.84% ± 3.14%).[21]

BAFMD diminished with advancing gestational age in both the pregnant HDP group (the decrease appeared earlier in the late second to the early third trimester) and the pregnant non-HDP group (noted at >30 weeks of gestational age). The trend observed in the pregnant non-HDP group agrees with the results of Savvidou *et al*.[22] who reported an initial increase (between 10 and 30 weeks) in the BAFMD of healthy pregnant women which decreased to prepregnancy levels after 30 weeks of gestation.[22] Furthermore, Mannaerts *et al*.[23] noted that low-grade systemic inflammation, which possibly contributes to the alteration of endothelial function, is present in normal pregnancies and preeclamptic pregnancies.

The BAFMD decreased with increasing EGA in the pregnant HDP group. A similar pattern was reported by Quinton *et al*.,[24] Yoshida *et al*.,[15] Seeliger *et al.,*[25] and Malhotra *et al*.[26] It suggests that the brachial artery tends to reach maximum

Table 7: Predictive accuracy of selected parameters for hypertensive disease of pregnancy

Variable	PHDP versus PnHDP (%)	PHDP versus NPC (%)
NPV	82.5	82.5
PPV	86.4	93.8
Sensitivity	83.1	84.3
Specificity	85.7	93.0
Accuracy	84.4	88.1

NPC: Nonpregnant control, PHDP: Pregnant hypertensive disorders of pregnancy, PnHDP: Pregnant nonhypertensive disorders of pregnancy, NPV: Negative predictive value, PPV: Positive predictive value

dilation toward term pregnancy. Furthermore, the third trimester is a state of maternal pro-inflammatory response with increased levels of cytokines implicated in vascular inflammation. The endothelium acts as an integral part of the inflammatory response leading to an enhanced intravascular inflammatory response in pregnancy, especially in the third trimester.[26] In contrast, Dørup *et al.* reported that BAFMD increased from the first trimester reaching the highest value in the last trimester.[27] The conflicting results might be due to differences in the study populations (we recruited pregnant women from the late second trimester to term) and presumably better ultrasound equipment used in this study.

BAFMD had a significant negative correlation with BMI in the nonpregnant control group. This is consistent with a study of Olson *et al.* which noted reduced BAFMD in overweight and obese women.[28]

Preinflation BAD (baseline diameter) increased significantly with advancing gestational age in the pregnant participants. This trend was also observed by Malhotra *et al*.[26] and Quinton *et al*.[24] This gradual but statistically significant increase in vessel size with advancement of pregnancy may be attributed to increasing estrogen and progesterone levels toward the third trimester of pregnancy, which in turn causes an increase in nitric oxide production, with consequent increase in basal arterial diameter.[7,26]

On regression analysis, BAFMD, BAD, EGA, and maternal age were not independently associated with hypertension in pregnancy (making them nonpredictive markers). This outcome was quite different from studies where BAFMD was found to be a predictive marker of hypertension in pregnancy. Kamat *et al.*[29] found BAFMD measured at 18–24 weeks of gestational age to be a sensitive and useful early predictor of pregnancy-induced hypertension. Savvidou *et al.*[21] and Maruhashi *et al*.[30] also found BAFMD to be a predictive marker of preeclampsia and gestational hypertension. The differences in this result may be attributed to the differences in the population studied and gestational age at which participants were examined.

On the other hand, BMI appeared to be an independent predictive parameter for hypertension in pregnancy in this study. This observation is in consonance with the study of Briese *et al*.[31] who reported that obese primiparas were nearly seven times more likely (OR: 6.72; 95% CI: 6.3–7.17) to have preeclampsia or gestational hypertension than their nonobese counterparts. In another study, Hendler *et al*.[32] found that obese women were two times more likely (OR: 1.7; 95% CI: 1.3–2.3) to develop HDP. Similarly, Musa *et al*.[33] and Olayemi *et al*.[34] observed that women with a history of overweight and obesity in pregnancy had a significantly high tendency of developing HDP.

This study had a few limitations. First, it was not a population-based study, thus introducing some bias as participants were recruited from the teaching hospital. Second, antihypertensive therapy could not be discontinued temporarily in the pregnant HDP group before the measurement of BAFMD as doing so would be highly unethical.

To sum up, BAFMD correlated inversely with gestational age in the late second to the third trimester of pregnancy. BAFMD was not a good marker for hypertension in pregnancy.

Declaration of patient consent

The authors certify that they have obtained all appropriate patient consent forms. In the form the patient (s) has/have given his/her/their consent for his/her/their images and other clinical information to be reported in the journal. The patients understand that their names and initials will not be published and due efforts will be made to conceal their identity, but anonymity cannot be guaranteed.

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Nil.

Conflicts of interest

There are no conflicts of interest.

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