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The natural course of microalbuminuria among African American with type 2 diabetes: A 3 year study

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Abstract

Background—The natural course of microalbuminuria in African Americans (AA) with type 2 diabetes is not well established.

Method—Longitudinal analysis of 186 AA with type 2 diabetes enrolled in Project Sugar, a randomized controlled trial of primary care-based interventions to improve diabetes control.

Results—Mean age was 59.4 and 85% were female. Mean estimated glomerular filtration rate (eGFR) and urinary albumin-to-creatinine ratio (ACR) were 75.90 ml/min/1.73 m² and 1.62 respectively. Thirty nine patients had macroalbuminuria and significantly higher systolic blood pressure compared to those with microalbuminuria ($p=0.01$). Sixty patients had microalbuminuria, 19 progressed to macroalbuminuria and none regressed. Progression was significantly associated with systolic blood pressure ≥ 115 and requirement for blood pressure medication in the univariate model. In the multivariate model, the degree of ACR (odds ratio [OR] = 35.51, 95% CI 2.21, 571.65) and need for blood pressure medication (OR= 8.96, 95% CI 1.35, 59.70) were independently associated with progression. No association observed with the use of specific antihypertensive agent.

Conclusion—This study suggests that AA with type 2 diabetes and microalbuminuria experience irreversible disease that not infrequently progresses to overt proteinuria. The degree of microalbuminuria and blood pressure are key determinants in this process and should be primary targets in treating this population regardless of the antihypertensive class used.

Keywords

Microalbuminuria; Type 2 Diabetes; African Americans; Nephropathy

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Clinical Significance

- The natural history of microalbuminuria in African Americans with type 2 diabetes is not well established.
- Microalbuminuria in this high risk group is not infrequently irreversible compared to Caucasians with either type 1 or type 2 diabetes and microalbuminuria.
- Aggressive blood pressure control should be the primary goal in treating this population.

Introduction

In the general population, African Americans are at higher risk for kidney disease than other race groups.^{1, 2} Further, diabetic nephropathy remains the leading cause of end-stage renal disease in United States accounting for 49% of new cases.³ Compared to subjects without diabetes, the risk of end stage renal disease has been estimated to increase 12-fold in those with diabetes.⁴ The majority of these patients have type 2 DM because of aging and increased survival of this population.

It is estimated that 30–40% of persons with type 1 diabetes develop diabetic nephropathy though the incidence of end stage renal disease due to type 1 diabetes has been declining over the past 4 decades.⁵ On the other hand, renal involvement and progression to end stage renal disease vary in patients with type 2 diabetes depending on ethnic group.^{6–8} Microalbuminuria, defined as urinary albumin excretion between 30–300mg/24 hours is an important independent risk factor for progressive renal disease in both patients with type 1 and type 2 diabetes.^{9–11} Furthermore, microalbuminuria, particularly in type 2 diabetes, is recognized as a strong predictor of macrovascular complications and overall mortality due to the increase in both cardiovascular and all cause mortality.^{12–21} These associations are independent of other established risk factors in diabetes.¹¹ With the high incidence of microalbuminuria (approximately 40 %) in patients with type 2 diabetes²² and the ample of data to suggest its significant association with cardiovascular and renal events, studies examining its natural course, preventive, and intervention measures are important.

Though the data on the course of microalbuminuria in type 1 diabetes and certain racial groups with type 2 diabetes are plentiful, only a few reports have explored this issue in African Americans with type 2 diabetes.^{23, 24} For example, Perkins et al. demonstrated regression of microalbuminuria in a significant number of Caucasians patients with type 1 diabetes over the course of 6 years of follow-up.²⁵ In a cohort of 386 Caucasians with microalbuminuria and type 1 diabetes, microalbuminuria of short duration, salutary levels of glycosylated hemoglobin (HbA1C), low systolic blood pressure, and low levels of both cholesterol and triglycerides were identified as being independently associated with regression of microalbuminuria.²⁵ By identifying factors associated with regression of microalbuminuria, the study provided clues to preventive and treatment measures for patients with type 1 diabetes. Whether this approach is also germane to patients with type 2 diabetes, particularly among African Americans, is not known. This study aimed to determine the natural course of microalbuminuria among African Americans with type 2 diabetes and to identify demographic and modifiable health related factors associated with either regression of microalbuminuria to normoalbuminuria or its progression to macroalbuminuria. We also examined the relationship between baseline clinical measures and change in renal function over 3 years.

Patients and Methods

Study Participants

The study population consisted of 186 African-American adults with type 2 diabetes living in East Baltimore, a predominately African American, inner-city community who were enrolled in Project Sugar 1, an NIH-funded, randomized controlled trial of primary care-based interventions. Participants were included in the study on a rolling basis between April 1995 and February 1997 and followed until 2000. In summary, eligibility for the trial was determined by medical chart review and two screening visits. Criteria for eligibility included, age (35–75), African American ancestry, presence of type 2 diabetes (as indicated by physician diagnosis), and residence in one of seven East Baltimore zip codes. In addition, all participants attended either the Johns Hopkins outpatient center or the East Baltimore Medical Center for primary care within the previous year. Participants were excluded if they had comorbid conditions limiting probable life span to <4 years (e.g., cancer, acquired immune deficiency syndrome) or indication of end-stage complications of diabetes (kidney dialysis or transplant, blindness, or lower extremity amputation). The design and results of Project Sugar are presented elsewhere.²⁶ In brief, the study compared the effects of nurse case manager and community health worker interventions to usual care on risk factors for diabetes-related complications in urban African Americans. The intervention group had approximately 3 visits per year with the Nurse-Case Manager and 3 visits per year with the Community Health Worker, plus additional contacts as needed. All initial interventions focused on the following domains: diet, physical activity, foot care, vision care, glucose self-monitoring, blood pressure control, medication and appointment adherence, referrals, and smoking cessation. 26 Patients who completed renal evaluations at baseline and after three years were included in this study (n=99). The study was approved by the Johns Hopkins University School of Medicine Joint Committee on Clinical Investigation. All subjects provided written informed consent.

Data Collection and Definitions

Participants attended baseline screening visits and the 3-year follow-up visit at the Johns Hopkins Outpatient Department General Clinical Research Center, where they underwent standardized interviews, physical assessment, and laboratory testing. All data were collected by trained technicians. Blood pressure was assessed using a random-zero sphygmomanometer; the mean of three readings over one visit was used at baseline and follow-up. Systolic blood pressure ≥ 115 mm Hg was considered elevated based on previous studies in type 1 and type 2 diabetes.^{25, 27} Duration of diabetes was determined by interview where the age at diabetes diagnosis was subtracted from the age at baseline. Microalbuminuria was defined as ACR <0.3 (g albumin/g creatinine) and macroalbuminuria was defined as ACR ≥ 0.3 (g albumin/g creatinine). eGFR was calculated based on the Modification of Diet in Renal Disease 4 variable equation.²⁸ Lipid profile (HDL and LDL cholesterol, and triglycerides) was measured after a 10- to 12-hours fast.

Statistical analysis

Descriptive statistics: frequencies for categorical variables mean and standard deviations for continuous variables were used to summarize the data. Chi square (χ^2) statistics and student's t-tests with unequal variance were used to compare baseline and follow up after 3 years with respect to sociodemographic and clinical variables and to compare sociodemographic and clinical variables by microalbuminuria status. Linear regression analysis was used to examine the relationship between baseline clinical measures and change in renal function. Multiple logistic regression analysis was used to identify risk factors associated with the progression from microalbuminuria to macroalbuminuria over 3 years. All models were adjusted for Project Sugar intervention groups. All analyses were conducted using STATA statistical software

(version 9.0; Stata Corporation, College Station, Texas). All tests of significance were two tailed ($\alpha=0.05$).

Results

Of the 186 enrolled African-Americans participants with type 2 diabetes, 99 had data for both the baseline visit and the three year follow-up visit. Compared to the study participants, those with no follow up data at 3 years, had significantly higher proportion of males, $p=0.001$, smokers, $p=0.03$, lower proportion of patients with $HDL \geq 40$, $p=0.02$, and lower proportion of patients with hypertension, $p=0.03$ at baseline.

Mean age was 59.4 ± 8.5 and the majority of patients were female (85%) with a mean duration of diabetes of 9.8 years. Hypertension was the most common comorbid condition (81%) and though the majority (76%) was treated with one or more antihypertensive agent at baseline, only 36% of participants were on angiotensin converting enzyme inhibitors (ACE inhibitors).

Table 2 shows selected socio-demographic and health-related variables by microalbuminuria and macroalbuminuria status. There was a higher proportion of obese and extreme obesity among participants with macroalbuminuria ($p=0.01$). All participants with macroalbuminuria have a systolic blood pressure of 115 mmHg or above compared to 86.2 % of those with microalbuminuria. There was a 12.87 mmHg difference in systolic blood pressure for those with macroalbuminuria compared to those with microalbuminuria ($p=0.01$).

Table 3 shows selected clinical variables at baseline and year 3 and the change by albuminuria status. Among patients with microalbuminuria there was an increase in urine to albumin creatinine ratio as well as an increase in proportion of participants with systolic blood pressure ≥ 115 mmHg. Systolic blood pressure increased by 14.6 mmHg and, as expected, was associated with an increase in proportion of participants taking blood pressure medications. On the other hand, there was a 0.65% decrease in HbA1c as well as in proportion of participants with cholesterol levels ≥ 198 mg/dL and in the proportion of participants with LDL cholesterol levels ≥ 130 mg/dL. The mean decrease in total cholesterol and LDL cholesterol was 11.62 mg/dL and 19.98 mg/dL respectively. In addition, HDL increased by 4.75 mg/dL ($p<0.001$). Among patients with macroalbuminuria, there was a decrease in the proportion of patients with HbA1c >8 , but the decrease when HbA1c is examined as a continuous variable was not statistically significant. There was a significant decrease in LDL cholesterol by 20.56 mg/dL ($p=0.02$) and significant increase in HDL by 4.74 mg/dL ($p=0.04$).

In Figure 1, the changes in both eGFR (A) by albuminuria status over the period of the study are displayed as box plots. In patients with microalbuminuria, a significant increase in eGFR, likely due to hyperfiltration, was noted by the end of the follow up. Figure 1 (B) is dot plot displaying the changes in ACR in each individual patient at baseline and at the end of follow up.

Table 4 shows the results of the logistic regression models that included covariates age, sex, diabetes duration, baseline ACR and baseline blood pressure medication with and without treatment. The degree of ACR and the need for blood pressure medications were independently associated with the transition from microalbuminuria to macroalbuminuria. Participants on blood pressure medications at baseline had an 8 fold odds of progressing from microalbuminuria to macroalbuminuria. When intervention group was added to the model, only the degree of ACR remained independently associated with transition from microalbuminuria to macroalbuminuria though the point estimates were imprecise given the smaller sample size. There was no association of the progression to macroalbuminuria with age, duration of diabetes, the presence of an underlying cardiac disease, the use of antilipidemic agents, or the use of angiotensin converting enzyme inhibitors.

Discussion

This study highlights the evolution of diabetic nephropathy in AA with type 2 diabetes. Our cohort of African American patients with type 2 diabetes and microalbuminuria not infrequently experienced progression to overt proteinuria and none had evidence of regression over 3 years of follow up. Our study demonstrates that the level of blood pressure is a key determinant in the progression of microalbuminuria to macroalbuminuria and its control should be a primary goal in treating this population regardless of the class of antihypertensive used.

The findings in our study are in contrast to those reported in patients with type 1 diabetes with microalbuminuria.²⁵ However, this is not unexpected in light of the fundamental differences in the epidemiology and evolution of the two disorders. First, though no gross structural differences in renal morphology is observed between type 1 and 2 diabetes²⁹, there is clinical evidence of the heterogeneity in the processes leading to the onset of microalbuminuria in patients with type 1 diabetes as only those who develop microalbuminuria early in the course of diabetes progress to overt proteinuria.³⁰ Patients with microalbuminuria and long duration of type 1 diabetes (>25 years) have low risk of progression to advanced diabetic nephropathy. Structural differences were, however, noted by Fioretto et al who reported different patterns of renal injury in older patients with type 2 diabetes compared to younger patients with type 1 diabetes suggesting heterogeneous renal structural injury in type 1 and type 2 diabetes.³¹ Second, there are racial disparities in the incidence of diabetic renal disease according to the type of diabetes with increased risk of diabetic renal disease among African Americans compared to Caucasians.³² Third, the risk of renal disease in AA may, in part, be independent of diabetes.³³ Finally, genetic and/or familial factors are likely to play an important role in these disparities.^{34–37} Though the data on the natural course of microalbuminuria in Caucasians with type 2 diabetes is limited, Patrick et al demonstrated regression of microalbuminuria in some of these patients after only 1 year of follow up.³⁸

The degree of microalbuminuria was an important factor in the progression to overt proteinuria in this cohort. Experimental animal models and human studies suggest that proteinuria itself may contribute to renal injury even in the early stages.^{39, 40} Microalbuminuria independent of diabetes is also an important predictor of endothelial dysfunction, atherosclerosis, cardiovascular events, and mortality.^{11, 13, 15–17, 20, 41–43} Therapeutic interventions that targeted proteinuria have demonstrated an improvement in both microvascular and macrovascular complications of diabetes.^{27, 44–47}

The relationship between hypertension and poor renal outcome is independent of diabetes and other confounding factors and a sustained reduction in blood pressure is probably the most important factor in slowing the course of renal disease for both type 1 and type 2 diabetes. Crook et al. demonstrated better renal survival in AA patients with type 2 diabetes and who had mean arterial pressure between 100 and 110 mm Hg during the course of 5 years of follow-up compared to those who fell outside of that range.⁴⁸ In a randomized, double-blind, placebo-controlled trial in normotensive patients with diabetes type 2, Ravid et al. demonstrated that the initiation of an ACE inhibitor during the early stages of diabetic nephropathy results in long-term stabilization of plasma creatinine levels and of the degree of urinary loss of albumin.⁴⁹ Although the benefit of ACE inhibitors use was not demonstrated in our study, the small number of patients (36%) treated with these agents and relatively short duration of follow-up (3 years) may explain our results. Conversely, in microalbuminuric and proteinuric patients with type 1 and type 2 diabetes, other reports have demonstrated that irrespective of the agent used, treatment of hypertension resulted in a beneficial effect on renal disease.¹⁸ Aggressive targets for blood pressure control in patients with diabetes have been shown to result in

reduction in the development and progression renal disease, as well as decreasing macrovascular events. 50

Our study showed no correlation between glycemic control and the progression of diabetic nephropathy. Although several studies have demonstrated this tight correlation between HbA1c and the rate of eGFR decline in patients with type 1 diabetes^{51–54} most of the studies in patients with type 2 diabetes and proteinuria, in agreement with ours, have failed to demonstrate any significant impact of glycemic control on the progression to overt nephropathy.^{55, 56} It is postulated that glycemic control may have an impact on the onset but not progression of renal disease in patients with type 2 diabetes.^{57, 58} In addition, in contrast to patients with type 1 diabetes^{59–61}, we found no relationship between duration of diabetes in and the progression of microalbuminuria potentially reflecting the overall shorter duration of follow-up in patients with type 2 diabetes (10 years in our cohort) compared to patients with type 1 diabetes (18 years in the study by Perkins et al.²⁵

The study has several limitations. First, it is limited by the overall representativeness of the study population as it only pertains to clinic samples. However, the majority of patients with diabetes are followed in the ambulatory setting and our findings can be applied to this clinic population. Second, there was significant drop out of participants during the follow-up period. However, except for the proportion of smokers, patients with hypertension, and higher HDL values, there was no difference in base line demographics and clinical characteristics among participants who dropped out and those who completed the follow-up. Third, though interventions in Project Sugar may explain our findings, none of these interventions were specifically targeting or had significant effect on renal outcomes. Fourth, although the small sample size with limited power may temper negative results, it may in fact strengthen our positive findings. Finally, the small sample size limited our ability to perform a gender-specific analysis and more studies in African Americans with type 2 diabetes are needed to determine if our findings are gender-specific. Despite the limitations, our study has several advantages including the long follow-up, standardized data collection, and the homogeneity of the studied population. All participants were enrolled in a primary care setting in urban Baltimore. Furthermore, only a few studies have examined the natural history of diabetic nephropathy in African Americans with type 2 diabetes.^{23, 24}

Our study has several clinical implications. Our findings should provide evidence for the development of cost-effective clinical protocols in order to capture the target population at risk for progressive renal disease and provide specific therapeutic interventions. Most importantly, primary prevention should be applied to high risk diabetic AA patients with normal urinary albumin excretion. The incidence of microalbuminuria was shown to be reduced in hypertensive diabetic patients after the use of ACE inhibitor alone or in combination with calcium channel blocker⁶². Though there was no regression of microalbuminuria in our cohort, the majority (68%) experienced no transition to overt proteinuria during the three years of follow-up. The degree of microalbuminuria was a strong predictor for this transition to overt proteinuria, highlighting the importance of early screening in order to identify those with higher degrees of microalbuminuria. Implementing therapeutic interventions for this high risk group is likely to diminish the burden of not only diabetic renal disease but also cardiovascular disease. Interventions should particularly emphasize strategies that target microalbuminuria, aggressive blood pressure control, and life style changes.

In conclusion, our study provides data on the natural history of microalbuminuria in African Americans with type 2 diabetes. In contrast to Caucasians with microalbuminuria and type 1 and type 2 diabetes, our study demonstrates no evidence of regression to normoalbuminuria. In addition, the degree of microalbuminuria and the need for blood pressure medications are independent risk factors in the process of progression to overt nephropathy. These findings

should provide evidence for active primary and secondary prevention programs in order to reduce the burden of diabetic renal disease in this population.

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Figure 1.

Figure 1 (A). Box plot of Glomerular Filtration Rate at baseline and at year 3 by baseline albuminuria status

Figure 1 (B). Dot plot of albumin to creatinine ratio at baseline and at year 3 by baseline albuminuria status for all 99 patients.

Table 1

Selected Socio-Demographic and Health-Related Variables among 99 African Americans with Type 2 Diabetes by Microalbuminuria (<0.3) and Macroalbuminuria (0.3+) Status at Baseline

Characteristics	Microalbuminuria (n=60)	Macroalbuminuria (n=39)	p
Socio-demographic Variables			
Gender			
Male	8 (13.3)	6 (15.4)	0.78
Female	52 (86.7)	33 (84.6)	
Age (years) (cont)	60.0 ± 8.4	58.5 ± 8.8	0.41
Annual Income ^o			
< \$7500	31 (52.5)	21 (53.9)	0.90
\$7500	28 (46.7)	18 (46.2)	
Health Related Variables			
Duration of diabetes (years) (continuous)	9.3 ± 7.6	10.5 ± 13.4	0.52
Duration of diabetes (years) (categorized by median)			
<7	24 (40.0)	20 (51.3)	0.27
7+	36 (60.0)	19 (48.7)	
Smoking			
Never	29 (48.3)	19 (48.7)	0.98
Former	24 (40.0)	16 (41.0)	
Current	7 (11.7)	4 (10.3)	
Medication			
Diabetes Medication			
Yes	53 (88.3)	38 (97.4)	0.10
No	7 (11.7)	1 (2.6)	
Insulin ^d			
Yes	24 (44.4)	13 (35.1)	0.38
No	30 (55.6)	24 (64.9)	
Ace Inhibitor ^d			
Yes	18 (33.3)	15 (40.5)	0.48
No	36 (66.7)	22 (59.5)	
Beta Blocker ^d			
Yes	9 (16.7)	2 (5.4)	0.11
No	45 (83.3)	35 (94.6)	
Calcium Blocker ^d			
Yes	18 (33.3)	14 (37.8)	0.66
No	36 (66.7)	23 (62.2)	
Diuretic ^d			
Yes	21 (38.9)	11 (29.7)	0.37
No	33 (61.1)	26 (70.3)	
Antihyperlipidemic ^d			

Characteristics	Microalbuminuria (n=60)	Macroalbuminuria (n=39)	p
Yes	14 (25.9)	4 (10.8)	0.08
No	40 (74.1)	33 (89.2)	
Comorbid Illness			
Hypertension ^ℓ			
Yes	43 (78.2)	32 (84.2)	0.47
No	12 (21.8)	6 (15.8)	
Ischemic Heart Disease ^ℓ			
Yes	11 (20.0)	3 (7.9)	0.11
No	44 (80.0)	35 (92.1)	
Congestive Heart Failure ^ℓ			
Yes	2 (3.6)	3 (7.9)	0.37
No	53 (96.4)	35 (92.1)	
Hypercholesterolemia ^ℓ			
Yes	17 (30.9)	12 (31.6)	0.95
No	38 (69.1)	26 (68.4)	
Clinical Variables			
Serum Creatinine (md/dL)	1.02± 0.21	1.14± 0.38	0.08
	1.0[0.9 – 1.1]	1.0[0.9 – 1.2]	
eGFR (mL/min)	78.0 ± 16.5	72.6 ± 19.7	0.33
	74.7 [65.0 – 86.5]	72.4 [65.3 – 84.0]	
BMI (kg/m ²) ^ℓ			
Normal (<25)	2 (3.4)	6 (15.8)	0.01 *
Overweight (25–29.9)	21 (35.6)	3 (7.9)	
Obese (30–40)	25 (42.4)	21 (55.3)	
Extreme Obesity (>40)	11 (18.6)	8 (21.1)	
BMI (kg/m ²) ^ℓ	34.21 ± 8.11	34.21 ± 6.64	0.31
HbA1C			
8+	32 (53.3)	25 (64.1)	0.29
<8	28 (46.7)	14 (35.9)	
HbA1C continuous	8.47 ± 1.88	8.77 ± 1.93	0.45
Systolic Blood Pressure (mmHg) ^ℓ			
115+	50 (86.2)	38 (100)	0.02 *
<115	8 (13.8)	0 (0)	
Systolic Blood Pressure (mmHg) ^ℓ continuous	135.10 ± 19.80	147.97 ± 22.95	0.01 *
Blood Pressure (mmHg)			
BP>=120/80	24 (40.0)	20 (51.3)	0.27
BP<120/80	36 (60.0)	19 (48.7)	
Blood Pressure Medication ^ℓ			
Yes	41 (69.5)	33 (84.6)	0.09
No	18 (30.5)	6 (15.4)	

Characteristics	Microalbuminuria (n=60)	Macroalbuminuria (n=39)	p
Cholesterol (mg/dL)			
198+	38 (63.3)	26 (66.7)	0.74
<198	22 (36.7)	13 (33.3)	
Cholesterol (mg/dL) continuous	213.70 ± 40.99	218.95 ± 52.06	0.55
Triglycerides (mg/dL)			
145+	12 (20.0)	12 (30.8)	0.22
<145	48 (80.0)	27 (69.3)	
Triglycerides (mg/dL) continuous	113.47 ± 47.40	125.59 ± 50.56	0.24
LDL Cholesterol (mg/dL)			
130+	36 (60.0)	25 (64.1)	0.68
<130	24 (40.0)	14 (35.9)	
LDL Cholesterol (mg/dL) continuous	140.93 ± 33.95	146.64 ± 48.16	0.38
HDL Cholesterol (mg/dL)			
<40	8 (13.3)	9 (23.1)	0.58
40+	52 (86.7)	30 (76.9)	
HDL Cholesterol (mg/dL) continuous	49.73 ± 12.40	47.13 ± 11.75	0.30
Waist Hip Ratio ^o	0.92 ± 0.06	0.92 ± 0.08	0.80

All results presented as n (%) or mean ± SD,

^o
n=98,

^o
n=97,

^o
n=96 at,

^o
n=91,

^o
n=93

*
P<0.05

Table 2

Selected Clinical Variables among African Americans with Type 2 Diabetes at Baseline and Year 3 by Microalbuminuria and Macroalbuminuria status.

Characteristics	Microalbuminuria				Macroalbuminuria			
	Baseline	Year 3	Δ	p	Baseline	Year 3	Δ	p
Serum creatinine	1.02 $\pm$ 0.21	0.97 $\pm$ 0.25	0.05 $\pm$ 0.17	0.03*	1.14 $\pm$ 0.38	1.39 $\pm$ 1.28	-0.25 $\pm$ 0.98	0.11
eGFR (mL/min)	1.0 [0.9 – 1.1]	0.9 [0.8 – 1.1]			1.0 [0.9 – 1.2]	1.0 [0.8 – 1.2]		
	78.04 $\pm$ 16.46	84.74 $\pm$ 24.12	6.71 $\pm$ 18.08	0.02	72.60 $\pm$ 19.75	78.30 $\pm$ 36.13	5.70 $\pm$ 25.25	0.19
	74.71 [65.04 – 86.45]	80.68 [70.00 – 95.92]			72.37 [65.31 – 84.05]	78.03 [57.72 – 97.41]		
Urine Albumin to Creatinine Ratio (g/g)	0.12 $\pm$ 0.07	0.77 $\pm$ 1.82	0.65 $\pm$ 1.82	<0.001*	3.92 $\pm$ 5.89	8.97 $\pm$ 15.09	5.04 $\pm$ 14.30	0.40
	0.11 [0.06 – 0.18]	0.14 [0.08 – 0.40]			1.24 [0.55 – 4.42]	1.14 [0.38 – 10.80]		
BMI (kg/m ²) ^a								
Normal	2 (3.4)	2 (3.5)		0.11	6 (15.8)	5 (15.2)		0.43
Overweight	21 (35.6)	9 (15.8)			2 (7.9)	0 (0)		
Obese	25 (42.4)	33 (57.9)			21 (55.3)	20 (60.6)		
Extreme Obesity	11 (18.6)	13 (22.8)			8 (21.1)	8 (24.2)		
	34.21 $\pm$ 8.11	34.80 $\pm$ 7.04	0.59 $\pm$ 4.71	0.16	34.21 $\pm$ 6.64	34.00 $\pm$ 7.39	-0.21 $\pm$ 5.67	0.95
HbA1C								
8+	32 (53.3)	26 (43.3)		0.27	25 (64.1)	14 (35.9)		0.01*
<8	28 (46.7)	34 (56.7)			14 (35.9)	25 (64.1)		
	8.48 $\pm$ 1.88	7.83 $\pm$ 1.77	-0.65 $\pm$ 2.37	0.05*	8.77 $\pm$ 1.93	8.44 $\pm$ 2.75	-0.34 $\pm$ 2.30	0.27
Systolic Blood Pressure (mm Hg) ^b								
115+	50 (86.2)	58 (96.7)		0.04*	38 (100)	36 (92.3)		0.08
<115	8 (13.8)	2 (3.3)			0 (0)	3 (97.7)		
	135.10 $\pm$ 19.80	149.71 $\pm$ 24.14	14.60 $\pm$ 25.01	<0.001*	147.97 $\pm$ 22.95	149.69 $\pm$ 23.85	1.72 $\pm$ 31.11	0.81
Blood Pressure (mm Hg)								
BP $\geq$ 120/80	24 (40.0)	24 (40.0)		1.00	20 (51.3)	18 (46.1)		0.65
BP < 120/80	36 (60.0)	36 (60.0)			19 (48.7)	21 (53.9)		
Blood Pressure Medication ^c								
Yes	41 (69.5)	50 (83.3)		0.08	33 (84.6)	32 (82.1)		0.76
No	18 (30.5)	10 (16.70)			6 (15.4)	7 (18.0)		

Characteristics	Microalbuminuria				Macroalbuminuria			
	Baseline	Year3	Δ	p	Baseline	Year 3	Δ	p
Cholesterol (mg/dL)								
198+	38 (63.3)	27 (45.0)		0.04*	26 (66.7)	24 (61.5)		0.64
<198	22 (36.7)	33 (55.0)			13 (33.3)	15 (38.5)		
	213.70 ± 40.99	202.40 ± 41.09	-11.30 ± 42.37	0.04*	218.95 ± 52.06	206.69 ± 51.46	-12.26 ± 52.14	0.31
Triglycerides(mg/dL)								
145+	12 (20.0)	16 (26.7)		0.39	12 (30.8)	9 (23.1)		0.44
<145	49 (80.0)	45 (73.3)			27 (69.2)	30 (76.9)		
	113.47 ± 47.40	129.48 ± 94.55	16.02 ± 73.02	0.12	125.59 ± 50.86	124.77 ± 58.81	-0.82 ± 37.50	0.76
LDL Cholesterol (mg/dL) ^ω								
130+	36 (60.0)	21 (36.2)		0.01*	25 (64.1)	18 (46.2)		0.11
<130	24 (40.0)	37 (63.8)			14 (35.9)	21 (53.9)		
	139.79 ± 32.48	120.00 ± 34.00	-19.79 ± 36.61	<0.001*	146.64 ± 48.16	126.08 ± 50.59	-20.56 ± 50.78	0.03*
HDL Cholesterol (mg/dL)								
<40	8 (13.3)	8 (13.3)		1.00	9 (23.1)	9 (23.3)		1.00
40+	52 (86.7)	52 (86.7)			30 (76.9)	30 (76.9)		
	49.23 ± 12.40	54.48 ± 12.19	4.75 ± 9.29	<0.001*	47.13 ± 11.75	51.87 ± 13.56	4.74 ± 15.30	0.04*
Waist Hip Ratio	0.92 ± 0.06	0.92 ± 0.07	0.002 ± 0.07	0.86	0.92 ± 0.08	0.93 ± 0.07	0.01 ± 0.08	0.49

All results presented as %, mean ± SD, median [interquartile range].

^ρ n=91,
^λ n= 97 @ baseline,
^ω n=98 at follow up,
^ζ n=99 @ baseline
* P<0.05

Table 3

Odd Ratio of Progression to Macroalbuminuria for 61 African Americans with Type 2 Diabetes and microalbuminuria

Covariate	Model 1		Model 2	
	Odds Ratio	95% Confidence Interval	Odds Ratio	95% Confidence Interval
Age	0.94	0.85, 1.04	0.92	0.82, 1.03
Sex	0.83	0.11, 6.07	0.81	0.11, 5.85
Diabetes Duration	0.93	0.82, 1.04	0.92	0.82, 1.04
Baseline blood pressure medication	8.96	1.35, 59.70	8.24	0.97, 70.24
Baseline albumin to creatinine ratio				
Q1 [0.06]	1.00		1.00	
Q2 [0.06 – 0.11]	15.96	0.93, 274.59	24.70	1.12, 543.80
Q3 [0.11 – 0.19]	50.47	3.10, 821.72	91.7	3.77, 2229.75
Q4 [>0.19]	35.51	2.21, 571.65	48.07	2.16, 1069.38

Model 1 includes age, sex, diabetes duration, baseline blood pressure medication use and baseline albumin to creatinine ratio

Model 2 includes age, sex, diabetes duration, baseline blood pressure medication use and baseline ACRin quartiles with the 1st quartile as the reference and intervention group