Arterial stiffness and wave reflection one year after a pregnancy complicated by hypertension

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Abstract

Hypertensive disorders of pregnancy (HDP) are associated with cardiovascular disease (CVD) later in life. We investigated the association of HDP with blood pressure (BP) and arterial stiffness 1-year postpartum. Seventy-four participants, 33 with an HDP and 41 with uncomplicated pregnancies, were examined using applanation tonometry to measure BP, carotid-femoral pulse-wave velocity (cfPWV) and augmentation index (AIx). On average, women with HDP had a 9 mm higher systolic BP (p<.01), 0.8 m/s faster cfPWV (p=.09), and 5.4% greater AIx (p=.09) at the 1-year exam. After adjustment for covariates, there was no significant difference in cfPWV between groups, while a 7.3% greater AIx (p<.05) remained. These findings suggest reduced endothelial function may be detected 1 year after HDP. Large prospective studies are needed to further understand of the contribution of arterial stiffness and endothelial dysfunction to the evolution of CVD disease after these complicated pregnancies.

Keywords

Hypertension; pregnancy; arterial stiffness

Introduction

Epidemiologic studies have demonstrated a link between hypertensive disorders of pregnancy (HDP; preeclampsia and gestational hypertension) and women's' future risk of hypertension and cardiovascular disease (CVD).[1–3] Specifically, estimates suggest that women with a history of preeclampsia are four times as likely to develop hypertension, and have twice the risk of early CVD and CVD mortality, when compared to women who had

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healthy pregnancies.[1,4] The American Heart Association (AHA) and others recommend this pregnancy history be considered a CVD risk factor.[5, 6]

Increasing arterial stiffness has been found, in diverse populations, to be an independent predictor of hypertension and CVD.[7–12] Carotid-femoral pulse wave velocity (cfPWV) is currently considered to be the gold standard measure of arterial stiffness.[9] Both cfPWV and wave reflection, as quantified by the augmentation index (AIx), appear to reflect global endothelial function.[11] If arterial stiffness contributes to future CVD risk among women who had pregnancy complications, it could be used to further identify those at greatest risk.[8] A number of small studies have revealed increased arterial stiffness during a preeclamptic pregnancy, thought to be related to endothelial dysfunction characteristic of preeclampsia.[12] However, it is unclear if this increase in stiffness persists following delivery[13] and if it reflects residual effects of the pregnancy on endothelial function.

In the present study we examine the relationship between HDP, blood pressure, and arterial stiffness, measured 12–18 months after delivery. In addition we investigate association of the pregnancy complication and stiffness measures with systemic biomarkers of inflammation thought to be associated with CVD risk.[14]

Materials and Methods

Subjects

The source population for this study was the Mom's Cohort, recruited in 2011–2012 from the postpartum service of a large community-based academic health care system. This prospective cohort included women who had a complicated pregnancy and women who were part of an uncomplicated pregnancy comparison group recruited using a stratified approach to ensure equal percentages of women who were obese prior to pregnancy (BMI>= 30 kg/m^2) and African American. Women were excluded if they had prepregnancy diabetes or were < 18 years of age at delivery.

Women were invited to attend a study visit 3 months and again 12 – 18 months after delivery if they had consented to future contact. The 3-month visit included a standardized interview and measures of blood pressure and weight. The 1-year visit was attended between 12 and 18 months postpartum, and included a standardized interview, anthropometry, and non-invasive measures of arterial stiffness using the SphygmoCor CVMS system (AtCor Medical, Sydney, Australia). All subjects were instructed to arrive after an overnight fast, to hold any antihypertensive or decongestive medications, and to avoid any products containing nicotine or caffeine. Blood and urine samples were collected and stored. The study was approved by the Christiana Care Health System Institutional Review Board and all subjects provided written informed consent.

In the analysis presented here we included all subjects who attended both the 3-month and 1-year visits. The exposed group was comprised of women had a clinical diagnosis of HDP, defined as new-onset BP >=140/90 mm Hg after 20 weeks gestation as identified by their admitting clinician during their hospital stay for labor and delivery and confirmed by review of inpatient medical records. Women with a diagnosis of chronic hypertension or known

hypertension prior to pregnancy were excluded. The unexposed control group included women recruited from the uncomplicated pregnancy comparison group.

Measures

Height and waist were measured to the nearest centimeter; weight was measured with patients' shoes and heavy clothing removed, using a mechanical weigh beam scale (Detecto, USA). Peripheral blood pressure was measured using a hospital-grade, automated oscillometric device (Welch Allyn, USA) with patient rested in a seated position using a standard protocol.

Applanation tonometry was used to record a radial arterial waveform by placing a high-fidelity strain-gauge transducer over the radial artery (Millar Instruments, USA). All studies were conducted by a single operator. Applanation tonometry has previously been shown to record a pressure wave that does not differ from waveforms obtained from intra-arterial measurements.[15] The radial waveform was calibrated from the brachial sphygmomanometric measurement of systolic and diastolic pressures as reported elsewhere.[16] A central aortic pressure wave was synthesized from the measured radial artery pressure waveform with the SphygmoCor CVMS system (AtCor Medical, Sydney, Australia), which uses a transfer function and is FDA approved. The use of a transfer function to approximate the central pressure wave from the radial wave has been validated using both intra-arterially[17–19] and noninvasively[20] obtained radial pressure waves. Central pressures and augmentation index (AIx) were obtained from the synthesized wave. AIx is an index of wave reflection and is influenced by arterial stiffness. AIx was defined as the ratio of reflected wave amplitude and pulse pressure, or $AI = (P_s - P_i)/(P_s - P_d)$, where P_s is peak systolic pressure, P_d is end-diastolic pressure, and P_i is an inflection point marking the beginning upstroke of the reflected pressure wave. AIx75 is calculated by the device, normalizing the measure to a heart rate of 75 bpm. Mean arterial pressure (MAP) was estimated from the pulse wave analysis. With the subject supine, carotid to femoral pulse wave velocity (cfPWV) was measured using a 3-lead ECG and applanation tonometry (SphygmoCor CVMS system).

Biomarkers hs-CRP, hs-TNF-α, and IL-6 were measured[12] using commercially available enzyme-linked immunosorbant assays (R&D Systems, Minneapolis, MN). All assays were performed according to the manufacturer's instructions at the University of Delaware Neuroendocrine Core Lab (Newark, DE).

Statistical Analysis

Data were nearly complete for all measures except cfPWV, where there were 8 missing measures, 4 from the HDP group and 4 from the unexposed control group; and AIx, where there were 2 missing measures from the unexposed control group. The characteristics of the subjects with missing data suggest their exclusion did not bias the results. Analyses of AIx were also conducted in the group of subjects with complete data for cfPWV to ensure there were no substantive differences in the results.

Descriptive statistics were used to examine characteristics of the subjects at baseline, 3 months, and at the 1-year follow-up visit, reporting means and standard deviations or

median and inter-quartile range where measures were not normally distributed. Significance testing, comparing the HDP group to the unexposed control group, was conducted using Student's t-test and chi-square for continuous and categorical measures respectively, setting an alpha of .05 to define significance. Rank-sum test was used for non-normally distributed measures. Univariate and multiple linear regressions models were also used to isolate the independent associations.

We found a strong relation of cfPWV to both BMI and waist circumference. This was likely related to measurement error introduced during the determination of the linear distances used for the velocity measure, as has been reported previously.[21] To account for this, and improve the precision of our measures, we used the method developed for the CRIC study[21] and used both the unadjusted (cfPWV) and adjusted (adjPWV) in the analyses.

All analyses were conducted using R version 2.15.3 (R Foundation for Statistical Computing, Vienna, Austria).

Results

We analyzed data for a total of 74 study subjects: 33 with a diagnosis of HDP and 41 from the unexposed control group. Overall, 24.3% of the women were African American and 73.0% were privately insured. More than half the subjects were obese prior to pregnancy: 54.5% in the HDP and 48.8% in the control group. The average age of women with HDP was 30.4 years, which was not significantly different that the control group average of 32.0 years. There were no significant differences between the groups in prepregnancy BMI, tobacco use, or the percentage of women who were African American or nulliparous. Women in the control group were less likely to be privately insured and more likely to report a positive family history of CVD (p<.05 for each). Those in the HDP group were more likely to have delivered preterm and the mean birth weight of their offspring was 248 grams (g) less (p<.05 for each).

At both the 3-month and 1-year visits, women weighed more on average than their self-reported prepregnancy weight, but there was no difference in the differences between the groups. There were also no significant differences between the groups in mean BMI, waist circumference, or percentage with a waist circumference of 35 inches (89 cm) or more at the 1-year visit.

As shown in Table 1, HDP was significantly associated with all measures of central, peripheral, and mean BP measures (p<.01) at both the 3-months and 1-year visits. These differences were substantial, with an 8.6 mm difference in peripheral SBP, a 5.5 mm difference in peripheral DBP, and a 7.5 mm difference in MAP, between groups. In addition, incident hypertension at 1 year was more common among women in the HDP group than in the control group (p<.01).

Table 2 shows the measures of arterial stiffness using the SphygmoCor CVMS system and inflammatory biomarkers collected at the 1-year visit. There were no significant differences in unadjusted cfPWV between groups, though the 0.8 m/s difference in the mean velocity among women who had HDP was nearly significant (p=.10) and potentially clinically

meaningful. After the adjustment for waist circumference, the difference between groups fell to 0.5 m/s, also of marginal significance (p<.1). The average unadjusted AIx was 5.4% higher in the HDP group (p<.1), and the 16.4% difference in the heart rate adjusted augmentation index (AIx 75) was statistically significant (p<.05). Central augmented pressure was also significantly higher (p<.05) in the HDP group.

Measures of the inflammatory biomarkers are shown in Table 3. On average, TNF-α was significantly higher among HDP affected women (p<.05), while differences in CRP or IL-6 were not significant. All were positively related to the unadjusted cfPWV with Spearman correlation coefficients of 0.13, 0.16, and 0.20, respectively. However, there were no significant relationships between the inflammatory biomarkers and the adjPWV or AIx.

Adjustment of the adjPWV for MAP using linear regression (results not shown) eliminated any difference between the group, as did further adjustment for age, BMI, race, family history of CVD, ever tobacco use and insurance type. The two factors which were significantly related to the adjPWV were MAP (p<.001) and age (p<.05).

There was a significant association of AIx with HDP after adjusting for heart rate and height. The results of the full multiple linear regression model of AIx, adjusted also for age, BMI, race, insurance, family history of CVD and a history of ever using tobacco, is shown in Table 3. The association with HDP remained significant (p<.05) and age at the time of the measure contributed significantly (p<.001).

Because prior studies suggest there may be differences in the associations among women had a clinical presentation consistent with preeclampsia (PE) rather than gestational hypertension (GHTN), we conducted a subgroup analysis. The 16 women with PE had a lower mean gestational age at delivery (p<.01), and their offspring had a lower mean birth weight (p<.01), than the 17 women with GHTN. When we examined the HDP subgroups of PE and GHTN in relation to adjPWV and AIx75, we found small differences between the groups. There was a higher mean AIx75 in the GHTN group (mean difference of 6%), and higher mean adjPWV measures in the PE group (mean difference of 0.2 m/s), but neither difference was statistically significant.

Discussion

We found differences in BP and AIx, but not PWV, among women one year after a pregnancy complicated by HDP when compared to similarly obese women with unaffected pregnancies. Through the use of an unexposed control group of women with a similar degree of adiposity, but who did not develop HDP, we were better able to account for the confounding effects of prepregnancy obesity, postpartum weight retention, and other CVD risk factors, than earlier studies. Though the sample size was modest, by accounting for both BP and measurement error related to abdominal adiposity we provide a more precise comparison of PWV between groups than previous studies. The significant difference between groups in AIx suggests this might be a more sensitive indicator of early changes in the cardiovascular system of women only 1 year postpartum.

Our main finding is the significantly higher AIx, after adjustment for confounders, but no significant difference in cfPWV when women with HDP were compared to equally obese women in the control group. Though PWV is the gold-standard non-invasive measure of arterial stiffness as measured by PWV, AIx depicts reflected wave characteristics of individuals representing a composite of arterial stiffness, vascular resistance, heart rate and left ventricular ejection. One potential mechanism for the increased wave reflection in HDP may be altered amplitude and site of wave reflections as a result of increased resistance, suggested by differences in MAP. Previous work has demonstrated that declines in endothelial function are associated with increased PWV and wave reflection,[22] thus impaired endothelial function in HDP may explain the increase in AIx.

The higher peripheral and central BP measures among women with HDP, and their elevated rate of incident hypertension, are consistent with prior studies.[23, 24] As expected, the inflammatory markers were positively correlated with cfPWV,[11, 25] but the magnitudes were small and not statistically significant. None of these markers were associated with AIx. Together these findings thus do not provide evidence to support inflammation outside of pregnancy in a mechanistic role.

A 2011 systematic review, and meta-analysis of 23 studies, examined the association between preeclampsia and arterial stiffness both during and after pregnancy.[13] A synthesis of the evidence suggested that measures of arterial stiffness and wave reflection are increased during pregnancy among women with both preeclampsia and gestational hypertension. However, data were conflicting as to whether these changes persist after delivery. Heterogeneity in findings across the studies may have been related to diseases severity. For example, small case-control studies that found women with early onset, but not late onset, preeclampsia had differences in PWV and AIx months postpartum when compared to women with healthy pregnancies.[26, 27] Other differences could be related to the lack of precision of the stiffness measures related to central adiposity as we found here and was found in a study of the CRIC cohort.[21] A failure to account for measurement error might lead to an erroneous association of HDP with PWV.

The main limitation of this study is the small sample size, which may have masked a significant independent association of diagnosis with arterial stiffness measures with a small effect size. Some heterogeneity among women may have resulted from timing of menstrual cycle, since there is evidence of hormonal effects on measures of stiffness.[26] In addition, we included women with both preeclampsia and gestational hypertension and had small numbers of women with more severe disease, potentially decreasing the effect size.

Like prior investigations, the absence of prepregnancy data limits our ability to draw conclusions about a causal role of HDP on blood pressure trajectory and arterial stiffness. Early pregnancy data might not adequately reflect prepregnancy characteristics because of changes in BP typically observed very early in the first trimester of pregnancy, limiting its potential utility as a proxy for prepregnancy values.[28, 29]

Conclusions

The higher average blood pressure among women one year after delivery of a pregnancy complicated by HDP was independent of obesity and of sufficient magnitude to play an important role in their future risk of CVD. Their higher AIx suggests the complicated pregnancy may play a role as a causal factor. Large prospective studies, starting prior to conception, are needed to further our understanding of the contribution of arterial stiffness and endothelial dysfunction to the evolution of hypertension and CVD disease in women after affected pregnancies.

Acknowledgments

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Table 1

3-month and 1-year Measures

Timing	Measure	Control, (n=41)	Hypertensive disorder of pregnancy, (n=33)	P value
			Mean (SD) or n (%)	
3-month visit				
	Peripheral SBP, mm Hg	108.9 (8.8)	117.3 (10.6)	<0.01
	Peripheral DBP, mm Hg	71.1 (5.5)	77.7 (8.5)	<0.01
1-year visit				
	Central SBP, mm Hg	101.3 (10.6)	110.1 (11.6)	<0.01
	Central DBP, mm Hg	73.8 (7.6)	79.5 (9.0)	<0.01
	Peripheral SBP, mm Hg	111.1 (9.7)	119.7 (11.5)	<0.01
	Peripheral DBP, mm Hg	72.9 (7.4)	78.4 (9.0)	<0.01
	Central pulse pressure, mm Hg	27.5 (5.4)	30.6 (5.5)	<0.05
	Mean arterial pressure, mm Hg	86.4 (8.9)	93.9 (10.0)	<0.01
	HTN or BP>=140/90, n (%)	1 (2.4)	7 (21.2)	<0.01
	Anti-hypertensive medication, n (%)	0 (0)	2 (6.2)	NS

Abbreviations: SD. standard deviation; SBP, systolic blood pressure; DBP, diastolic blood pressure; HTN, diagnosis of chronic hypertension reported; NS, not significant

Table 2

Comparison of pulse wave velocity, augmentation index, and inflammatory biomarkers

Measure	Control, (n=41)	Hypertensive disorder of pregnancy, (n=33)	P value
		Mean (SD)	
Vascular measures			
Carotid femoral pulse wave velocity, unadjusted, m/s (n=66)	6.3 (1.2)	7.1 (2.0)	NS
Carotid femoral pulse wave velocity, adjusted for waist size, m/s (n=66)	5.7 (1.0)	6.2 (1.5)	<0.01
Augmentation Index, unadjusted	16.6 (13.0)	22.0 (13.4)	<0.01
Augmentation Index, adjusted for HR 75	12.0 (12.5)	18.4 (13.1)	<0.05
Central augmented pressure	4.9 (4.2)	7.1 (4.6)	<0.05
Central augmented pressure, HR 75	3.3 (3.8)	5.8 (4.1)	0.01
Mean arterial pressure, mm Hg	86.4 (8.9)	93.9 (10.0)	<0.01
Heart rate	65.5 (9.6)	67.8 (12.3)	NS
Inflammatory biomarkers			
hsCRP, ng/ml	8.66 (12.1)	12.0 (15.3)	NS
IL-6, pg/ml	8.14 (2.47)	8.28 (2.36)	NS
TNF-α, pg/ml	3.93 (5.29)	8.46 (12.83)	<0.05

Abbreviations: cfPWV, carotid femoral pulse wave velocity; AIx, augmentation index; hsCRP, c-reactive protein; IL-6, interleukin 6; TNF- α, tumor necrosis factor alpha; NS, not significant.

Table 3

Multiple linear regression predicting augmentation index, n=72

	Estimate (Standard Error)	Standardized Beta
Hypertensive disorder of pregnancy	7.281 (2.995)*	0.274
Heart rate, bpm	−0.293 (0.134)*	−0.242
Height, inches	−0.983 (0.541)	−0.192
Age, years	0.871 (0.247)***	0.388
Body mass index, kg/m^2	−0.026 (0.183)	−0.017
African American race	3.835 (3.861)	0.123
Privately insured for delivery	−5.817 (3.715)	−0.194
Family history of CVD	3.831 (3.255)	0.140
Smoking	2.621 (3.190)	0.094
Intercept	73.255 (35.559)*	
Adjusted R^2	0.25	

BPM, beats per minute; CVD, cardiovascular disease

*
p<0.05

**
p<0.01

***
p<0.001