



ORIGINAL ARTICLE

Microalbuminuria, renal function and waist:hip ratio in black hypertensive Jamaicans

M Reid, F Bennett, R Wilks and T Forrester

Tropical Metabolism Research Unit, University of the West Indies, Mona, Kingston 7, Jamaica, WI

Objective: To investigate the relationship between blood pressure (BP), renal haemodynamics, anthropometric measures of obesity and urinary albumin excretion in hypertension and in a control group.

Methods: Urinary albumin, BP and anthropometric measurements were carried out in patients attending the hypertension clinic of the University Hospital of the West Indies. A randomised stratified sample was then selected for renal haemodynamic assessment. A normoalbuminuric control group without hypertension or diabetes was also selected. Renal haemodynamics was assessed by measuring glomerular filtration rate using 51-chromium edetic acid (51Cr-EDTA) and renal blood flow using 125-iodohippurate (125-PAH).

Results: Urinary albumin excretion was positively and significantly correlated with systolic pressure ($\beta = 0.011$, $P < 0.003$, $R = 0.22$), current body weight ($\beta = 0.014$, $P < 0.04$, $R = 0.15$) and the presence of diabetes ($\beta = 0.9$, $P < 0.001$, $R = 0.3$). In the sample selected for

renal haemodynamics, patients with microalbuminuria had lower age-adjusted corrected renal blood flow ($P < 0.006$), effective renal plasma flow ($P < 0.006$) and higher filtration fraction ($P < 0.006$) when compared with patients without microalbuminuria. Glomerular filtration rate in patients with microalbuminuria was not different from those without. Urinary albumin excretion was positively and significantly correlated with systolic pressure ($\beta = 0.016$, $P < 0.003$, $R = 0.40$) and inversely related to corrected renal blood flow ($\beta = -1.13$, $P < 0.0002$, $R = 0.46$). Waist:hip ratio was inversely related to corrected renal blood flow ($\beta = -1.74$, $P < 0.02$, adjusted $R = 0.48$).

Conclusion: Systolic BP, diabetes and body weight were significant predictors of albuminuria in our patients. Microalbuminuria and body fat distribution as assessed by waist:hip ratio were important determinants of renal haemodynamics in this population.

Keywords: microalbuminuria; renal function; waist:hip ratio; black; Jamaica; hypertension; diabetes mellitus

Introduction

Microalbuminuria is defined as urinary albumin excretion that is greater than normal ($20 \mu\text{g}/\text{min}$ or $30 \text{mg}/\text{l}$) but less than that detectable by conventional dipstick methods ($200 \mu\text{g}/\text{min}$ or $300 \text{mg}/\text{l}$).¹ It has emerged as a useful prognostic marker in diabetes mellitus, where microalbuminuria is associated with progression to chronic renal failure, and a greater risk of morbid and fatal cardiovascular complications.²

The risk of renal pathology associated with microalbuminuria in essential hypertension is less clear. Several studies have examined the relationship between blood pressure (BP) and urinary albumin excretion and have reported a positive relationship in the hypertensive range of BP, ie, systolic $>140 \text{mm Hg}$ and diastolic $>90 \text{mm Hg}$.^{3–5} The mechanism underlying this association is unclear and interventional studies which have sought to reduce

urinary albumin excretion by lowering systemic and intraglomerular pressure have produced conflicting results.^{6,7}

At the population level, body mass index and abdominal obesity as assessed by waist:hip ratio have been found to be independent risk factors for cardiovascular morbidity.⁸ They are both positively correlated with urinary albumin excretion and BP.⁹ In addition, body mass index is positively correlated with renal blood flow and inversely related with renal vascular resistance.^{9,10} In the Caribbean, body mass index has emerged as the major environmental determinant of hypertension prevalence.¹¹

Renal haemodynamic measurements are sensitive markers of renal function.¹² Blacks in the Caribbean may be prone to developing renal complications as a consequence of essential hypertension.¹³ The renal complications may be related to greater severity of BP, poor access to health care, or the interaction of known risks factors with a fertile genetic substrate. We wished to determine in this population of black hypertensives whether microalbuminuria was correlated with BP and measures of adiposity. This study also sought to explore the relationship between microalbuminuria and renal pathophysiology as quantified by renal haemodynamics.

Correspondence: Professor Terrence Forrester, Tropical Metabolism Research Unit, University of the West Indies, Mona, Kingston 7, Jamaica, WI

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Patients and methods

The hypertension clinic of the University Hospital of the West Indies is a referral centre for the management of hypertension in Jamaica. Patients with primary hypertension attending this clinic, were considered eligible for participation in this study. Eligibility criteria included, age less than 75 years, mild-to-moderate hypertension (supine diastolic pressure 95–115 mm Hg inclusive), and serum creatinine <200 $\mu\text{mol/l}$ and informed consent. Exclusion criteria were history of malignant hypertension, congestive cardiac failure, myocardial infarction, pregnancy and proteinuria >300 mg/l.

Two hundred and eight patients were eligible for recruitment. Seventy-nine of these patients also had diabetes mellitus. All patients had a mid-stream urine collected for culture and if there was no growth, patients had another clinic urine specimen for urine albumin estimation. Patients were asked to complete an interviewer-administered questionnaire to ascertain information about the duration of their illness (hypertension and diabetes), alcohol consumption, smoking habits and the presence or absence of a first degree relative with hypertension. BP was measured by a trained nurse using a mercury sphygmomanometer. Height and weight were measured in the clinic using a beam balance and a mounted stadiometer. Blood was taken for serum urea and creatinine estimation. Patients were subsequently divided into two subgroups based on urinary albumin excretion: Group 1, hypertensives with microalbuminuria ($n = 56$); Group 2, hypertensives without microalbuminuria ($n = 152$). The patients in each of these groups were further stratified by the presence or absence of diabetes mellitus and age-decade. In each of these strata, patients were then randomly selected from the normoalbuminuric group to match patients in the microalbuminuric group for renal haemodynamic measurement. A sample size of 35 patients in each of these groups was required to demonstrate a significant difference of 10 ml/min/1.73 m² in mean glomerular filtration rate with a standard deviation of 15 at the 5% significance level and 80% power. Seventy-three patients were studied, 34 with microalbuminuria and 39 without microalbuminuria. A control group of eight patients without hypertension and diabetes was selected from patients attending the surgical out-patient clinic of the University Hospital.

Procedures

Baseline measurements: After a drug washout period of 3 days, patients were admitted to the ward of the Tropical Metabolism Research on the evening prior to the measurement of the haemodynamic variables. Each patient was fasted (8 h) overnight and had a timed overnight collection of urine for measurement of urinary albumin excretion from midnight until 08.00 am. Urinary albumin was measured using a fluoroimmunoassay¹⁴ and microalbuminuria was defined as albuminuria greater than normal 20 $\mu\text{g/min}$ but less than 200 $\mu\text{g/min}$.

Immediately following the urinary collection,

anthropometric measurements were made on the left side of the body, with the patients in undergarments, and included triceps and subscapular skinfolds, waist circumference at the umbilicus, and hip circumference at the level of the greater trochanter. The patients' weight was measured using an electronic Sartorius Balance (Model F 150S KR), height with a Holtain stadiometer, and skinfolds by Holtain calipers.¹⁵

Renal haemodynamics: Patients then had an intravenous catheter inserted in each arm at the antecubital fossa. The catheter in the right arm was used for the infusion of the radioactive markers used to estimate glomerular filtration rate and renal blood flow, whilst that in the left arm was used for venous sampling. Blood was taken for measuring haemoglobin, haematocrit, plasma glucose, serum cholesterol, urea and creatinine.

Glomerular filtration rate and renal plasma flow were measured as the clearance of 51-Chromium EDTA (Amersham) and 125-Iodohippurate Sodium (Amersham), respectively. Thirty microcurie of 51-Chromium EDTA and 30 microcurie of 125-Iodohippurate Sodium was mixed with 0.9% normal saline to obtain a 20 mls solution. A bolus of 3 mls was given and the remainder infused at a rate of 2.6 mls per hour using a Travenol syringe infusion pump (model AS5D). During the 1 h equilibration period, BP was measured in the supine position at 15-min intervals using an oscillometric technique (Dinamap model 1846SX, Criticon, USA). The average of the five measurements made was used in the analysis. Urine was collected hourly for the next 4 h and blood taken at the midpoint of each clearance period. The blood and urine samples were assayed for 125-Iodohippurate and 51-Chromium EDTA by counting gamma emissions (Beckman Gamma counter model 5500b). Patients drank 300 mls of water before the infusion began and during each clearance period.

Glomerular filtration rate was calculated as the clearance of 51 Cr-EDTA. Renal plasma flow was taken as the Clearance of 125-Iodohippurate. Renal vascular resistance was calculated using Gomez formula:¹⁶

$$((\text{MBP} - 10 \text{ mm Hg}) * 60 / \text{RBF}) * 1328$$

where MBP is the mean BP during the equilibration period, RBF is corrected renal blood flow and 10 mm Hg is the assumed pressure in the renal vein.

The absolute values for the renal haemodynamic variables were adjusted for body surface area. Body surface area was calculated using the formula of Dubois.¹⁷ Urinary albumin excretion rate was calculated as:

$$\text{urine albumin concentration } (\mu\text{g/l}) * \text{urine volume (l)/time of collection in minutes.}$$

Waist:hip ratio was calculated as the ratio of waist-to-hip circumferences. Subscapular-triceps ratio was calculated as the ratio of subscapular skinfold to triceps skinfold thicknesses. Percentage body fat was calculated according to Durnin's equation.¹⁸ Ethical approval for the study was obtained from the

Medical Ethics Committee of the University Hospital of the West Indies.

Statistical analysis

Data are expressed as the mean \pm s.e.m. or 95% confidence intervals. For comparisons of the proportion of subjects in the two groups the χ^2 test was used as appropriate. Differences in group means were analysed using the independent t -test as well as a two way analysis of covariance (ANCOVA). This model compared the main effects of albumin excretory status and the presence or absence of diabetes mellitus as well as their interaction with age as the covariate. The variables, albumin excretion, corrected renal blood flow, RBF, corrected renal plasma flow, RPF, and renal vascular resistance, RVR were skewed. These variables were Napier logarithmically transformed and geometric means used in the analysis. Anthropometric measures, fasting blood glucose, BP (systolic, diastolic), albuminuric excretory status, diabetic status, sex and age were entered into a stepwise multiple regression to determine the relationship between these factors and renal blood flow, renal plasma flow and renal vascular resistance. The SPSS statistical package was used for analysis.

Results

There were no differences between the microalbuminuric and normoalbuminuric group in mean age, serum creatinine, body mass index, weight, height, systolic BP, diastolic BP and duration of hypertension even after adjusting for age and the presence of diabetes (Table 1). The sex distribution and the frequency of current cigarette smokers and current consumers of alcohol was similar between groups. The serum urea was, however, significantly higher in the microalbuminuric group than the normoalbuminuric group (6.5 ± 3.7 vs 5.4 ± 1.9 , $P <$

0.02). There was an association between the presence of diabetes and microalbuminuria, in that a significantly greater proportion (61%) of microalbuminuric patients were diabetic ($\chi^2 = 16.8$, $P = 0.0004$).

Within the entire population, systolic BP ($\beta = 0.6$, $P < 0.01$) and serum urea ($\beta = 0.05$, $P < 0.01$) varied positively with age. Urinary albumin excretion was positively and significantly correlated with systolic pressure ($\beta = 0.011$, $P = 0.013$, Figure 1), current body weight ($\beta = 0.77$, $P < 0.025$, Figure 2) and the presence of diabetes ($\beta = 0.79$, $P < 0.005$).

In the sample selected for renal haemodynamic measurements there were no differences in sex distribution, biochemical or anthropometric indices between patients with microalbuminuria and patients without microalbuminuria. The hypertensive patients with diabetes were, however, older than those without (Table 2, $P < 0.003$).

In this sub-sample, systolic pressure also varied positively with age ($\beta = 0.9$, $R^2 = 0.13$, $P < 0.002$). As expected, the control group had a significantly

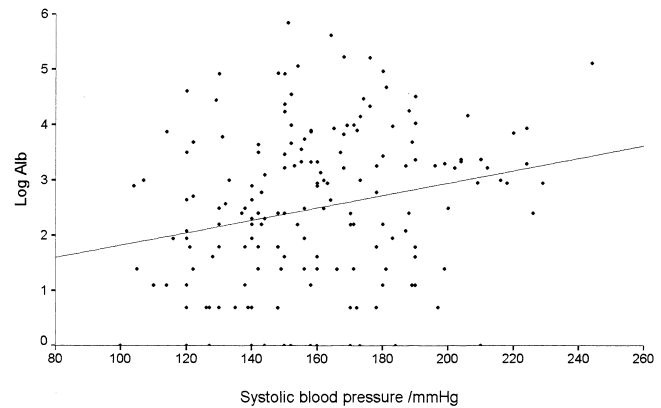


Figure 1 Relationship between albumin excretion and systolic pressure. (Slope = 0.011; $R = 0.22$; $P = 0.003$; Alb = albumin concentration).

Table 1 Clinical characteristics of patients on enrolment

Variables	Microalbuminuric patients (n = 56)	Normoalbuminuric patients (n = 152)
Age (yrs)	54.3 (48.9, 58.2)	50.9 (46.4, 51.9)
Sex (M/F)	18/38	58/94
Presence of diabetes mellitus (count/proportion)	34/61% [†]	45/42%
Number of patients with positive family history of hypertension	33	102
Duration of hypertension (yrs)	11.8	9.8 (8.5, 11.2)
Number of patients who were smokers	11	18
Weight (kg)	77.7 (73.2, 86.4)	75.8 (72.8, 78.8)
Height (cm)	164 (161, 168)	164 (162, 166)
Body mass index (kg/m ²)	29 (27.1, 31.9)	28 (27.2, 29.4)
Serum urea (mmol/l)	6.5 (5.1, 7.0) [‡]	5.4 (5.0, 5.7)
Serum creatinine (μ mol/l)	121 (109, 144)	113 (104, 118)
Systolic blood pressure (mm Hg)	166 (156, 177)	158 (151, 162)
Diastolic blood pressure (mm Hg)	95 (91, 102)	92 (89, 96)
Urinary albumin concentration (mg/l) ^a	66.7 (78.6, 56.6) [§]	6 (5, 7.2)
Urinary albumin/creatinine ratio (g/mol) ^a	5.1 (4.1, 6.4) [§]	0.5 (0.4, 0.6)

^aValues are geometric means with 95% confidence intervals. [†] $P < 0.0004$, [‡] $P < 0.02$, [§] $P < 0.0001$.

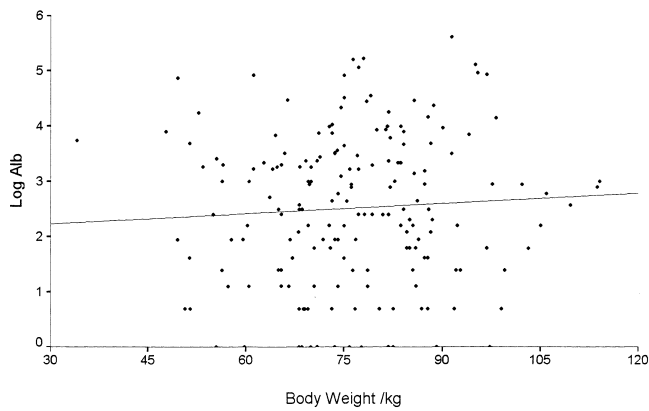


Figure 2 Relationship between albumin excretion and body weight. (Slope = 0.014; $R = 0.15$; $P = 0.04$; Alb = albumin concentration).

lower BP than the diseased groups (age-adjusted mean difference with s.e.m. = 44 mm Hg \pm 11, $P < 0.0001$, Table 3). Differences in systolic and diastolic BPs between hypertensive patients with and without microalbuminuria, were dependent upon whether there was concomitant diabetes. Thus, in patients with hypertension alone systolic and diastolic BPs were significantly higher in those with microalbuminuria compared to those without (age-adjusted mean systolic pressure with s.e.m. = 184 \pm 4 mm Hg vs 161 \pm 6 mm Hg, $P < 0.02$; age-adjusted mean diastolic pressure = 99 mm Hg vs 86 mm Hg, $P < 0.008$). In those hypertensive patients with dia-

betes, BP was not different in those with microalbuminuria compared to those without (age-adjusted systolic BP means with s.e.m., 161 \pm 6 mm Hg vs 167 \pm 7 mm Hg; age-adjusted mean diastolic pressures = 83 mm Hg \pm 3 vs 91 mm Hg \pm 3, Table 3).

There were no differences between controls and diseased groups for age adjusted means for glomerular filtration rate, corrected renal blood flow, filtration fraction and renal vascular resistance (Table 3). Corrected renal plasma flow ($\beta = -0.008$, $P < 0.048$), corrected renal blood flow ($\beta = -0.009$, $P < 0.03$) and glomerular filtration rate ($\beta = -0.95$, $P < 0.0001$) varied inversely with age in this sub-sample. Microalbuminuric patients had significantly lower corrected renal blood flow ($P < 0.006$) and corrected renal plasma flow ($P < 0.006$) but higher filtration fraction ($P < 0.006$), than normoalbuminuric patients. In patients with hypertension alone with microalbuminuria, renal vascular resistance was 1.8 times higher than those hypertensives without microalbuminuria ($P < 0.0002$). Similarly, hypertensive microalbuminuric patients had renal vascular resistance which was 1.6 times higher than controls ($P < 0.03$). However, in those hypertensive patients with diabetes, renal vascular resistance was not different in those with microalbuminuria, compared to those without (Table 3).

Within the entire sub-sample, $n = 81$, urinary albumin excretion was positively and significantly related to systolic pressure ($\beta = 0.016$, $P < 0.003$, R

Table 2 Clinical characteristics of patients who had haemodynamic measurements

Variables	Microalbuminuric hypertensives			Normoalbuminuric hypertensives			Controls ($n = 8$)
	All microalbuminuric patients ($n = 34$)	Hypertension only ($n = 15$)	Hypertension and diabetes ($n = 19$)	All patients without albuminuria ($n = 39$)	Hypertension only ($n = 22$)	Hypertension and diabetes ($n = 17$)	
Age (yrs)	56 (51, 60)	51 (42.9, 59.2)	59 (52.8, 65.2)*	49 (45, 52)	46 (41.4, 50)	56 (51.6, 60.9)*	41.7 (29.4, 54)
Sex (M/F)	11/23	6/9	5/14	7/32	4/18	3/14	3/4
Duration of diabetes mellitus (yrs)			12.4 (8.2, 16.1)			10.1 (5.3, 14.8)	
Weight (kg)	75.4 (68.5, 82.3)	73.04 (66, 80.1)	71.3 (65.7, 89)	73.7 (70.2, 77)	75.9 (71.5, 80.2)	74.8 (68.2, 81.4)	63.7 (56.8, 70.7)
Height (cm)	167 (164, 170)	166 (162, 170)	167 (162, 172)	165 (163, 167)	164 (161, 167)	163 (159, 167)	169 (160, 179)
Body mass index (kg m ²)	27 (24.7, 29.1)	26.4 (24.1, 28.6)	27.4 (23.7, 31.1)	27 (26, 28)	28.1 (26.4, 29.9)	28 (25.8, 30.1)	22.4 (19.7, 25)
Percent body fat (%)	32 (30, 35)	32 (28, 36)	33 (29, 36)	32 (30, 35)	34 (31, 37)	35 (32, 37)	22 (11, 34)
Subscapular: triceps ratio	1.3 (1.2, 1.4)	1.3 (1.1, 1.6)	1.3 (1.1, 1.4)	1.1 (1.0, 1.2)	1.1 (0.9, 1.3)	1.4 (1.0, 1.3)	1.2 (0.8, 1.5)
Waist:hip ratio	0.98 (0.94, 1)	0.95 (0.9, 1)	0.99 (0.95, 1)	0.94 (0.92, 0.96)	0.93 (0.9, 0.96)	0.99 (0.97, 1)	0.85 (0.82, 0.88)
Fasting glucose (mmol/l)	6.6 (5.6, 7.6)	4.7 (4.3, 5.1)	8.2 (6.7, 9.6)	5.8 (4.6, 7)	4.8 (4.4, 5.2)	7.5 (4.5, 10.6)	4.6 (3.8, 5.4)
Cholesterol (mmol/l)	5.4 (4.8, 5.9)	4.9 (4.3, 5.5)	5.8 (5.4, 6.2)	5.5 (4.3, 6.7)	5.6 (5.2, 6)	5.4 (3.4, 7.4)	4.5 (3.9, 5.1)
Haemoglobin (g/dl)	12.3 (11.5, 13.1)	12.4 (11.3, 13.5)	12.2 (11, 13.4)	12.6 (12.1, 13)	12.4 (11.6, 13.1)	12.5 (11.9, 13.2)	13.4 (11.8, 15)

Values are means with 95% confidence intervals.

* $P < 0.003$ comparing hypertensive patients with diabetes vs hypertensive patients without diabetes.

Table 3 Blood pressure and renal function

Variables	Hypertensive patients with microalbuminuria			Hypertensive patients without microalbuminuria			Controls (n = 8)
	All microalbuminuric patients (n = 34)	Hypertension only (n = 15)	Hypertension and diabetes (n = 19)	All normoalbuminuric patients (n = 39)	Hypertension only (n = 22)	Hypertension and diabetes (n = 17)	
Systolic blood pressure (mm Hg)	172 ± 4	184 ± 4 [‡]	161 ± 6	164 ± 5	161 ± 6	167 ± 7	117 ± 5 [†]
Diastolic blood pressure (mm Hg)	91 ± 2	99 ± 3	83 ± 3	88 ± 2	86 ± 3	91 ± 3	64 ± 4
Serum urea (mmol/l)	5.0 ± 0.2	4.1 ± 0.2	5.8 ± 0.3	4.5 ± 0.2	4.1 ± 0.3	4.9 ± 0.3	4.4 ± 0.2
Serum creatinine (μmmol/l)	95 ± 7	102 ± 10	87 ± 8	75 ± 5	74 ± 6	76 ± 8	80 ± 6
Glomerular filtration rate (ml/min/1.73 m ²)	78 ± 4	75 ± 7	82 ± 8	80 ± 4	79 ± 6	81 ± 8	84 ± 12
Filtration fraction (%)	24 ± 1 [‡]	24 ± 2	24 ± 2	20 ± 1	17 ± 1	22 ± 1	20 ± 2
Renal blood flow ^a (ml/min/1.73 m ²)	492 (446, 543) [‡]	494 (445, 549)	452 (419, 487)	656 (528, 814)	713 (676, 753)	626 (592, 662)	745 (643, 864)
Renal plasma flow ^a (ml/min/1.73 m ²)	301 (269, 337) [‡]	303 (278, 331)	278 (260, 298)	403 (330, 492)	436 (416, 457)	389 (370, 409)	455 (399, 520)
Renal vascular resistance (dynes* sec/cm ⁵) ^a	17223 (14379, 20629)	17075 (15285, 19076) [§]	18764 (17340, 20306)	12338 (12224, 12454)	11209 (10597, 11857)	12888 (12098, 13732)	10171 [¶] (9080, 11384)
Albumin excretion rate (μg/min) ^a	41 (26, 66)	38 (31, 48)	45 (38, 53)	13 (12, 14)	11 (10, 13)	14 (13, 16)	11 (4, 34)

^aValues are geometric means with 95% confidence intervals.

[†]*P* < 0.0001 comparing controls to hypertensive patients; [‡]*P* < 0.006 comparing patients with microalbuminuria vs patients without microalbuminuria; [§]*P* < 0.002 comparing microalbuminuric hypertensives with normoalbuminuric hypertensives; [¶]*P* < 0.03 comparing microalbuminuric hypertensives to controls.

= 0.4) and inversely related to corrected renal blood flow ($\beta = -1.13$ *P* < 0.0002, *R* = 0.46). Anthropometric measures were not significant predictors of urinary albumin excretion. However, waist:hip ratio was inversely related to corrected renal blood flow (Figure 3, $\beta = -1.74$, *P* < 0.02, adjusted *R* = 0.48) after adjusting for age, albumin excretory status and diabetic status.

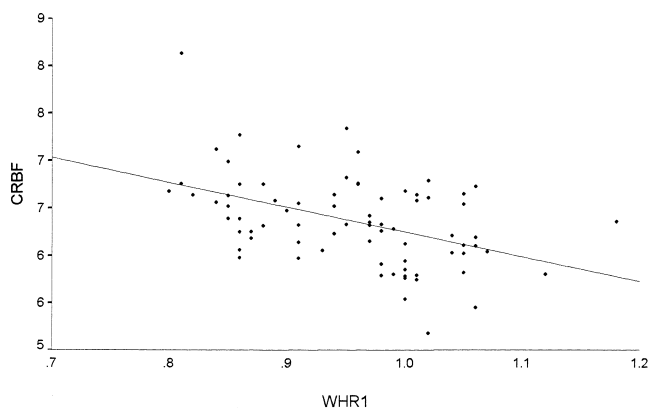


Figure 3 Relationship between renal blood flow and waist:hip ratio. (Slope = -1.74; *R* = 0.48; *P* = 0.02; CRBF = natural log of corrected renal blood flow; WHR = waist:hip ratio).

Discussion

The reported prevalence of microalbuminuria among patients with hypertension is about 30% (0–40% range).¹⁹ In the present study, the prevalence was 27%.

In the Caribbean, BP has been noted to rise with age, a characteristic finding of ‘westernized society’. This rise in BP with age has been related to an increased prevalence of obesity as measured by body mass index.¹¹ In our enrolled sample, the mean body mass index was 28.5 kg/m² reflecting the relative obesity of our hypertensive population.

Significant predictors of albuminuria in our patients were systolic pressure, body weight and the presence of diabetes. The aetiology of the relationship between measures of obesity and albuminuria is unclear but may be related to hyperinsulinaemia, or renal haemodynamic changes.²⁰ In our population of relatively obese subjects, we were not able to demonstrate a relationship between body mass index and albuminuria and this may have been due to the small variation in body mass index in our sample.

Renal haemodynamic measurements are sensitive indicators of renal functional abnormalities. Our controls had lower age-adjusted values for renal

blood flow (681 ml/min/m² vs 880 ml/min/m²) and higher renal resistance (9947 vs 9281 dynes cm⁻⁵) than published norms. This is consistent with findings by Levy *et al*²¹ that blacks have different renal haemodynamic measurements compared to whites.

The kidney in essential hypertension undergoes haemodynamic changes characterised by a reduction in glomerular filtration rate, effective renal plasma flow and increases in renal vascular resistance, RVR and filtration fraction (FF). These functional changes are thought to be associated with loss of renal mass, related to pathological changes and altered flow in intra renal blood vessels.²²

In this study, hypertensive patients exhibited the pattern of changes described in primary hypertension. However, hypertensive patients with microalbuminuria, had more severe renal haemodynamic changes and a higher BP when compared to normoalbuminuric hypertensives and controls. The lower blood flow seen in microalbuminuric hypertensives probably represents an exaggerated renal constrictor response or perhaps a greater degree of nephrosclerosis. An exaggerated renal circulatory response is probably the result of an imbalance in neurohumoral, paracrine and endocrine control of renal flow. Thus in studies of normotensive relatives of patients with essential hypertension increased renal vasoconstriction in response to mental stress and postural changes has been shown.²³ Further, in spontaneously hypertensive rats, an experimental model of essential hypertension, chronic inhibition of nitric oxide synthesis, a renal vascular paracrine factor, resulted in a pathophysiological state indistinguishable from hypertensive nephrosclerosis.²⁴

Albumin excretion in hypertension is thought to be due to renovascular aberrations, as well as the loss of glomerular capillary permselectivity.²⁵ Glomerular permselectivity was not assessed in this study, but cross-sectional studies which have used β^2 -microglobulin as an index of glomerular permselectivity have not shown increased albumin permeability in the renal tubules in essential hypertension. Further, there was a significant positive correlation between systolic pressure and albuminuria implicating glomerular hypertension as an aetiological factor in increased urinary albumin excretion.

We were not able to demonstrate any relationship between urinary albumin excretion and absolute measures of obesity such as body mass index, or percent body fat. However, we found a significant inverse relationship between waist:hip ratio and corrected renal blood flow, a result similar to that reported by Scaglione *et al*²⁶ employing a different study design. In their study, hypertensive and normotensive patients with central obesity defined as a waist:hip ratio >0.81 for females and >0.92 for men, had lower renal blood flow and lower plasma flows compared to patients with peripheral fat distribution. The mechanism underlying this relationship is unclear but may be due to humoral or genetic factors.

In conclusion, systolic BP, body weight and concomitant diabetes were significant predictors of albuminuria in our patients. Microalbuminuria and

body fat distribution as assessed by waist:hip ratio were important determinants of renal haemodynamics in this population.

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