Title

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THE ASSOCIATION OF RENAL ARTERY CALCIFICATION WITH HYPERTENSION IN COMMUNITY-LIVING INDIVIDUALS: THE MULTI-ETHNIC STUDY OF ATHEROSCLEROSIS

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Abstract

Hypertension (HTN) is a modifiable risk factor for cardiovascular disease (CVD). Renal artery calcium (RAC) may signal the presence of flow-limiting atherosclerotic disease that may contribute to changes in the kidney's regulation of blood pressure. We hypothesized that RAC is independently associated with HTN. We examined a multi-ethnic cohort of 1,285 participants who underwent abdominal computed tomography (CT) scans in five US communities. After adjustment for age, gender, race/ethnicity, CVD risk factors, abdominal aortic calcium score and kidney function, the presence of RAC was associated with a 50% higher odds of HTN (OR: 1.54; 95% CI 1.11–2.13). Similarly, the presence of RAC was associated with a 8.5-mmHg higher systolic blood pressure (SBP), a 2.1 mmHg higher diastolic blood pressure (DBP), and a 7.4-mmHg higher pulse pressure (PP). In conclusion, independent of CVD risk factors, abdominal aortic calcium, and kidney function, the presence of RAC is associated with HTN prevalence.

Keywords

Hypertension; Kidney disease; Renal artery calcification; Multi-ethnic; atherosclerosis

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INTRODUCTION

Cardiovascular disease (CVD) remains the leading cause of mortality among Americans, accounting for one-third of all deaths in the U.S.[1] Of the modifiable risk factors for CVD, hypertension (HTN) has been estimated to account for 18% of the population attributable risk for first myocardial infarction.[2,3] Furthermore, chronic kidney disease (CKD) is a strong risk factor for both HTN and CVD,[4] and HTN is a leading cause of CKD in the U.S, second only to diabetes as a factor in the progression to end-stage renal disease (ESRD).[5–7]

Renal mechanisms appear to play a primary role in the pathophysiology of blood pressure elevation.[8] Calcified atherosclerotic plaques can be detected and quantified using computed tomography (CT).[9–12] In the coronary arteries, the degree of arterial calcification is highly correlated with atherosclerotic plaque burden.[13–15] While significant atherosclerosis of the renal artery can lead to flow-limiting stenosis and the development of renovascular HTN, subclinical atherosclerosis of the renal arteries may also be associated with changes in blood pressure regulation. Even if not flow limiting, it is possible that calcification within the renal arteries marks the burden of small vessel vascular disease within the kidney, which in turn may lead to salt and water retention and resultant HTN. However, whether or not RAC is associated with HTN is uncertain.

To our knowledge, only two prior studies have examined the relationship of RAC with HTN. Both showed that renal artery calcium (RAC) was associated with a higher prevalence of HTN.[16,17] However, these studies evaluated predominantly Caucasian populations; whether these associations extend to different race/ethnicities is uncertain. Furthermore, it remains uncertain if the association of RAC with HTN is independent of kidney function in a multiethnic cohort. Given this, the present study aimed to examine the cross-sectional association of RAC with HTN in a community-living, multi-ethnic cohort, and to determine if such associations persist when accounting for traditional CVD risk factors, calcification of non-renal vasculature, and kidney function. As a secondary objective, this study aims to examine the association of RAC with different components of blood pressure, including systolic blood pressure (SBP), diastolic blood pressure (DBP) and pulse pressure (PP).

METHODS

Participants

Between August 2000 and July 2002, 6,814 men and women ages 45–84 years were recruited from six US communities to participate in the Multi-Ethnic Study of Atherosclerosis (MESA), a population-based longitudinal study aimed at examining subclinical CVD and the progression to clinical disease. Men and women were eligible to participate if they self-identified as African-American, Chinese-American, Caucasian, or Hispanic and had no known clinical CVD at baseline. Complete methods have been previously published.[18]

Participants for this analysis consisted of 1,959 MESA subjects who were randomly selected from five of the MESA field centers (Chicago, Illinois; Forsyth County, North Carolina; Los Angeles County, California; New York, New York; and St. Paul, Minnesota) to participate

in the MESA Abdominal Aortic Calcium Study during which abdominal computed tomography (CT) scans were conducted at clinic exams 2 or 3 (July 2002 – July 2005).[19] This study was approved by the institutional review boards at all field centers, and all participants gave informed consent prior to participation.

Procedures

Data for this study were collected during clinic visits 2 or 3 (corresponding to the visit when the abdominal CT was conducted). Demographic and behavioral information, including age and smoking, were collected via a self-administered questionnaire, while trained interviewers collected information on medication usage and medical history.

Trained study staff took blood pressure and anthropometric measurements. Three blood pressure measurements were taken after the participant was seated quietly for 5 minutes using a Dinamap® automated blood pressure device (Dinamap Monitor Pro 100®, Critikon, Inc., Tampa, FL); the average of the 2nd and 3rd measurements was used for analysis. PP was calculated as SBP –DBP. HTN was defined as SBP ≥ 140 mmHG or DBP ≥ 90 mmHG, or current use of antihypertensive medication. Pack-years of smoking was calculated as (the number of cigarettes smoked per day/20) multiplied by the number of years smoked. Anthropometric measurements were taken with participants wearing light clothes and no shoes. Body mass index (BMI) was calculated as weight (kg)/height (m^2).

Blood sample collection and processing was performed according to a standardized protocol. Venipuncture was conducted by trained phlebotomists among participants in a 12-hour fasting state for measurement of serum creatinine and lipid profiles. Dyslipidemia was defined as total cholesterol-to-high-density lipoprotein (HDL) ratio> 5 or use of a lipid-lowering medication.[20,21] Diabetes status was determined based on 2003 American Diabetes Association criteria: fasting blood glucose of ≥ 7-mmol/L (126-mg/dl) or current insulin or oral hypoglycemic medication usage.[22] Estimated glomerular filtration rate (eGFR) was calculated from serum creatinine and demographic variables using the Chronic Kidney Disease Epidemiology Collaboration equation.[23] Urine was collected for albumin and creatinine, which were used to calculate the urinary albumin to creatinine ratio (UACR).

Given its anatomic proximity to the renal arteries and its association with CVD mortality and calcification in other vascular beds, abdominal aorta calcification was included as a marker of system vascular atherosclerotic calcification.[24,25] The presence and extent of calcification in the abdominal aorta and the left and right renal arteries were measured using abdominal CT scans conducted using electron-beam CT (EBCT) scanners (Imatron C-150; Imatron, Inc., San Francisco, California) or prospective electrocardiogram-triggered scanners (Siemens S4+ Volume Zoom; Siemens, Erlanger, Germany; and General Electric Hi Speed LX, GE Medical Systems, Milwaukee, Wisconsin). The distal 15 cm of the abdominal aorta terminating at the aortic bifurcation was scanned. The L5-S1 intervertebral disc space was identified on scout films to approximate the level of the aortic bifurcation.

CT images were centrally reviewed by trained study technologists at the MESA CT Reading Center (Los Angeles, California). Calcified foci were defined as those regions with a density of >130 Hounsfield units (HU) and an area of ≥3 contiguous pixels (1.0-mm^2). Total RAC

scores were calculated by summing left and right renal ostia Agatston scores and left and right renal artery Agatston scores. Calcium in the abdominal aorta was scored using an 8-cm segment of the distal abdominal aorta ending at the aortic bifurcation. Calcium in the descending thoracic aorta was scored using the segment of thoracic aorta extending from the lower edge of the pulmonary artery to the cardiac apex. Calcium in the ascending aorta was scored using the segment of the thoracic aorta extending from the aortic annulus to the lower edge of the pulmonary artery. The aortic arch was not visualized. All calcium scores were quantified using Agatston methodology.[9]

Statistical Analysis

Descriptive statistics were computed for all variables, including mean and standard deviation or median and interquartile range (IQR) for continuous measures; and frequency and percentage for categorical measures. Differences between those with any RAC (RAC >0) and those with no RAC (RAC=0) were examined using chi-square tests for categorical variables, or parametric t-tests or non-parametric Wilcoxon rank-sum tests for continuous variables.

RAC was examined both as a dichotomous variable (RAC>0 vs RAC=0) and, among those with RAC > 0, as a continuous variable. Logistic regression was used to examine the association of RAC with HTN. A sequence of models was evaluated. An initial model adjusted for age, gender and race/ethnicity; Model 2 adjusted for model 1 covariates plus traditional CVD risk factors (BMI, pack years of smoking, diabetes, dyslipidemia and family history of CVD) and abdominal aorta calcium (AAC) score; Model 3 adjusted for model 2 covariates plus measures of kidney function (eGFR and UACR). The descending thoracic aorta calcium (DTAC) score was substituted in place of the AAC score in Models 2 and 3 to assess for possible associations of thoracic aortic calcification on blood pressure regulation.

To further examine associations with blood pressure, additional analyses were conducted between RAC and three continuous blood pressure (BP) outcome measures: SBP, DBP and PP. Since participants taking anti-hypertensive medication may have had some of the highest “underlying” blood pressures, but may have quite low observed BP, censored normal regression (CNR) was used to examine the association of RAC with the continuous blood pressure measures.[26] CNR assumes right censoring of observed blood pressure measures among individuals taking antihypertensive medication and is a technique for adjusting for the treatment effects of medication usage.

Interactions between RAC and gender, as well as RAC and race/ethnicity were investigated with $p<0.10$ considered to be significant. A post-hoc power estimation for each race/ethnicity was performed based on a 2-sided chi-square test for two independent proportions and a 0.05 type I error rate. In all subgroups, the sample sizes are sufficient to detect a difference with over 90% power.

Data were analyzed using SAS 9.3 (Cary, NC); analyses were two-tailed with $p<0.05$ considered statistically significant for all primary analyses except for interaction terms.

RESULTS

A total of 1,959 participants underwent abdominal CT scans at visits 2 or 3, of which 1,285 had scans that continued cephalad enough to contain the renal arteries and were included in the analysis. Overall, the average age was 66.3 years (standard deviation [SD] 9.6); 54.8% were female, 36.8% Caucasian, 14.1% Chinese-American, 21.4% African-American and 27.7% Hispanic American. The prevalence of HTN was 55.4%, and 46.7% were taking one or more medications for HTN.

Approximately 33% of participants had RAC > 0 (women: 33.6%; men: 32.2%). Caucasians had the highest prevalence of RAC (38.0%), followed by Hispanics (31.1%), Chinese-Americans (30.0%), and African-Americans (28.7%). Among those with any RAC (RAC > 0), the median Agatston score was 63.9 (interquartile range [IQR] 17.1 – 179.9) (Table 1).

Compared to those with no RAC, those with RAC>0 were significantly older, had greater pack-years of smoking, were more likely to have dyslipidemia, diabetes and a family history of CVD, and a larger proportion were Caucasian. Moreover, those with RAC > 0 had significantly higher SBP, lower DBP, and higher PP in unadjusted analysis, and a larger percentage were classified as hypertensive and taking antihypertensive medications. RAC > 0 was also associated with a lower eGFR, higher UACR and a greater AAC (Table 1). In contrast, gender and BMI were similar irrespective of RAC presence.

Figure 1 displays the results of logistic regression analyses examining the association of RAC and HTN. After adjustment for age, gender, race/ethnicity, CVD risk factors, and AAC (model 2), those with RAC>0 had 60% higher odds of HTN compared to those with no RAC (OR 1.61; 95% CI 1.12 – 2.21). Additional adjustment for eGFR and UACR (model 3) only modestly attenuated the odds of HTN (1.54; 1.11 – 2.13). When adjusting for DTAC instead of AAC in Models 2 and 3, the presence of RAC was significantly associated with HTN (OR 1.61 [95% CI 1.19–2.18] and OR 1.56 [95% CI 1.14 to 2.12], respectively).

When analyzed as a continuous variable (1-SD increment), increasing severity of RAC was significantly associated with HTN in model 1 (1.22; 1.07 – 1.40), but was attenuated and rendered no longer statistically significant in models 2 (1.12; 0.99 – 1.28) and 3 (1.11; 0.97 – 1.27). This attenuation was primarily driven by the abdominal aorta calcium score.

The association of RAC with HTN was similar by race/ethnicity (p interaction=0.61), however point estimates were highest in African Americans and lowest in Caucasians and Hispanics (Table 2). The association of RAC and HTN was similar by sex (p interaction=0.17, Table 3).

Table 4 shows the results of CNR analyses examining the association of RAC and BP taking into account the use of HTN medication. After adjusting for age, gender, race, and CVD risk factors, and AAC (model 2), the presence of RAC was associated with 9.5 mmHg higher SBP, 2.6 mmHg higher DPB, and 8.1 mmHg higher PP. These associations remained statistically significant after further adjustment of eGFR and UACR (model 3). When analyzed as a continuous variable, increasing severity of RAC was significantly associated

with higher SBP (β 1.5; 95% CI 0.0 – 3.1) and PP (β 1.2; 0.00 – 2.4) in model 2 but not in model 3.

DISCUSSION

In this relatively large multi-ethnic cohort of middle to older aged men and women in five US communities, we demonstrate that RAC is independently associated with HTN after adjustments for CVD risk factors, AAC, and kidney function. Results were similar in men and women and across different races/ethnicities. Associations of the severity of RAC with blood pressure had similar point estimates, but was not statistical significant in fully adjusted models.

In this study we report an association between RAC and HTN while accounting for eGFR and UACR, suggesting that these markers of kidney function do not significantly confound the association of RAC with HTN. Furthermore, RAC is associated with HTN when adjusted for AAC and DTAC, suggesting that beyond its association with systemic atherosclerosis,[17] RAC may signal local changes in the renal vasculature that may be linked to HTN.

The prevalence of RAC found in the current study is similar to that found in the Framingham Heart Study, which estimated a 28.2% prevalence of RAC among a community-based population of 2,699 predominantly Caucasian participants of similar age to the MESA cohort.[16] Another study among a clinical-based population of 1,435 predominantly Caucasian participants in San Diego who were free of prevalent CVD estimated a 17.1% prevalence of RAC.[17] The twofold increase in prevalence seen in our study compared to the San Diego cohort could be due to our population being older, having higher average BMI, higher prevalence of diabetes and HTN, and not being self-referred. Despite differences in prevalence estimates and ethnic distributions, all three populations reported higher odds of HTN among those with RAC.

Our study adds to the literature in several important ways. First, by adjusting for eGFR and UACR, we have demonstrated that RAC is associated with HTN independent of changes in these markers of kidney function. Second, in this multi-ethnic cohort, RAC was more prevalent in whites, but the association of RAC with HTN appeared smallest in this racial group. Third, we had the opportunity to adjust for calcification in the abdominal aorta as a marker of the burden and severity of systemic calcific atherosclerosis, and found that RAC remained associated with HTN nonetheless. This finding suggests that the relationship of RAC with HTN may be specific to alterations within the renal artery or renal parenchyma.

The presence and extent of RAC identified via non-invasive CT scan may be a marker of subclinical atherosclerosis within the renal vasculature. As a reflection of arteriosclerosis of the renal arteries, RAC may be linked to local renal vasoconstriction, with the subsequent up-regulation of the renin-angiotensin system and an increase in blood pressure.[27,28] Alternatively, RAC may mark the burden of atherosclerosis within the kidney.

Despite several strengths of our study, it also has important limitations. The cross-sectional nature of this analysis precludes the possibility of determining temporal relations between

the RAC and the development of HTN. Although, there are valid biological mechanisms that could explain how RAC may lead to HTN, animal models and longitudinal data are required to examine these hypotheses more fully. The effect of arterial calcification in other vascular beds was not evaluated in this study. A significant proportion of patients without visualization of the renal arteries on abdominal CT were excluded, potentially biasing the results of this analysis. In examining the association between RAC and continuous BP measures, there is the potential for treatment bias due to lower observed BP measures among those taking antihypertensive medications. We used CNR to adjust for the treatment effect of medication use, which has been reported to be a reasonable approach to adjust for antihypertensive medication use.[26] Nevertheless, the effect of medication use would have been a reduction in the effect sizes observed towards the null making our estimates conservative. The types of anti-hypertensive, diabetic, and dyslipidemic medications were not considered in this study. Finally, MESA recruited persons without clinically apparent CVD, and age 45–84. Whether results generalize to other settings is presently uncertain.

This population-based study confirms previous reports of an association between RAC and HTN independent of CVD risk factors. Our study advances the literature by demonstrating that this association persists even after the adjustment of eGFR and UACR, is not meaningfully attenuated when accounting for atherosclerosis in the abdominal aorta, and demonstrates that the association may differ by racial group. These data suggest that local effects of RAC in the renal vasculature may contribute to development of HTN and supports future studies to evaluate mechanisms. Such mechanisms may ultimately provide new opportunities for prevention or treatment of HTN in community-living individuals.

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- Hypertension (HTN) is a common risk factor for cardiovascular disease
- Renal artery calcification (RAC) is associated with HTN in a multi-ethnic cohort
- RAC may signal atherosclerosis that contributes to the kidney's regulation of HTN

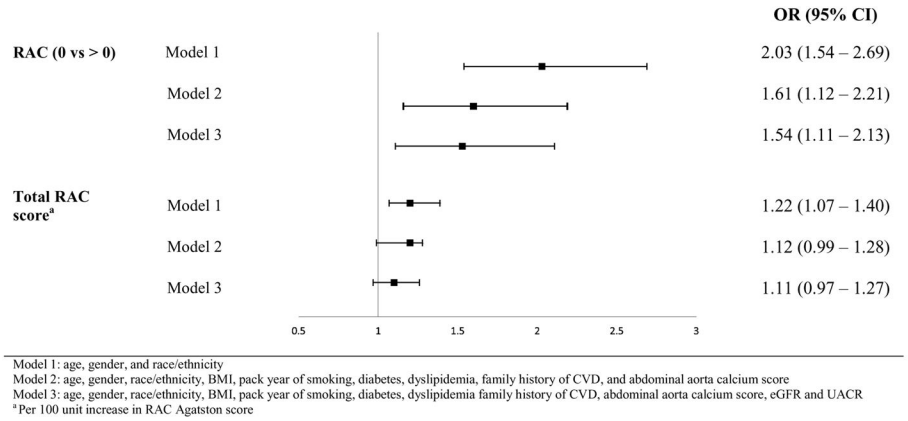

Figure 1.

Adjusted Odds Ratios (OR) and 95% Confidence Intervals (CI) Examining the Association of RAC with HTN

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Table 1

Bivariate analysis of selected characteristics by RAC status among MESA participants

Characteristic	Total (n=1285)		RAC > 0 (n=423)		RAC = 0 (n=862)		p-value[a]
Total RAC[d]	0	(0–9.4)	63.9	(17.1–179.9)	0	NA	<0.01
Age (years)[b]	66.3	(s9.6)	71.9	(s7.6)	63.5	(s9.2)	<0.01
Gender[c]							0.61
Female	704	(54.8%)	236	(55.8%)	468	(54.3%)	
Male	581	(45.7%)	187	(44.2%)	394	(45.2%)	
Race/ethnicity[c]							0.03
Caucasian	473	(36.8%)	180	(42.5%)	293	(40.0%)	
Chinese-American	181	(14.1%)	54	(12.8%)	127	(14.7%)	
African-American	275	(21.4%)	79	(18.7%)	196	(22.7%)	
Hispanic	356	(27.7%)	110	(26.0%)	246	(28.5%)	
BMI (kg/m^2)[b]	28.0	(s5.3)	28.0	(s4.9)	28.0	(s5.4)	0.74
Pack years of smoking[d]	0	(0–15.5)	1.5	(0–24.5)	0	(0–12.6)	<0.01
Diabetes[c]	188	(14.7%)	79	(18.7%)	109	(12.7%)	<0.01
SBP (mmHg)[b]	125.8	(s21.6)	132.7	(s22.2)	122.5	(s20.5)	<0.01
DBP (mmHg)[b]	70.0	(s10.0)	69.1	(s9.9)	70.4	(s10.0)	0.03
Pulse pressure (mmHg)[b]	55.9	(s17.9)	63.6	(s18.8)	52.1	(s16.1)	<0.01
Hypertension[c]	694	(55.4%)	303	(72.7%)	391	(46.8%)	<0.01
Hypertension medication[c]	581	(46.7%)	265	(64.2%)	316	(38.0%)	<0.01
Dyslipidemia[c]	488	(38.0%)	194	(45.9%)	294	(34.1%)	<0.01
Family history of CVD[c]	332	(25.8%)	126	(29.8%)	206	(23.9%)	0.02
Abdominal aorta calcium score[d]	321.7	(0–1793.5)	2128.1	(658.9–4232.0)	76.7	(0–558.0)	<0.01
UACR[d]	6.4	(3.9–12.7)	7.9	(4.5–16.1)	5.7	(3.7–11.3)	<0.01
eGFR (ml/min/1.73 m^2)[b]	77.8	(s17.6)	71.5	(s17.6)	80.9	(s16.8)	<0.01

[a] p-values are based on chi-square tests, parametric t-tests or non-parametric Wilcoxon rank sum tests, and demonstrate overall significance of differences between RAC status by each characteristic;

[b] mean (SD);

[c] n (%);

[d] median (IQR)

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Table 2

Logistic regression analyses examining the association of renal artery calcification and hypertension stratified by ethnicity

	All participants (n=1285)			Caucasian (n=473)			Asian (n=181)			Black (n=275)			Hispanic (n=356)		
	OR	**95% CI**	**p-value**	**OR**	**95% CI**	**p-value**	**OR**	**95% CI**	**p-value**	**OR**	**95% CI**	**p-value**	**OR**	**95% CI**	**p-value**
HTN															
RAC (0 vs > 0)															
Model 1	2.01	1.52 – 2.64	<0.01	2.11	1.38 – 3.23	<0.01	2.09	0.99 – 4.42	0.05	2.87	1.30 – 6.34	<0.01	1.78	1.03 – 3.08	0.04
Model 2	1.62	1.18 – 2.22	<0.01	1.46	0.90 – 2.37	0.12	1.92	0.82 – 4.49	0.13	2.24	0.90– 5.62	0.08	1.64	0.88 – 3.05	0.12
Model 3	1.56	1.13 – 2.15	<0.01	1.40	0.86 – 2.28	0.18	1.93	0.78 – 4.72	0.15	2.30	0.88 – 6.03	0.09	1.50	0.78 – 2.88	0.22
Total RAC[a]															
Model 1	1.23	1.07 – 1.40	<0.01	1.22	1.00 – 1.50	0.05	1.94	1.01 – 3.72	0.04	3.00	0.93 – 9.70	0.07	1.07	0.91 – 1.27	0.40
Model 2	1.13	0.99 – 1.28	0.07	1.09	0.89 – 1.32	0.42	1.54	0.76 – 3.11	0.23	2.19	0.61– 7.94	0.23	1.10	0.93 – 1.31	0.25
Model 3	1.12	0.98 – 1.28	0.10	1.08	0.89 – 1.31	0.45	1.53	0.68 – 3.42	0.30	1.89	0.54 – 6.67	0.32	1.09	0.91 – 1.30	0.37

Model 1: age, gender, and race/ethnicity

Model 2: age, gender, race/ethnicity, BMI, pack year of smoking, diabetes, dyslipidemia, family history of CVD, and abdominal aorta calcium score

Model 3: age, gender, race/ethnicity, BMI, pack year of smoking, diabetes, dyslipidemia, family history of CVD, abdominal aorta calcium score, eGFR and urinary albumin to creatinine ratio

[a] Per 100 unit increase in RAC Agatston score

Table 3

Logistic regression analyses examining the association of renal artery calcification and hypertension stratified by gender

	Women (n=704)			Men (n=581)		
	OR	**95% CI**	**p-value**	**OR**	**95% CI**	**p-value**
RAC (0 vs > 0)						
Model 1	2.18	1.47 – 3.22	<0.01	1.86	1.25 – 2.77	<0.01
Model 2	1.57	1.03 – 2.50	0.04	1.57	1.00 – 2.48	0.05
Model 3	1.54	0.98 – 2.44	0.06	1.44	0.90 – 2.29	0.13
Total RAC[a]						
Model 1	1.20	0.98 – 1.47	0.08	1.23	1.03 – 1.47	0.02
Model 2	1.06	0.86 – 1.32	0.58	1.15	0.98 – 1.15	0.09
Model 3	1.03	0.82 – 1.30	0.77	1.15	0.97 – 1.15	0.11

Model 1: age, gender, and race/ethnicity

Model 2: age, gender, race/ethnicity, BMI, pack year of smoking, diabetes, dyslipidemia, family history of CVD, and abdominal aorta calcium score

Model 3: age, gender, race/ethnicity, BMI, pack year of smoking, diabetes, dyslipidemia, family history of CVD, abdominal aorta calcium score, eGFR and urinary albumin to creatinine ratio

[a]Per 100 unit increase in RAC Agatston score

Table 4

Censored normal regression analyses examining the association of RAC and blood pressure taking into account HTN medication use

	RAC (0 vs > 0)			Total RAC		
	β	95% CI	p-value	β [a]	95% CI	p-value
SBP						
Model 1	13.0	9.0 – 17.0	<0.01	2.6	1.1 – 4.2	<0.01
Model 2	9.5	5.3 – 13.7	<0.01	1.5	0.0 – 3.1	0.05
Model 3	8.5	4.4 – 12.6	<0.01	1.4	−0.1 – 3.0	0.08
DBP						
Model 1	3.5	1.7 – 5.3	<0.01	0.7	0.1 – 1.4	0.03
Model 2	2.6	0.6 – 4.5	<0.01	0.6	−0.1 – 1.3	0.08
Model 3	2.1	0.2 – 4.0	0.03	0.6	−0.2 – 1.3	0.10
PP						
Model 1	11.2	8.7 – 14.2	<0.01	2.3	1.1 – 3.4	<0.01
Model 2	8.1	5.0 – 11.2	<0.01	1.2	0.0 – 2.4	0.04
Model 3	7.4	4.4 – 10.5	<0.01	1.0	−0.1 – 2.2	0.08

Model 1: age, gender, and race/ethnicity

Model 2: age, gender, race/ethnicity, BMI, pack year of smoking, diabetes, dyslipidemia, family history of CVD, and abdominal aorta calcium score

Model 3: age, gender, race/ethnicity, BMI, pack year of smoking, diabetes, dyslipidemia, family history of CVD, abdominal aorta calcium score, eGFR and urinary albumin to creatinine ratio

[a] Per 100 unit increase in RAC Agatston score