Published in final edited form as:

Atherosclerosis. 2013 July ; 229(1): 212–216. doi:10.1016/j.atherosclerosis.2013.04.012.

Hypertension in Pregnancy is a Risk Factor for Peripheral Arterial Disease Decades after Pregnancy

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Abstract

Background—An ankle-brachial index (ABI) (the ratio of ankle to brachial artery systolic blood pressure) value ≤ 0.9 identifies patients with peripheral arterial disease (PAD) and elevated cardiovascular event risk. This study examined whether women with a history of hypertension in pregnancy are more likely to have an ABI ≤ 0.9 decades after pregnancy.

Methods and Results—ABI was measured in nulliparous women (n=144), and women with a history of normotensive (n=1,272) or hypertensive (n=281) pregnancies who participated in the Genetic Epidemiology Network of Arteriopathy (GENOA) study [non-Hispanic white (39%) and black (61%) women, 60 (mean) $\pm$ 10 (SD) years of age]. Relationships between PAD and pregnancy history were examined by logistic regression. Compared to women with a history of normotensive pregnancy, women with a history of hypertensive pregnancy had greater odds of PAD (1.61 (odds ratio); 1.04–2.49 (95% confidence interval), p=0.03, adjusted for age, race, height and heart rate). Additional adjustment for ever smoking, hypertension, diabetes, dyslipidemia, a family history of hypertension or coronary heart disease, body mass index and education did not attenuate this relationship (1.63; 1.02–2.62, p=0.04). PAD risk did not differ between women with a history of normotensive pregnancy and nulliparous women (1.06; 0.52–2.14, p=0.87).

Conclusions—Hypertension in pregnancy is an independent risk factor for PAD decades after pregnancy after adjusting for race, age, height, heart rate, ever smoking, hypertension, diabetes,

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Disclosures: None.

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dyslipidemia, a family history of hypertension or coronary heart disease, body mass index and education.

Keywords

hypertension in pregnancy; peripheral vascular disease; ankle-brachial index

1. Introduction

Cardiovascular disease is the leading cause of death in women, but primary prevention is hindered by the fact that risk factors for women are not as well elucidated as for men [1]. Cardiovascular disease risk predictors specific for women are urgently needed. A history of pregnancy complications is emerging as one such potential risk factor [2,3]. Hypertension complicates 8% of pregnancies, and includes four conditions; gestational hypertension, preeclampsia, chronic hypertension and chronic hypertension with superimposed preeclampsia [4]. Gestational hypertension is *de novo* hypertension that develops after 20 weeks gestation and resolves post-partum [4]. Preeclampsia is gestational hypertension accompanied by proteinuria after 20 weeks gestation [4]. Chronic hypertension in pregnancy is hypertension that is diagnosed prior to conception or develops before 20 weeks gestation, and does not resolve post-partum [4]. One in four women with chronic hypertension will develop superimposed preeclampsia [4]. A history of any form of hypertension in pregnancy identifies women with an elevated risk of hypertension, coronary heart disease and stroke later in life [5–8].

Less is known about the relationship between hypertension in pregnancy and peripheral arterial disease (PAD). Whereas one study reported increased PAD risk decades after a pregnancy complicated by a maternal placental syndrome [9], others did not observe an increased risk of PAD [10] or its classic symptom, intermittent claudication [8], in women with previous preeclampsia. Existing studies have identified PAD solely by diagnostic codes [9,10], which is a significant limitation. PAD tends to be under diagnosed, as only 20–25% of patients with PAD by objective criteria present with intermittent claudication [11]. Pregnancy history studies which include women with atypical symptoms or asymptomatic PAD are urgently needed, as disease progression does not differ between PAD patients with and without intermittent claudication [11].

The ankle-brachial index (ABI) is used in the clinical setting to diagnose PAD and is also an indicator of cardiovascular event risk [11,12]. The ABI is calculated as the ratio of ankle systolic blood pressure to brachial artery systolic blood pressure [11,12]. An ABI ≥ 0.9 confirms suspected PAD in clinical settings, and identifies patients with symptomatic and asymptomatic PAD in epidemiology studies [11,12]. In addition, an ABI ≥ 0.9 is strongly associated with cerebral and coronary artery disease [11]. Cardiovascular mortality is 3–6 times more likely in patients with an ABI ≥ 0.9 [11].

Our objective was to examine the relationship between a history of hypertension in pregnancy and PAD, defined in accordance with current guidelines as an ABI ≥ 0.9 [11,12]. We hypothesized that women with a history of hypertension in pregnancy would be more likely to have PAD decades after pregnancy, demonstrating that hypertension in pregnancy is a risk factor for PAD. An ABI ≥ 0.9 also identifies individuals with an elevated risk of cardiovascular mortality [11]; therefore, such data would also be consistent with previous findings of elevated cardiovascular event risk in women with a history of hypertensive pregnancy [5].

2. Material and Methods

2.1. Participants

This study includes 1,697 women from 907 sibships who participated in the Family Blood Pressure Project (FBPP) Genetic Epidemiology Network of Arteriopathy (GENOA) study. The FBPP was established in 1995 to investigate genes that influence hypertension in racial and ethnic groups in the United States [13]. GENOA was one of four networks that enrolled sibships in which two or more siblings had hypertension diagnosed prior to age 60. Study design and recruitment were described previously [13]. ABI was measured at the Rochester, Minnesota and Jackson, Mississippi GENOA sites. Rochester enrolled non-Hispanic whites. Jackson enrolled non-Hispanic blacks. The present study includes women who completed both the Phase 2 study examination pregnancy history questionnaire and whose ABI was measured. All subjects provided written informed consent prior to participating, and the protocol was approved by each site's institutional review board.

2.2. Questionnaires

Questionnaires were administered by a trained interviewer. The Phase 1 (1996–2000) questionnaire examined personal and family medical history. Phase 2 (2000–2004) included an additional standardized, previously validated questionnaire concerning hypertension in pregnancy [14]. Women were asked 'Have you had at least one pregnancy lasting more than 6 months?' Women who responded 'yes' were asked how many pregnancies they had, and whether they developed hypertension in any pregnancy lasting more than 6 months.

2.3. Physical Examination

Blood pressure, height and weight were measured using a standardized protocol. Blood pressure was measured with an automated oscillometric device. Height was measured with the subject standing with her heels together, without shoes, against a vertically mounted ruler. Venipuncture was performed after an overnight fast (8 hours). Serum triglyceride, total cholesterol and HDL cholesterol concentrations were measured on a Hitachi 911 Chemistry Analyzer (Roche Diagnostics, Indianapolis, Indiana, USA).

2.4. Phase 2 Study Definitions

'Ever' smoking was defined as a lifetime history of having smoked at least 100 cigarettes. Hypertension was defined as a self-reported physician diagnosis of hypertension and prescription antihypertensive medication use, or average blood pressures 140 mmHg systolic and/or 90 mmHg diastolic at the study examination. Coronary heart disease was defined as self-reported myocardial infarction, coronary angioplasty, coronary bypass surgery, balloon dilatation or stent placement. Dyslipidemia was defined as one or more abnormal lipid measurements at the study examination (total cholesterol 200 mg/dl, triglycerides 150 mg/dl, or HDL 40 mg/dl) or use of lipid-lowering drugs. Diabetes and hypertension in pregnancy were defined by self-report.

2.5. Ankle Brachial Index

ABI was measured in accordance with current guidelines [11,12], using the same standardized protocol at both the Rochester and Jackson sites. Examiners were trained at the Mayo Clinic's non-invasive vascular laboratory. Following 5 minutes of supine rest, appropriately sized blood pressure cuffs were placed on the participant's upper arms and ankles. Each cuff was inflated to 10 mm Hg above systolic pressure, then deflated at 2 mm Hg/s. Systolic pressure was the pressure at which the pulse first reappeared on Doppler ultrasound (Medsonics, Minneapolis, MN). The ABI for the dorsalis pedis and posterior tibial artery on each side was calculated as the systolic pressure at the ankle divided by the

higher of the two brachial systolic pressures. An average ABI for each side was calculated by averaging the posterior tibial and dorsalis pedis ABI values. The ABI was then defined as the lower of the right or left values. In accordance with current guidelines [11], PAD was diagnosed as an ABI ≤ 0.9 . Poorly-compressible leg arteries were defined as an ABI > 1.4 [11]. Intermittent claudication and non-typical PAD symptoms were not assessed.

2.6. Statistical Analysis

Pairwise differences in clinical characteristics between pregnancy groups (nulliparous women, women with a history of normotensive pregnancy, and women with a history of hypertensive pregnancy) were assessed using chi-square tests for categorical variables and two-sample t-tests for continuous variables. The ABI was associated with both age and race; therefore regression modeling was used to assess differences in the ABI between pregnancy history groups after adjusting for age and race, sibling relationships, and other variables that may influence the ABI by affecting pressure wave propagation and amplification (i.e., height, heart rate) [15]. From these models, we calculated an adjusted percentage of women with PAD or the estimated mean $\pm$ standard deviation of ABI in each pregnancy group. A second set of logistic and linear models, were fit adjusting for age, race, height, heart rate, and other CVD risk factors (smoking, hypertension, diabetes, BMI, dyslipidemia, family history of hypertension, family history of coronary heart disease and education). All regression models were fit with generalized estimating equations to account for sibling relationships. Since further studies beyond the ABI are required to diagnose PAD in women with poorly-compressible arteries (ABI > 1.4 [11], $n=6$), these women were excluded from all models predicting PAD. These results remained the same in a sensitivity analysis in which the fit the models excluded women with ABI > 1.3 ($n=50$). These women have poorly-compressible arteries as defined by the American College of Cardiology and American Heart Association guidelines [12]. Analyses were performed using SAS Version 9.2 (SAS Institute Inc., Cary, NC). Statistical significance was defined as $p < 0.05$.

3. Results

3.1. Participant Characteristics

There were 144 (8%) nulliparous women, 1,272 (75%) women with normotensive pregnancies, and 281 (17%) women with previous hypertensive pregnancies. Women were 60 ± 10 (mean $\pm$ SD) years of age. Women with previous hypertensive pregnancies were more likely to have hypertension and less likely to have dyslipidemia or have ever smoked than women in the other two groups (Table 1). Compared to women with previous normotensive pregnancies, women with previous hypertensive pregnancies were younger, had higher BMIs, and were more likely to have diabetes and a family history of hypertension and coronary heart disease. Height, heart rate and the percentage of women taking statins were not significantly different between the three groups. Nulliparous women were significantly more likely to be white and have higher levels of education than women in the other two groups, and were significantly younger and had higher BMIs than women with previous normotensive pregnancies.

3.2. ABI, Race and Age

Compared to white women, black women had lower mean ABI values (0.99 ± 0.12 vs. 1.13 ± 0.13 , $p < 0.001$), and were more likely to have PAD as defined by an ABI ≤ 0.9 (11% vs. 5%, $p < 0.001$). Older age was also associated with lower ABI (Estimate: -0.04 per 10 years, $p < 0.001$), and an increased odds of PAD (2.74, 95% CI: 2.11–3.54, $p < 0.001$). Subsequent analyses were adjusted for race and age.

3.3. ABI and PAD ($ABI \leq 0.9$) According to Pregnancy History

Nine nulliparous women, 102 women with normotensive pregnancies, and 29 women with a history of hypertensive pregnancy had PAD. Since height and heart rate may influence the ABI by affecting pressure wave reflection and amplification in the extremities, we controlled for these variables in addition to age and race. After adjusting for these variables, 7.2% of nulliparous women, 7.7% of women with a history of normotensive pregnancy, and 11.3% of women with a history of hypertensive pregnancy had PAD. Compared to women with a history of normotensive pregnancy, women with a history of hypertensive pregnancy were significantly more likely to have PAD after adjusting for age, race, height and heart rate (Table 2, odds ratio (95% confidence interval) 1.61 (1.04–2.49), $p=0.03$). Adjusting for additional cardiovascular disease risk factors did not attenuate the strength of this relationship (1.63 (1.02–2.62), $p=0.04$). Women with a history of normotensive pregnancy were not significantly more likely to have PAD than nulliparous women after all adjustments (1.06 (0.52–2.14), $p=0.87$).

3.4. ABI, Cardiovascular Characteristics and Pregnancy History

In addition to examining associations of pregnancy history and PAD, we also examined differences in ABI as a continuous variable by pregnancy group. The mean ABI was 1.03 ± 0.13 in women with previous hypertensive pregnancies compared to 1.05 ± 0.11 in women with previous normotensive pregnancies after adjusting for age, race, height, and heart rate (Estimate: -0.017 , $p=0.04$, Table 1S). This difference persisted in a multivariate model adjusting for age, race, height, and heart rate and additional co-morbidities and cardiovascular risk factors (Estimate: -0.021 , $p=0.01$, Table 2S). The mean ABI did not significantly differ between nulliparous women and women with previous normotensive ($p=0.90$) or hypertensive ($p=0.33$) pregnancies, after adjusting for age, race, height and heart rate or after adjusting for additional co-morbidities and cardiovascular risk factors ($p=0.82$ and $p=0.26$, respectively). However, we had limited power to detect differences due to the small number of nulliparous women.

4. Discussion

In accordance with our hypothesis, hypertension in pregnancy is a risk factor for PAD decades after pregnancy. This relationship was not attenuated by adjusting for conventional cardiovascular risk factors and co-morbidities. The higher prevalence of an $ABI \leq 0.9$ in women with a previous hypertensive pregnancy is also consistent with results of previous studies demonstrating that these women have a greater cardiovascular event risk. This adds a new vascular health marker to the growing body of evidence [5–8] indicating that a previous hypertensive pregnancy identifies women with increased cardiovascular disease risk.

Results of previous studies examining the relationship between PAD and hypertensive disorders of pregnancy are conflicting. A study of over 1 million Canadian women reported a 4-fold increase in PAD risk three decades after a pregnancy complicated by a maternal placental syndrome (gestational hypertension, preeclampsia, placental abruption, or placental infarction) [9]. Smaller studies did not observe an increased risk of PAD [10] or its classic symptom, intermittent claudication [8] in women with previous preeclampsia or gestational hypertension. Two factors could contribute to these differing results among existing studies, and between existing studies and the current study. First, previous studies identified PAD by diagnostic codes [9,10] or by intermittent claudication, as assessed by the Rose questionnaire [8]. In contrast, in the current study we measured the ABI in all participants and defined PAD as an $ABI \leq 0.9$. This objective criterion is an important strength of the current study, as it prevents under diagnosis in the 75–80% of PAD patients who do not have intermittent claudication [11]. Second, previous studies examined women

with a history of maternal placental syndromes [9], toxemia [10], or preeclampsia and gestational hypertension [8], whereas the current study included women with all forms of hypertension in pregnancy.

The prevalence of PAD was approximately 3% among women between the ages of 50 and 69 who participated in the National Health and Nutrition Examination Survey in 1999–2000 [16]. The higher prevalence of PAD among women in the present study (whites: 5%; blacks: 11%) may in part be due to the high prevalence of diabetes, dyslipidemia, and hypertension in this cohort. The higher prevalence of PAD in blacks, compared to whites, was described in detail in a previous publication from the GENOA study [17]. Black participants were older and had a higher incidence of diabetes and hypertension than white participants, however ABI was still lower in blacks after adjusting for these confounding variables [17]. Other studies have also reported that the prevalence of PAD is higher in blacks [16].

The present study has several limitations. First, 77% of participants had hypertension, as GENOA recruitment targeted sibships with at least 2 hypertensive members. Results may not apply to populations with lower rates of hypertension. However, a family or personal history of hypertension is a risk factor for both hypertension in pregnancy [4] and PAD [11,12]. This suggests that there may have been a stronger relationship between previous hypertension in pregnancy and PAD if this cohort had included women with no personal or family history of hypertension. Second, pregnancy history and several other co-morbidities were determined by self report. However, the pregnancy history questionnaire had a sensitivity of 80% and specificity of 90% for the determination of preeclampsia [14]. Third, exertional leg symptoms were not evaluated, therefore we cannot determine what proportion of PAD cases were symptomatic vs. asymptomatic, or had intermittent claudication vs. non-traditional symptoms. This makes it difficult to compare the current study with previous studies which based diagnoses on codes or intermittent claudication. In conclusion, hypertension in pregnancy is a risk factor for PAD (ABI 0.9) decades after pregnancy. This relationship was not attenuated by adjusting for cardiovascular risk factors or co-morbidities. The increased odds of an ABI 0.9 in women with previous hypertension in pregnancy is consistent with previous studies demonstrating that these women are more likely to experience future cardiovascular events. Future studies should examine whether PAD screening of women with a history of hypertension in pregnancy could identify those at greatest risk for CVD events.

Supplementary Material

Refer to Web version on PubMed Central for supplementary material.

Acknowledgments

Grant Support: This work was supported by grants from the National Heart, Lung, and Blood Institute and NIH (U01HL054481, U01HL054471, U01HL054512, and U01HL054498). The project described was supported by Award Number K08 HD051714 (V.D. Garovic) from the Eunice Kennedy Shriver National Institute of Child Health & Human Development and by Award Number P-50 AG44170 (V.D. Garovic, V.M. Miller) from the National Institute on Aging. Tracey Weissgerber was supported by the Office of Women's Health Research (Building Interdisciplinary Careers in Women's Health award K12HD065987). The content is solely the responsibility of the authors and does not necessarily represent the official views of the NIH. The writing of the manuscript and the decision to submit it for publication were solely the authors' responsibilities.

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Highlights

- We examined women with previous normotensive or hypertensive pregnancies
- The ankle-brachial index was measured decades after pregnancy
- Peripheral arterial disease (PAD) was defined as an ankle-brachial index < 0.9
- Women with a previous hypertensive pregnancy were more likely to have PAD

Table 1

Participant Characteristics

Variable	Nulliparous (n = 144)	Normotensive Pregnancy (n = 1,272)	Hypertensive Pregnancy (n = 281)
Race			
Non-Hispanic white	82 (57%)	480 (38%)	106 (38%)
Non-Hispanic black	62 (43%)	792 (62%)	175 (62%)
Significance		*	*
Age (years)	57 ± 12	61 ± 10 *	59 ± 10 †
Body mass index (kg/m ²)	32.6 ± 8.3	31.3 ± 6.7 *	33.9 ± 7.1 †
Education			
Less than high school (< 8 years)	9 (6%)	94 (7%)	31 (11%)
Some high school (9–11 years)	8 (6%)	162 (13%)	41 (15%)
High school graduate or GED (12 years)	34 (24%)	461 (36%)	94 (33%)
Post high school (>12 years)	93 (65%)	555 (44%)	115 (41%)
Significance		*	*
Ever smoked	54 (38%)	451 (35%)	76 (27%) *, †
Diabetes	27 (19%)	254 (20%)	74 (26%) †
Hypertension	113 (78%)	943 (74%)	249 (89%) *, †
Dyslipidemia	115 (80%)	962 (76%)	193 (69%) *, †
Statins	24 (17%)	258 (20%)	59 (21%)
Family history of hypertension	115 (80%)	973 (76%)	236 (84%) †
Family history of coronary heart disease	65 (45%)	509 (40%)	132 (47%) †
Height (cm)	164 ± 7	164 ± 6	163 ± 6
Heart rate (beats/min)	68 ± 12	67 ± 10	66 ± 11

Values are mean ± standard deviation or n (%).

Significant difference from: Nulliparous,

* p<0.05; Normotensive pregnancy,

† p<0.05.

Abbreviations: BMI, body mass index; GED, general educational development.

Table 2

Odds Ratios for low ABI (< 0.9) According to Pregnancy History

Groups	Model 1 [*]		Model 2 [†]	
	OR (95% CI)	p	OR (95% CI)	p
Normotensive pregnancy vs. nulliparous	1.07 (0.52, 2.21)	0.86	1.06 (0.52, 2.14)	0.87
Hypertensive pregnancy vs. nulliparous	1.72 (0.76, 3.88)	0.19	1.73 (0.76, 3.89)	0.19
Hypertensive vs. normotensive pregnancy	1.61 (1.04, 2.49)	0.03	1.63 (1.02, 2.62)	0.04

Excludes women with an ABI > 1.4 (1 nulliparous, 4 normotensive pregnancies, 1 hypertensive pregnancy). Abbreviations: OR, odds ratio; CI, confidence interval

^{*} Adjusted for race, age, height, heart rate

[†] Adjusted for race, age, height, heart rate, education, log body mass index, ever smoking, hypertension, diabetes, dyslipidemia, family history of hypertension and coronary heart disease

All models were fit with generalized estimating equations to account for relationships among siblings.