Abdominal Aortic Calcification Among Individuals With and Without Diabetes: The Jackson Heart Study

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Diabetes increases mortality by two- to fourfold from atherosclerosis-related causes, including coronary artery disease, stroke, and peripheral artery disease (1). There is a paucity of population-based data on subclinical atherosclerotic disease in arterial beds other than the coronary arteries in persons with diabetes (2).

In the Jackson Heart Study (JHS), we examined the association of diabetes with abdominal aortic calcification (AAC) among African Americans. Among included individuals ($n = 1{,}664$), the mean age was 57 ($\pm$ 11) years, 69% were female, and 18.3% had diabetes (based on fasting blood glucose [FBG], HbA_{1c}, use of glucose-lowering medications, or physician diagnosis). The median AAC and coronary artery calcification (CAC) scores were 904.15 (interquartile range 0–1093.10) and 0 (0–96.19), respectively. The prevalence of any AAC or CAC was 69% and 49%, respectively. Individuals with diabetes were older, had higher BMI, had higher systolic blood pressure and prevalence of hypertension, had lower HDL levels, were less affluent or physically active, had poorer nutritional intake, and had higher levels of hs-CRP.

After multivariable adjustment (Table 1), diabetes was associated with the presence of AAC (prevalence ratio [PR]: 1.13, 95% CI: 1.06–1.20) or CAC (PR: 1.24, 95% CI: 1.13–1.37). Prediabetes was not associated with higher prevalence of AAC (PR: 0.99, 95% CI: 0.93–1.07) or CAC (PR: 1.10, 95% CI: 0.99–1.23). Defining diabetes or prediabetes with only one biochemical criteria (FBG or HbA_{1c}) did not change the significance or direction of these associations (Table 1). One SD change in FBG (27.54 mg/dL) was associated with higher AAC (β coefficient: 0.29) and CAC (β coefficient: 0.37) (all $P \leq 0.01$). One unit increase in SD of HbA_{1c} (1.7%) was associated with higher AAC (β coefficient: 0.33) and CAC (β coefficient: 0.51) (all $P \leq 0.01$). In models including AAC and CAC simultaneously, CAC was more strongly associated with diabetes than AAC ($P < 0.05$). Diabetes status or glycemic traits (FBG or HbA_{1c}) did not interact with sex to influence AAC or CAC.

A limited number of previous studies have assessed the extent and severity of AAC among those with diabetes compared with those without diabetes. These studies have seldom included an examination of AAC versus CAC and were also limited by a small sample size, a low diabetes prevalence, and a lack of ethnic diversity (3–5). Our study included a well-characterized sample of African Americans with a high prevalence of diabetes, standardized assessments of vascular calcification, and exploration of several glycemic phenotypes. The limitations of our study include the cross-sectional design limiting inferences on causality. Some of our participants had overt cardiovascular disease (CVD) with potentially higher degrees of AAC and CAC, thus driving the observed diabetes–vascular calcification relationship, which may be lower in people without CVD. Lastly, we cannot exclude residual confounding due to misclassification and imperfect ascertainment of risk factors.

In summary, diabetes was associated with the presence and extent of arterial calcification, more so with CAC than AAC, among African Americans. Our findings point to the potential utility of assessing multiple vascular beds in ascertaining diabetes-related calcification, which may have a prognostic value in refining CVD risk assessment. Additional prospective studies are needed to examine hyperglycemia and progression of calcification in various arterial beds, as well as related clinical outcomes.

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Table 1—Associations of glycemic traits/phenotypes with AAC or CAC (n = 1,664)

Glycemic traits/phenotypes	AAC, PR (95% CI)*	CAC, PR (95%CI)*
Categorical outcomes		
Diabetes status (ADA 2010 definition)		
No	1 (reference)	1 (reference)
Yes	1.13 (1.06–1.20)	1.24 (1.13–1.37)
Impaired glucose regulation (ADA 2010 definition)		
Normal glucose status	1 (reference)	1 (reference)
Prediabetes	0.99 (0.93–1.07)	1.10 (0.99–1.23)
Diabetes	1.13 (1.05–1.22)	1.32 (1.17–1.49)
FBG categories		
<100 mg/dL (reference)	1 (reference)	1 (reference)
100 to <126 mg/dL	0.99 (0.93–1.07)	1.10 (1.00–1.23)
≥126 mg/dL	1.12 (1.05–1.22)	1.40 (1.17–1.50)
HbA_{1c} categories		
<5.7% (reference)	1 (reference)	1 (reference)
5.7–6.4%	0.98 (0.92–1.05)	1.09 (0.98–1.21)
≥6.5%	1.15 (1.07–1.24)	1.33 (1.18–1.50)
1 SD of FBG (27.54 mg/dL)	1.04 (1.02–1.06)	1.05 (1.01–1.09)
1 SD of HbA_{1c} (1.7%)	1.04 (1.02–1.07)	1.09 (1.04–1.13)

	ln(AAC+1)		ln(CAC+1)	
Linear outcomes	β (SE)*	*P* value	β (SE)*	*P* value
Diabetes (yes vs. no)	0.88 (0.22)	<0.001	1.22 (0.26)	<0.001
FBG	0.01 (0.003)	0.001	0.01 (0.004)	0.002
HbA_{1c}	0.32 (0.081)	<0.001	0.48 (0.094)	<0.001
1 SD of FBG (27.54 mg/dL)	0.29 (0.08)	0.001	0.37 (0.10)	0.001
1 SD of HbA_{1c} (1.7%)	0.33 (0.09)	<0.001	0.51 (0.10)	<0.001

*Adjusted for age, sex, income, current smoking, alcohol use, dietary intake, physical activity, BMI, estimated glomerular filtration rate, hypertension status, HDL cholesterol, LDL cholesterol, use of statins, and C-reactive protein. The American Diabetes Association (ADA) 2010 definition of glucose categories includes fasting blood glucose (≥126 mg/dL [≥7.0 mmol/L]) and/or HbA_{1c} (≥6.5% [48 mmol/mol]).

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