ORIGINAL ARTICLE

# PREVALENCE OF MICROALBUMINURIA IN UNTREATED NIGERIAN HYPERTENSIVE PATIENTS

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**ABSTRACT**

**Background:** The burden of cardiovascular disease imposed by hypertension is a result of target organ damage. Microalbuminuria (MA) is the first clinical expression of nephropathy and has become a cardiovascular and/or renal disease prognostic indicator for hypertensive subjects.

**Objectives:** To establish the prevalence of MA among newly diagnosed hypertensive patients using the simple spot urine Albumin-Creatinine Ratio (ACR).

**Method**: One hundred and eighty six newly diagnosed hypertensive patients were enrolled for assessment of MA using spot urine ACR. Those with overt proteinuria, diabetes mellitus, overt kidney disease and other potential causes of albuminuria were excluded. Spot urine was obtained for measurement of albumin and creatinine. Anthropometric variables were measured and body mass index calculated. All patients had echocardiographic assessment. Statistical analysis was performed using SPSS version 11.0 software. Multiple regression analysis was used in determining predictors of MA. A p-value of ≤ 0.05 was considered significant.

**Results:** Results of 136 patients comprising of 66(48.53%) males and 70(51.47%) females was considered. The overall prevalence of microalbuminuria was 42.65%. Males had a prevalence of 51.52% compared to 34.27% for the females (p=0.29). Weight, BMI, LVM, LVMI, UAE, and ACR were significantly higher in patients with MA, whereas those without MA had a significantly higher urinary creatinine. Multiple regression analysis identified DBP, MAP, LVM and LVMI as significant predictors of increased urinary albumin excretion Microalbuminuria showed significant positive correlation with LVM and LVMI.

**Conclusion:** The prevalence of microalbuminuria is high among untreated Nigerian hypertensive patients. The spot urine ACR provides a simple, accurate and cost effective way of identifying this high risk group of hypertensive patients, allowing for more aggressive treatment to reduce cardiovascular outcomes.

**Keywords:** Microalbuminuria; Albumin-to-creatinine ratio; Hypertension.

## INTRODUCTION

Hypertension is a leading cause of cardiovascular disease in developed and developing countries. It remains the most common non-communicable disease, with a rising prevalence in Nigeria.[1] The burden of cardiovascular disease imposed by hypertension is a result of target organ damage (TOD), notably the heart, kidneys and the brain.[2]

Indicators of early end-organ damage in hypertensive patients include increased left ventricular mass, increased carotid wall thickness and microalbuminuria (MA).[3,4] Microalbuminuria is defined as urinary albumin excretion rate of 20200 µg/min (valid for overnight urine collection) or 30300 mg/24 hr. Using early morning spot urine albumin to creatinine ratio, MA is defined as Albumin/creatinine ratio of 2.525 mg/mmol (Europe) or 30300 mg/g (USA).[5] Microalbuminuria is the first clinical expression of nephropathy and has become a cardiovascular and/or renal disease prognostic indicator for both diabetic and non-diabetic subjects. The first demonstration/report of MA in hypertensive patients without diabetes was from the work of Parvin et al, in 1974 where they showed increased urinary albumin excretion rate in untreated hypertensive patients compared to the effectively treated and the normal group.[6]

Several factors determine the prevalence of MA in a given population. These include among others, the threshold used to define and the method employed in the detection of microalbuminuria. The 24 hour urine albumin estimation by radioimmunoassay is the gold standard for the diagnosis of MA. However, this is of

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**Table 1**: General characteristics of patients according to their gender

| | Female (n=70) | Male (n=66) | *p* |
|---|---|---|---|
| Age (Years) | 44.31±9.06 | 45.36±11.91 | 0.56 |
| Height (cm) | 161.33(7.18) | 170.27±6.65 | 0.00* |
| Weight (kg) | 67.18(12.94) | 73.83±13.62 | 0.00* |
| BMI (kg/m$^2$) | 25.85(5.00) | 27.47±4.29 | 0.04* |
| SBP (mmHg) | 160.94(20.76) | 161.79±16.51 | 0.79 |
| DBP (mmHg) | 97.54±9.88 | 98.64±10.18 | 0.52 |
| PP (mmHg) | 87.44±13.25 | 62.88±15.12 | 0.00* |
| MAP (mmHg) | 118.30±11.83 | 119.35±10.03 | 0.57 |
| UAE (µmol/L) | 3050.00±264.71 | 3413.64±291.10 | 0.35 |
| UCreat (mg/dl) | 114.58±10.36 | 103.01±8.27 | 0.38 |
| ACR (mg/g) | 41.31±9.06 | 50.49±6.67 | 0.42 |
| LVM (g) | 165.03±65.18 | 197.97±63.21 | 0.00* |
| LVMI (g/m$^2$) | 95.01±34.87 | 106.26±31.82 | 0.05* |
| RWT | 0.52±0.24 | 0.54±0.16 | 0.57 |
| TC (mmol/L) | 4.62±1.02 | 4.63±1.02 | 0.95 |
| LDL (mmol/L) | 2.98±1.02 | 3.10±1.01 | 0.32 |
| HDL (mmol/L) | 1.05±0.29 | 1.06±0.32 | 0.99 |
| TG (mmol/L) | 1.57±0.59 | 1.37±0.46 | 0.03 |
| FBS (mmol/L) | 4.68±0.87 | 4.50±0.87 | 0.23 |

*=significant *p* value; BMI=body mass index; SBP=systolic blood pressure; DBP=diastolic blood pressure; PP=pulse pressure; MAP=mean arterial pressure; UAE=urinary albumin excretion; UCreat=urinary creatinine; ACR=Albumin-Creatinine Ratio; LVM=left ventricular mass; LVMI=left ventricular mass index; RWT=relative wall thickness; TC=total cholesterol; LDL=low density lipoprotein cholesterol; HDL=high density lipoprotein cholesterol; TG=triglyceride; FBS=fasting blood sugar.

flawed by the high rate of incomplete urine collection. To avert this, collection of a random, single voided, spot urine sample and subsequent determination of Albumin-Creatinine Ratio (ACR) for the assessment of MA is increasingly being used.[7] The National Kidney Foundation (NKF) working group defined MA as ACR greater than 30mg/g in both men and women.[8]

Microalbuminuria is an integrated marker of cardiovascular risk and has been positively and linearly related to the presence and severity of TOD. The evaluation of MA using the spot urine ACR is a sensitive and inexpensive way to identify hypertensive patients with a very high cardiovascular risk and a search for MA as part of the initial work-up of all hypertensive patients has been proposed and adopted by some guidelines.[9,10,11]

Despite the increasing prevalence of hypertension and its related morbidities in our environment, there is paucity of data on albumin excretion. We sought to establish the prevalence of MA among newly diagnosed hypertensive patients using the simple spot urine ACR.

## MATERIALS AND METHODS

A total of 186 newly diagnosed adult hypertensive patients were consecutively recruited from the cardiology clinic of the University of Maiduguri Teaching Hospital (UMTH) between June 2007 and February 2008. Routine investigations including serum electrolytes, urea, creatinine, fasting blood sugar, haematocrit, serum cholesterol and resting ECG were carried out at the general out-patient department (GOPD) before referral to cardiac clinic. Approval for the study was granted by the research and ethics committee of the UMTH, and informed consent obtained from the patients.

A complete medical history and physical examination were carried out on each patient. Following a five minute rest, blood pressure (BP) was measured with a mercury sphygmomanometer using standard protocols.[11] Hypertension was defined as an average BP of ≥140 mmHg systolic and /or ≥90 mmHg diastolic.[9] Height (in m) was measured to the nearest 0.1m with a stadiometre and weight (in kg) was measured using a calibrated bathroom weighing scale. Body mass index (BMI) in kg/m$^2$ was computed from height (m$^2$) and the weight (kg).

Echocardiographic examination was carried out on all the selected patients with a scanner 250 (PIE Medical, Japan). Left ventricular dimensions and thicknesses were measured at end-systole and end-diastole following American Society of Echocardiography (ASE) recommendations. [12] Left ventricular mass (LVM) was computed using the ASE formula, while LVMI was determined by dividing the LVM by BSA.[13] Relative wall thickness (RWT) was determined using the formula: RWT = 2xPWT / LVIDD. Left ventricular hypertrophy was defined as LVMI of >115g/m$^2$ in males and >95g/m$^2$ in females.

**Table 2**: Comparison of some characteristics of patients with and without microalbuminuria

| | MA Present (n=58) | MA Absent (n=78) | *p* |
|---|---|---|---|
| Age (Years) | 44.90±11.58 | 44.77±9.72 | 0.93 |
| Height (cm) | 165.67±8.56 | 165.68±8.04 | 0.99 |
| Weight (kg) | 73.20±14.13 | 68.33±12.96 | 0.04* |
| BMI (kg/m$^2$) | 26.72±5.06 | 24.89±4.21 | 0.02* |
| SBP (mmHg) | 162.97±17.70 | 160.15±19.53 | 0.39 |
| DBP (mmHg) | 97.41±11.91 | 98.56±8.36 | 0.51 |
| PP (mmHg) | 65.55±17.02 | 61.36±16.95 | 0.16 |
| MAP (mmHg) | 118.92±11.98 | 118.73±10.23 | 0.92 |
| LVM (g) | 209.67±68.84 | 159.71±55.43 | <0.001* |
| LVMI (g/m$^2$) | 115.41±36.48 | 89.36±26.87 | 0.00* |
| RWT | 0.54±0.18 | 0.53±0.22 | 0.78 |
| TC (mmol/L) | 4.66±1.10 | 4.60±0.96 | 0.74 |
| LDL (mmol/L) | 3.07±1.14 | 3.01±0.92 | 0.73 |
| HDL (mmol/L) | 1.04±0.33 | 1.07±0.28 | 0.57 |
| TG (mmol/L) | 1.42±0.54 | 1.51±0.54 | 0.34 |
| FBS (mmol/L) | 4.44±0.90 | 4.71±0.84 | 0.07 |
| UAE (µmol/L) | 4572.41±360.91 | 2225.64±124.22 | <0.001* |
| UCreat (mg/dl) | 70.20±5.23 | 137.79±9.78 | <0.001* |
| ACR (mg/g) | 82.21±7.19 | 18.66±0.93 | <0.001* |

*=significant *p* value; BMI=body mass index; SBP=systolic blood pressure; DBP=diastolic blood pressure; PP=pulse pressure; MAP=mean arterial pressure; UAE=urinary albumin excretion; UCreat=urinary creatinine; ACR=Albumin-creatinine Ratio; LVM=left ventricular mass; LVMI=left ventricular mass index; RWT=relative wall thickness; TC=total cholesterol; LDL=low density lipoprotein cholesterol; HDL=high density lipoprotein cholesterol; TG=triglyceride; FBS=fasting blood sugar.

Ten millilitres of early morning spot urine sample was collected from each patient on the day of presentation and kept frozen at the Chemical Pathology department of UMTH where they were subsequently analyzed. Urine creatinine concentration (µmol/L) was determined using the routine Jaffe reaction. Urine albumin concentration was determined by immunoturbimetric assay using mALB antibody reagent and assay buffer by RANDOX (MA-2426, RANDOX laboratories Ltd. United Kingdom). To obtain the spot urine albumin-creatinine ratio in mg/g, urine albumin concentration in mg/dl was divided by the urine creatinine concentration in g/dl. Microalbuminuria was defined as ACR of more than 30mg/g. None of the samples had an ACR of 300mg/g and had an ACR of 300mg/g and above. Fifty patients were excluded from the study as follows: elevated serum creatinine (15 patients), hyperglycaemia (5 patients), proteinuria on dipstick urinalysis (7 patients), failure to submit urine (4), heart failure (6 patients), poor image quality on echo (11) and refusal of consent (2). Active smokers as well as those with urinary tract infection were excluded.

Statistical analysis was performed using SPSS version 11.0 software (SPSS, Chicago, IL, USA). Continuous variables were expressed as mean ± standard deviation. Urinary albumin excretion, urinary creatinine and ACR were expressed as mean ± standard error of mean. Students't-test was used in making comparison between males and females. The relationship between MA and other variables was assessed using the Pearson's correlation coefficient.

**Table 3:** Multiple linear regression analysis showing predictors of UAE in the study population

| Variable | Standardized Beta | *p* |
|---|---|---|
| Age (Years) | -0.05 | 0.55 |
| BMI (kg/m$^2$) | 0.15 | 0.19 |
| SBP (mmHg) | -0.95 | 0.35 |
| DBP (mmHg) | 1.68 | 0.02* |
| PP (mmHg) | 1.48 | 0.10 |
| MAP (mmHg) | -1.38 | 0.01* |
| LVM (g) | -1.99 | 0.03* |

*=significant *p* value; UAE=Urine albumin excretionBMI=body mass index; SBP=systolic blood pressure; DBP=diastolic blood pressure; PP=pulse pressure; MAP=mean arterial pressure; LVM=left ventricular mass; LVMI=left ventricular mass index.

Table 4: Correlation analysis showing the relationship of ACR and some variables

| Variable | *r* | *p* |
|---|---|---|
| Age (Years) | -0.12 | 0.15 |
| BMI (kg/m$^2$) | 0.05 | 0.55 |
| SBP (mmHg) | 0.03 | 0.72 |
| DBP (mmHg) | -0.01 | 0.96 |
| PP (mmHg) | 0.04 | 0.63 |
| LVM (g) | 0.36 | 0.00* |
| LVMI (g/m$^2$) | 0.41 | 0.00* |

*=significant *p* value; ACR=Albumin-Creatinine Ratio; BMI=body mass index; SBP=systolic blood pressure; DBP=diastolic blood pressure; PP=pulse pressure; LVM=left ventricular mass; LVMI=left ventricular mass index.

Multiple regression analysis was used in determining predictors of MA. A p-value of ≤ 0.05 was considered significant.

**RESULTS**

Results of 136 patients comprising of 66 (48.53%) males and 70 (51.47%) females was considered. The general characteristic of the patients is illustrated in table 1.

Height, weight, BMI, LVM and LVMI were significantly higher in males whereas the females had a significantly higher pulse pressure (PP) and triglyceride (TG). Other parameters did not show significant gender variation.

Stage I and stage II hypertension was observed in 43.38% and 55.15% of the patients respectively. The prevalence of combined systolic and diastolic hypertension was 89.70%. The prevalence of obesity was 17.67%, while overweight was observed in 35.29%. Overweight was observed in 41.43% of the females and 28.79% of the male patients (p=0.17). Obesity was observed in 21.21% of males and 14.29% of the female patients (p=0.40).

The overall prevalence of microalbuminuria was 42.65%. Males had a prevalence of 51.52% compared to 34.27% for the females (p=0.29). Although the prevalence was higher in stage I compared to stage II hypertension, and in obesity compared to overweight, the difference is not significant (p=0.19 and p=0.15 respectively).

Table 2 compares parameters in patients with and without MA. Weight, BMI, LVM, LVMI, UAE, and ACR were significantly higher in patients with MA, whereas those without MA had a significantly higher urinary creatinine.

Multiple regression analysis identified DBP, MAP, LVM and LVMI as significant predictors of increased urinary albumin excretion (table 3). Microalbuminuria showed significant positive correlation with LVM and LVMI (table 4).

**DISCUSSION**

To our knowledge, this is the first study to assess MA using single spot urine albumin-creatinine ratio in untreated Nigerian hypertensive patients.

The observed overall prevalence of 42.65% for MA is higher than the prevalence of 36% and 37% reported by Odili and Okeahialam;[14] and Salako et al[15] respectively. However, it is lower than the findings of Alebiosu et al among hypertensive patients with non-dipping nocturnal blood pressure in Sagamu.[16] It is also at variance with the prevalence of 24.50% reported by Rayner and Becker in non-diabetic South African hypertensive patients.[17] This reflects the general inconsistencies in the prevalence of MA in hypertensive patients.[18,19]

The first documentation of increased UAE in hypertensive patients was by Parving et al, who showed an increased UAE among insufficiently treated hypertensive.[6] This finding generated a lot of interest in MA especially among non-diabetic hypertensive patients, and has been amply confirmed by various workers. [20,21]

Microalbuminuria has been identified to have significant implication for morbidity and mortality among hypertensive patients. In one of the largest longitudinal studies on the predictive role of MA for CVD, the MONICA study showed hypertensive subjects with MA to have almost four-fold increase in the risk of coronary heart disease.[22]In another study involving 11363 non-diabetic hypertensive patients, those with MA had a significantly higher prevalence of stroke, LVH, coronary artery disease, and peripheral vascular disease.[23]

The clustering of MA with obesity, LVH, increased BP as well as other metabolic risk factors as observed in the study has been previously reported.[24] Among a group studied in Finland, MA showed the strongest relationship with mortality compared to all other risk factors of the metabolic syndrome.[25] Similarly, the prevalence of MA increased linearly as a function of the number of metabolic syndrome risk factors in cohorts evaluated in the third National Health and Nutrition Examination Survey (NHANES III).[26]

Microalbuminuria correlated strongly and positively with LVM and LVMI, a finding in keeping with by

reports of previous workers.[21,27]Increased UAE has been associated with increased prevalence of hypertensive target organ damage including increased LVM, hypertensive retinopathy, increased carotid intimal thickness and plaque burden.[28] In the MAGIC study, patients with elevated albumin excretion rate showed a significantly higher prevalence of electrocardiographic changes compatible with LVH and/or ischaemia.[21] In a study by Tsioufis et al., hypertensive subjects with MA exhibited higher incidence of unfavourable LV geometric patterns compared to those without.[29] They also documented that absence of MA is associated with normal LV geometric pattern whereas MA is associated with concentric LVH. In the LIFE study, the frequency and degree of MA were greater in patients with greater LVM.[30]This correlation was independent of SBP, age, race or co-existing diabetes. Increased urine ACR resulted in increasing risk for cardiovascular morbidity and mortality among hypertensive patients with LVH.

Multiple regression analysis identified DBP, MAP, LVM and LVMI as significant predictors of MA. Busari et al;[31] reported DBP to be significantly higher in hypertensive patients with MA compared to normoalbuminuric patients, a finding corroborated by Seiegl et al.[32] However, most of the studies on predictors of MA in hypertensive patients implicated SBP and PP as the most consistent determinants of MA.[33,34] Similarly, Akinsola et al demonstrated a significant correlation between MA and SBP, but not DBP.[35] These findings reflect the general inconsistencies regarding the correlates of MA in hypertensive patients.

## CONCLUSIONS

The prevalence of MA using the spot urine albumin to creatinine ratio is high among untreated Nigerian hypertensive patients. This identifies a subset of hypertensive patients at increased risk of future cardiovascular morbidity and mortality. The early morning spot urine ACR provides a simple, accurate and cost effective way of identifying this high risk group of hypertensive patients, allowing for more aggressive treatment to reduce cardiovascular outcomes.

## LIMITATIONS

This study is not without limitations. Lack of apparently healthy, normotensive control group is a major limitation. In addition, urinary albumin excretion was assessed using a single urine sample rather than multiple samples. The inherent poor reproducibility/variability of albumin excretion may impact on the results.

## ACKNOWLEDGMENT

We thank Mrs Rhoda Yathba for assisting with biochemical analysis of the samples. A great deal of appreciation is due to Prof. Dann Nel of the Stellenbosch University for his advice on statistical analysis.

**CONFLICT OF INTEREST**- None

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