International Journal of Advances in Medicine
*Oresegun OO et al. Int J Adv Med. 2020 Jul;7(7):1141-1149*
http://www.ijmedicine.com
pISSN 2349-3925 | eISSN 2349-3933
DOI: http://dx.doi.org/10.18203/2349-3933.ijam20202591

**Original Research Article**

# Correlation between echocardiographic left ventricular hypertrophy and microalbuminuria among hypertensive subjects in Olabisi Onabanjo university teaching hospital, Sagamu, Nigeria

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**Received:** 18 May 2020
**Accepted:** 10 June 2020

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## ABSTRACT

**Background:** Left ventricular hypertrophy is an adaptive response to volume and/or pressure overload, especially in hypertensive subjects. Microalbuminuria is a well-known predictor of poor cardiovascular outcomes in patients with hypertension. The purpose of this study was to determine the prevalence of microalbuminuria in hypertensive subjects and its relationship with LVH defined by echocardiogram in adult hypertensive subjects.
**Methods:** The study was a cross-sectional comparative one involving 88 hypertensive volunteers with age and sex-matched normotensive controls. Detailed history, physical examination, urine assessment for microalbuminuria and echocardiogram were carried out on all participants.
**Results:** The overall prevalence rate of microalbuminuria in the study was 29.5%. The prevalence of microalbuminuria in hypertensive subjects (43.2%) was significantly higher than in the normotensive counterparts (15.9%) (p=0.001). The prevalence of microalbuminuria in hypertensive subjects with echocardiographic LVH (81.0%) was significantly higher than in hypertensive participants without echocardiographic LVH (31.3%) (p=0.001). There was a positive correlation between left ventricular mass defined by echocardiogram and urinary albumin excretion in hypertensive subjects (r =0.422, p= 0.001).
**Conclusions:** There is a high prevalence of microalbuminuria among hypertensive adults, especially those with left ventricular hypertrophy. There is also a positive correlation between echocardiographic left ventricular mass and urinary albumin excretion among adult hypertensive subjects.

**Keywords:** Echocardiogram, Hypertension, Left ventricular hypertrophy, Microalbuminuria

## INTRODUCTION

Cardiovascular disease (CVD) is the leading cause of death globally, accounting for about 30% of deaths annually.[1] About half of CVD-related deaths are attributable to hypertension, a disease strongly associated with the overall cardiovascular risk.[1] Left ventricular hypertrophy (LVH) is an adaptive response and the hallmark of increased volume and/or pressure overload in exercise training (physiological) or in hypertension, cardiomyopathy, or valvular heart disease (pathological) and it serves to normalize ventricular wall stress, myocardial fibre shortening, and stroke volume.[2] It results from a complex interaction of several

hemodynamic (hypertension, renin-angiotensin-aldosterone system, and insulin resistance), non-hemodynamic (gender and ethnicity), and environmental factors, such as salt intake, obesity, and diabetes mellitus.[2] Although it involves a process of adaptive remodeling, which is usually a compensatory mechanism in response to increased hemodynamic load, left ventricular hypertrophy is ultimately characterized by structural changes, mainly in the form of myocardial fibrosis that lead to diastolic dysfunction and diminished contractility.[3]

Left ventricular hypertrophy (LVH) is a strong predictor of adverse cardiovascular outcomes and an important risk factor for sudden death and heart failure.[3] Morphologically, LVH may be characterized by increased wall thickness (concentric LVH), increased chamber volume (eccentric LVH) or both.

Microalbuminuria (MA) is the excretion of 30-300mg of albumin per 24hours or 30-300mcg/mg creatinine in two of three urine collections.[4] It is a well-known predictor of poor cardiovascular and renal outcome in patients with essential hypertension and type 2 diabetes mellitus.[5,6] Microalbuminuria, as a marker of early renal damage, reflects a pro-inflammatory state with high levels of IL-6, C-reactive protein, and fibrinogen and ensuing endothelial dysfunction.[7] In addition to hypertension, microalbuminuria can also result from other factors like advancing age, dyslipidemia(hypertriglyceridemia), male gender, hormone replacement therapy, diabetes mellitus, heart disease, stroke, smoking cigarette, drugs especially NSAIDS.[8]

Left ventricular hypertrophy and microalbuminuria have both been shown to predict increased cardiovascular morbidity.[9]

Multiple studies conducted in Europe, Africa, Asia on hypertensive ,non-diabetic participants documented a high prevalence of microalbuminuria(ranging between 32.3% and 39.5%), with even higher values noted in hypertensive subjects with LVH.[5-7,9,10] For instance, a study done in South-West Nigeria documented the prevalence of microalbuminuria of 32.3% in 96 newly diagnosed hypertensive subjects and a higher incidence of microalbuminuria among hypertensive participants with LVH.[7] These studies utilized either semi-quantitative methods for assessing microalbuminuria or electrocardiogram in assessing left ventricular hypertrophy, placing a limitation on the reproducibility of such results. Furthermore, there are limited studies on the relationship between these markers of cardiac and renal glomerular damage in a large hypertensive population in Nigeria with target organ damage, using echocardiography in South-West, Nigeria.

The aim of this study was to assess the prevalence of microalbuminuria (using quantitative assays) and its correlation with echocardiographic LVH in known hypertensive adults in a tertiary hospital in South-West Nigeria.

## METHODS

The study was carried out at the Olabisi Onabanjo University Teaching Hospital (OOUTH), Sagamu, Ogun State, South-West Nigeria.

This was a cross-sectional comparative hospital-based study. Eighty-eight hypertensive patients were consecutively recruited in the Cardiology clinic irrespective of their duration of hypertension, blood pressure control and whether on medications or not. Eighty-eight age- and sex-matched normotensive controls were also recruited from the same centre.

Subjects were participants aged 18years and above who are hypertensive and who gave informed consent. Controls were participants aged 18years and above, apparently healthy individuals including hospital staff and relatives of participants who have normal blood pressure and gave informed consent. They were matched by age and sex.

Individuals who were obese, pregnant, had kidney disease, heart failure, liver disease, malignancy, on medications that could cause false positive proteinuria (like NSAIDS, penicillamine, lithium carbonate) or had overt proteinuria (from dip-sticks screening) were excluded from the study.

### *Ethical consideration*

Approval for the study was obtained from the Health Research and Ethics Committee of OOUTH. Written informed consent was obtained from each participant. All relevant standards of Revised Declaration of Helsinki were followed.

### *Sampling design*

The sample size was calculated using the Cochran formula $N= Z2pq/d^2$. With a prevalence of 5.4%, this resulted in a sample size of 78.50, approximated to 88 for each study group to accommodate for 10% attrition.[11]

### *Data collection*

Detailed history including socio-demographic data, anthropometric indices, personal and family history of hypertension, diabetes and other diseases, medication history were obtained.

The Blood Pressure (BP) of all participants was measured after five minutes rest each to eliminate anxiety. There was no consumption of stimulants (coffee, cigarette smoking) before BP measurement. The blood pressure was taken with the participant seated comfortably upright with the feet on the floor and arm at the level of the heart,

free of any constrictive clothing or lying supine using the standard mercury sphygmomanometer (Accoson®) and appropriate-sized cuff. The BP of participants was taken in both arms, with five minutes between readings and the average was used.

The body weight of all subjects was determined in kilograms while the height of the participants was measured in meters using a stadiometer.

The Body mass index (BMI) of all participants was calculated as Weight (kg)/Height ($m^2$).

Laboratory assessment included collection of 3mls of venous blood taken from the ante-cubital vein via venipuncture into fluoride oxalate, Lithium heparin and plain specimen bottles after routine aseptic preparation.

***Echocardiogram***

Echocardiographic studies were done using the commercially available echo-machine (ALOKA SSD 900) with a 3.5MHz linear array transducer probe.

***Urine assay for microalbuminuria***

Participants with overt proteinuria (>+1) were excluded with a urinary dip stick test.

Early morning (before exercise or activity) spot urine samples (three) from the eligible participants were collected during three consecutive Clinic visits and tested for microalbuminuria using the HemoCue Albumin 201 Analyzer machine. The mean value of the microalbuminuria was recorded for each participant.

Left Ventricular Hypertrophy was defined as LVM greater than 51g/$m^{2.7}$ in both men and women.[12] Relative wall thickness was calculated as twice the posterior wall thickness/LV Internal dimension in diastole. Relative wall thickness of 0.44 or greater was considered abnormal.[13]

***Statistical analysis***

The data were analyzed using the statistical package for social sciences (SPSS) version 20 software (SPSS, Inc, Chicago Illinois, USA).

Continuous variables were expressed as mean±SD (standard deviation) and categorical variables as percentages. Differences in categorical variables among the two (2) groups were assessed by Chi-square analysis while unpaired t test was used for comparison of continuous variables. Comparison of normally distributed continuous variables among three (3) groups was performed by analysis of variance (ANOVA). Correlation between two (2) normally distributed variables was done with Pearson's correlation coefficient while Spearman's rank correlation was used for non-normally distributed variables.

Spearman's correlation analysis was used to assess variables with significant correlation with Left Ventricular Mass. A p-value above 0.05 was generally considered to be statistically insignificant. For this study, a p-value <0.05 was considered significant.

## RESULTS

***Demographic and clinical parameters of the study***

Eighty-eight (88) hypertensive subjects and the same number of age- and sex-matched normotensive controls were studied. The mean age of the hypertensive participants (subjects) was 53.7±8.5 years which did not differ significantly from that of controls at 53.8±8.0 years (p=0.899). The subjects had significantly higher body mass index (BMI) as depicted in Table 1.

The mean systolic and mean diastolic blood pressures of the hypertensive subjects were significantly higher than that of the normotensive controls (p= 0.001 respectively). Other sociodemographic characteristics of the study population are shown in Table 1.

**Table 1: Sociodemographic and clinical characteristics of all study participants.**

| Variables (Mean±SD) | Subject (%) N=88 | Control (%) N=88 | Test | Statistical test value | p |
|---|---|---|---|---|---|
| Age (years) | 53.7±8.5 | 53.8±8.0 | t | -0.127 | 0.899 |
| BMI (Kg/$m^2$) | 26.6±2.9 | 25.1±3.0 | t | 3.190 | 0.002* |
| SBP (mmHg) | 146.5±18.3 | 121.1±11.7 | t | 10.969 | 0.001* |
| DBP(mmHg) | 94.8±13.0 | 75.8±9.6 | t | 11.003 | 0.001* |
| Gender(Male) | 44 (50.0) | 44 (50.0) | $X^2$ | 0.000 | 1.000 |
| Microalbuminuria | 38 (43.2) | 14 (15.9) | $X^2$ | 14.439 | 0.001* |
| LVH | 15 (17.0) | 4 (4.5) | $X^2$ | 5.900 | 0.015* |

Key: BMI - Body Mass Index, SBP - Systolic Blood Pressure, DBP - Diastolic Blood Pressure, LVH-Left ventricular Hypertrophy, X2 - Chi Square, t - Student t test, * - Statistically Significant.

***Prevalence of microalbuminuria in the study***

Out of a total of 176 study participants, 52 had microalbuminuria giving a prevalence of 29.5%. The prevalence of microalbuminuria among the hypertensive subjects was 43.2% (38/88) compared with 15.9% (14/88) among normotensive controls as shown in Table 1. The difference was statistically significant (p=0.001).

Furthermore, the prevalence rate of microalbuminuria was significantly higher among hypertensive subjects with echocardiography-defined left ventricular hypertrophy compared with hypertensive subjects without echocardiography-defined left ventricular hypertrophy (81.0% vs 31.3%; p= 0.001) as shown in Table 2.

***Gender comparison of microalbuminuria in all study participants***

Although the prevalence rate of microalbuminuria was higher in the female hypertensive subjects when compared with their male counterparts in this study (47.7% versus 38.6%), it lacked statistical significance (p= 0.389).

Furthermore, there was no statistically significant difference in the prevalence rate of microalbuminuria when the female normotensive controls were compared with their male counterparts (15.9% respectively) (p= 1.000).

***Echocardiographic left ventricular geometry pattern among the study participants***

The pattern of left ventricular geometry among all the study participants as determined by echocardiography included: Normal left ventricular geometry- 63.6% (112/176), Left ventricular concentric remodeling- 22.2% (39/176), Eccentric left ventricular hypertrophy- 7.4% (13/176), Concentric Left ventricular hypertrophy- 6.8% (12/176).

However, left ventricular geometry pattern among hypertensive subjects as determined by echocardiography was as follows: Normal left ventricular geometry 47.7% (42/88), Left ventricular concentric remodeling- 28.8% (25/88), Concentric left ventricular hypertrophy- 12.5% (11/88), Eccentric Left ventricular hypertrophy- 11.4% (10/88).

The pattern of left ventricular geometry among normotensive controls was as follows: Normal left ventricular geometry-79.5% (70/88), Left ventricular concentric remodeling-15.9% (14/88), Eccentric left ventricular hypertrophy-3.4% (3/88), Concentric left ventricular hypertrophy-1.1% (1/88).

**Table 2: Sociodemographic, clinical and laboratory comparison between hypertensive subjects with and without LVH.**

| VARIABLES (Mean± SD) | | HTN+LVH (%) N=21 | HTN-LVH (%) N=67 | Test | Statistical test value | p |
|---|---|---|---|---|---|---|
| Age (years) | | 55.5±8.5 | 52.6±8.2 | t | -1.373 | 0.173 |
| Gender | Male | 12 (57.1) | 32 (47.8) | $X^2$ | 0.250 | 0.617 |
| | Female | 9 (42.9) | 35(52.2) | | | |
| BMI (Kg/m$^2$) | | 26.8±2.8 | 26.5±2.9 | t | -0.422 | 0.674 |
| SBP (mmHg) | | 150.5±18.3 | 145.3±18.2 | t | -1.141 | 0.257 |
| DBP(mmHg) | | 97.6±13.7 | 93.9±12.8 | t | -1.144 | 0.256 |
| Microalbuminuria | | 17 (81.0) | 21 (31.3) | $X^2$ | 18.642 | 0.001* |

KEY: HTN+LVH- Hypertension with LVH, HTN-LVH- Hypertension without LVH, SBP-Systolic Blood Pressure, DBP- Diastolic Blood Pressure, t- Student t test, X2 - Chi Square, df - degree of freedom, *- Statistically significant

**Table 3: Prevalence of microalbuminuria among the various left ventricular geometric patterns in hypertensive subjects and normotensive controls.**

| Group | VARIABLES | Normal (%) | Con LVH (%) | Ecc LVH (%) | CR (%) | $X^2$ | df | p |
|---|---|---|---|---|---|---|---|---|
| Subjects | Normal | 32 (76.2) | 2 (18.2) | 2 (20.0) | 14 (56.0) | 18.649 | 3 | 0.001* |
| | Microalbuminuria | 10 (23.8) | 9 (81.8) | 8 (80.0) | 11 (44.0) | | | |
| Controls | Normal | 58 (82.9) | 1 (100.0) | 2 (66.7) | 13 (92.9) | 1.952 | 3 | 0.582 |
| | Microalbuminuria | 12 (17.1) | 0 (0.0) | 1 (33.3) | 1 (7.1) | | | |

KEY- LVH - Left Ventricular Hypertrophy, CR - Concentric Remodeling, Con LVH - Concentric Left Ventricular Hypertrophy, Ecc LVH - Eccentric Left Ventricular Hypertrophy, CR - Concentric Remodeling, df - degree of freedom, X2 - Chi- square, *- statistically significant

**Table 4: Relationship between echocardiographic LVH, arterial blood pressure and microalbuminuria among hypertensive subjects.**

| Variables | | N=88n N=88 | Mean UAE (ng/L) | SD | Statistical test value | df | p |
|---|---|---|---|---|---|---|---|
| SBP (mmHg) | Normal | 44 | 22.06 | 21.44 | F=3.422 | 2 | 0.037* |
| | Stage 1 | 18 | 29.17 | 26.38 | | | |
| | Stage 2 | 26 | 49.12 | 68.84 | | | |
| DBP (mmHg) | Normal | 48 | 19.00 | 18.75 | F=29.746 | 2 | 0.001* |
| | Stage 1 | 30 | 26.07 | 24.78 | | | |
| | Stage 2 | 10 | 107.90 | 82.37 | | | |
| LVM/ht$^{2.7}$ | | 88 | 38.98 | 10.95 | r=0.422 | 2 | 0.001* |
| MA(ng/L) | | 88 | 31.51 | 43.17 | | | |

SBP - Systolic Blood Pressure, DBP - Diastolic Blood Pressure, MA-Microalbuminuria, SD - Standard Deviation, UAE- Urinary Albumin Excretion, df - Degree of Freedom, r- correlation co-efficient, *- statistically significant

***Prevalence of microalbuminuria among the various left ventricular geometry patterns in hypertensive subjects and normotensive controls***

The prevalence of microalbuminuria among the different left ventricular geometric patterns in hypertensive subjects as defined by echocardiography included: Normal LV geometry- 23.8%, Concentric left ventricular hypertrophy- 81.8%, Eccentric left ventricular hypertrophy- 80.0%, LV concentric remodeling- 44.0%. The difference in prevalence in microalbuminuria among the various left ventricular geometric patterns in hypertensive subjects was statistically significant (p= 0.001) as shown in Table 3.

The prevalence of microalbuminuria among the different left ventricular geometric patterns in normotensive controls as defined by echocardiography included: Normal LV geometry- 17.1%, Concentric left ventricular hypertrophy- 0.0%, Eccentric left ventricular hypertrophy- 33.3%, LV concentric remodeling-7.1%. The difference in prevalence in microalbuminuria among the various left ventricular geometric patterns in the controls lacked statistical significance (p= 0.582) as shown in Table 3.

***Relationship between microalbuminuria and left ventricular mass (defined by echocardiogram) among hypertensive subjects***

There was a positive correlation between microalbuminuria and left ventricular mass among hypertensive subjects in this study with a correlation co-efficient (r) of 0.422 (p=0.001), as depicted in Table 4.

***Relationship between arterial blood pressure and microalbuminuria among hypertensive subjects***

The relationship between grades of systolic blood pressure, diastolic blood pressure, and the mean urinary albumin excretion in hypertensive subjects was statistically significant (p= 0.037, p=0.001 respectively) as shown in Table 4.

## DISCUSSION

To the best of our knowledge, this study represents the first report on microalbuminuria (quantitatively determined) in adult hypertensive subjects in South-West Nigeria. The mean age of study participants was similar to that observed in previous studies.[7,14]

Data from this study revealed that hypertensive subjects had significantly higher body mass index (BMI) than normotensive controls. This was in agreement with prior studies.[15,16] Obesity-associated hypertension is usually due to inadequate vasodilatation in the presence of increased blood volume and cardiac output, which are natural consequences of an increased mass.[15] Furthermore, increased sympathetic nervous system activation has been implicated as causal factor in obesity-associated hypertension.[16]

In this study, the overall prevalence of microalbuminuria was 29.5%. In all the hypertensive subjects, the prevalence of microalbuminuria was 43.2%. This prevalence translates to two in every five adult (without overt proteinuria) with hypertension are likely to have microalbuminuria.

This high prevalence was similar to that documented in Abuja and Enugu, Nigeria Uganda and in Portugal.[6,14,5,17] Other researchers have, however, reported lower prevalence rates.[7,18,19] This variability in the prevalence rates may be due to factors such as patient selection procedures, prior antihypertensive treatment or naivety, prior or current use of some anti-hypertensive medications protective against microalbuminuria, duration of hypertension and differing methods of assessing microalbuminuria. For instance, the duration of systemic hypertension was not taken into consideration in this study.

The prevalence of microalbuminuria in normotensive controls in this study was 15.9%. This compared favorably with other studies.[20,21] A study from South-Eastern Nigeria, however, documented lower prevalence rates.[22] This variability may be attributed to patient-dependent characteristics and methods of estimating microalbuminuria. For instance, an author in Ido-Ekiti, South-West Nigeria used Micra test strips (semi-quantitative) for microalbuminuria assessment while another worker in Port-Harcourt, South-South Nigeria, utilized the immunoturbidometry (antibody-based) method for estimating microalbuminuria.[7,20] This could explain the similitude in the prevalence rate of microalbuminuria in the control population of this study in comparison with the latter study as the same (immunoturbidometry) method was utilized.

Microalbuminuria in normotensive individuals may be attributed to factors such as older age, male gender, dyslipidaemia (especially hypertriglyceridaemia), increased body mass index, increased salt intake, and use of inhibitors of the renin angiotensin aldosterone system.[23]

Data from this study also corroborated reports of similar studies which documented a high prevalence of microalbuminuria in hypertensive subjects with LVH.[24,25] This study found the prevalence of microalbuminuria in hypertensive subjects with LVH to be 81% while the prevalence in hypertensive subjects without LVH was 31.34%. Studies in other parts of southern Nigeria also documented similar prevalence rates.[7,18] It was concluded that microalbuminuria was highly prevalent among hypertensive subjects, especially in those with left ventricular hypertrophy. The high prevalence of microalbuminuria in this category of hypertensive subjects underscores the subtle changes occurring in the renal glomeruli of these patients. Thus, with increasing albumin excretion, renal damage and other target organ damages may worsen with the progression of left ventricular hypertrophy.

Furthermore, microalbuminuria was not only associated with LVH, but also correlated with a five-fold greater risk of inappropriate LVH and global myocardial performance impairment.[26] Albuminuria and LVH may be signs of target organ damage and reflect the severity and complication of hypertension or the result of a common underlying pathologic cause.[27] Common pathophysiological denominators include the increased hemodynamic load, sympathetic and renin-angiotensin system over-activity, increased subclinical inflammatory reaction, and the production of reactive oxygen species, that in combination or separately, induce fibrotic phenomena at the heart and the kidney.[28,29]

Concerning gender differences in urinary albumin excretion, study reports have documented divergent views. While some studies opined a significantly higher prevalence of microalbuminuria in female hypertensive subjects when compared to their male counterparts, the reverse was found in some other population.[20,30,31] Although, the prevalence of microalbuminuria in our study was higher in female hypertensive subjects, the difference was not statistically significant. The higher prevalence rate of microalbuminuria in females might be related to lower muscle mass (approximately 15% less), hence lower creatinuria in women. To this end, some authors have suggested the use of sex-related cut-off points to define microalbuminuria.[32]

Various left ventricular geometry patterns have been documented in hypertensive subjects using echocardiography. In this study, normal LV geometry (47.7%) was found to be the most prevalent pattern in hypertensive subjects, followed by LV concentric remodeling (28.4%). Eccentric hypertrophy was the least common geometric pattern in hypertensive subjects in this study (11.4%). This was at variance with similar studies carried out in Ile-Ife, South-West Nigeria which posited that LV concentric remodeling was the commonest LV geometric pattern among treated hypertensive subjects followed by normal left ventricular geometry.[33-35] The authors also concluded that eccentric hypertrophy was the least prevalent LV geometry pattern in hypertensive subjects in Nigeria, a position that was in agreement with this study.

The different predominant LV geometry distribution pattern obtained in this study may be attributed to the fact that majority of the hypertensive subjects in this study were already on anti-hypertensive medications which could have induced some reversal or at least stopped the progression of maladaptive left ventricular changes associated with increasing pressure or volume overload seen in systemic hypertension. Furthermore, this study did not consider the duration of hypertension in the subjects or the class (es) of anti-hypertensive medications used. In addition, a different echocardiographic criterion was employed in the definition of left ventricular hypertrophy in this study. In the above-cited studies, left ventricular mass indexed to body surface area with values greater than 134g/$m^2$ (for male) and 110g/$m^2$ (for female) used as cut-off point for left ventricular hypertrophy. However, in this study, the left ventricular mass was indexed to the allometric height of 2.7, with left ventricular hypertrophy defined as values greater than 51g/m 2.7 for both genders. This latter criterion has been documented to be more sensitive in Nigerians.[36]

Nevertheless, some authors have documented concentric left ventricular hypertrophy as the dominant geometry pattern in hypertensive subjects in a study carried out Abuja, North Central Nigeria while some others reported eccentric left ventricular hypertrophy as the predominant LV geometry pattern in a study conducted in Kano, North-Western, Nigeria.[37,38]

This study further revealed that approximately one in every three adult hypertensive subjects had concentrically

remodeled left ventricle on echocardiography. LV concentric remodeling geometry is not a benign adaptive process but rather an independent predictor of cardiovascular events and portends worse prognosis in hypertensive subjects. It is associated with increased structural and functional alterations in both great and small vessels and also with metabolic abnormalities like insulin resistance.[39]

The findings from this study also revealed that there was a significant (positive) correlation between echocardiographic left ventricular mass and urine albumin excretion rate among hypertensive subjects. This is consistent with findings in other studies which demonstrated that microalbuminuria is independently linked with risk for cardiovascular disease and complications in hypertensive patients.[5-7] This finding further reiterates the need for routine assessment for microalbuminuria in both newly diagnosed hypertensive subjects and those already on antihypertensive therapies to facilitate early initiation of add-on or reversal medications such as calcium-channel blockers (CCBs) and inhibitors of the renin angiotensin aldosterone system.

Besides old age, diabetes mellitus and smoking, systemic hypertension is a well-recognized risk factor for microalbuminuria. However, studies have reported divergent views on their relationship. While a study reported no significant association between systolic blood pressure, diastolic blood pressure and microalbuminuria among hypertensive subjects and another reported a significant relationship only between diastolic blood pressure and microalbuminuria in hypertensive subjects, some other authors documented significant relationship between both the systolic and diastolic blood pressures and microalbuminuria in hypertensive subjects.[18-21] Findings from this study were consistent with the latest group. In addition, this study further revealed that the higher the arterial blood pressure values, the higher the urinary albumin excretion.

Quantitative microalbuminuria estimation, which is relatively simple to carry out, cheap and readily available, may be used as a surrogate marker to alert physicians to the presence of target organ damages (left ventricular hypertrophy for instance) and the need to initiate or add-on reversal therapies. This measure may avert avoidable morbidity and mortality. Furthermore, as urine testing for microalbuminuria is sensitive and can be repeated frequently, it is an ideal marker for preventive strategies in a resource-poor environment such as ours.

This study has some limitations, the duration of hypertension was from self-reported history which may not be accurate, as parameters measured in the study are affected by both duration and degree of high blood pressure. Although an average of three measurements of urinary albumin excretion was taken, there was no correction for potential variability in urine concentration. A larger sample size may have added more power to the study. The type of antihypertensive medications taken by the participants was not taken into consideration as some could influence urine albumin excretion and left ventricular mass.

In conclusion, the prevalence of microalbuminuria in adult hypertensive subjects in OOUTH, Sagamu was high (43.2%), while the overall prevalence of microalbuminuria in the study was 29.5%. The prevalence of microalbuminuria in hypertensive subjects with LVH was 81% which was significantly higher than the prevalence of microalbuminuria in hypertensive subjects without LVH (23.8%) and normotensive controls (15.9%).

There was a positive correlation between microalbuminuria and left ventricular mass in adult hypertensive subjects in OOUTH, Sagamu.

## ACKNOWLEDGEMENTS

Authors would like to thank Dr. Tinuade Ogunlesi for his contributions to the success of this work.

*Funding: No funding sources*
*Conflict of interest: None declared*
*Ethical approval: The study was approved by the Institutional Ethics Committee of OOUTH*

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**Cite this article as:** Oresegun OO, Familoni OB, Olunuga TO, Akinpelu A. Correlation between echocardiographic left ventricular hypertrophy and microalbuminuria among hypertensive subjects in Olabisi Onabanjo university teaching hospital, Sagamu, Nigeria. Int J Adv Med 2020;7:1141-9.