Renal risk profiling in newly diagnosed hypertensives in an urban population in Nigeria

Aderoju Gbadegesin¹, Oluyomi Okunola², Olugbenga Ayodele¹, Fatiu Arogundade², Abubakre Sanusi², Adewale Akinsola²

1. LadokeAkintola University Teaching Hospital, Osogbo, Nigeria.

2. Obafemi Awolowo University Teaching Hospital, Ile-Ife, Nigeria.

Abstract

Introduction: Hypertension is a cause and consequence of chronic kidney disease globally. The other factors that work in concert with hypertension to cause CKD are yet to be clearly elucidated. Studies have identified proteinuria, dyslipidemia, obesity, smoking and family history of CKD as renal risk factors. Due to the high morbidity and mortality associated with occurrence of CKD including the enormous financial burden involved in its management, the knowledge of prevention and understanding of the risk factors for development of CKD is highly essential. Therefore, Identifying well defined risk factors that display strong graded association with the occurrence and progression of CKD can help in elucidating potential targets for disease modification.

Objective: The aim of this study was to determine the prevalence of renal risk factors and their impact on kidney function in newly diagnosed hypertensive Nigerians.

Methods: This was a case control study of two hundred and fifty newly diagnosed hypertensive Nigerians recruited from two contiguous hospitals in an urban setting in south western Nigeria. Another group of two hundred and fifty apparently healthy age and sex matched normotensive Nigerians in the same community were recruited as controls.

Results: Seventy (28%) of the newly diagnosed hypertensives had estimated glomerular filtration rate of less than 60ml/min, while 42.4% and 18.8% of the subjects and the controls had microalbuminuria respectively. The newly diagnosed hypertensives had significantly higher prevalence of analgesic use (86.4% versus 41.6%, p < 0.001), alcohol consumption (20.8% versus 12%, p = 0.008), use of canned salted food (18.8% versus 8.4%, p = 0.001) and central obesity (36.1% versus 26.8%, p = 0.025) compared to controls.

Conclusion: There is a significant occurrence of modifiable renal risk factors in newly diagnosed hypertensives and this offers a platform for instituting preventive strategies in the community.

Keywords: Renal risk, hypertensives, urban population, Nigeria.

DOI: https://dx.doi.org/10.4314/ahs.v19i4.8

Cite as: Gbadegesin A, Okunola O, Ayodele O, Arogundade F, Sanusi A, Akinsola A. Renal risk profiling in newly diagnosed hypertensives in an urban population in Nigeria. Afri Health Sci. 2019;19(4):2863-2873. https://dx.doi.org/10.4314/ahs.v19i4.8

Introduction

Hypertension is an important and a major global public health challenge with high prevalence. It affects more people in developing countries and it is projected that by 2025, almost three- quarters of people with hypertension will be living in developing countries¹. It is a major cause of chronic kidney disease (CKD) globally and it is associated with increased risk of cardiovascular diseases². It is

Corresponding author:

Oluyomi Okunola, Department of Medicine Obafemi Awolowo University Teaching Hospital, Ile-Ife, Nigeria. Email: yok8t@yahoo.com also the most prevalent cause of end stage renal disease in sub Saharan Africa with figures ranging from 25-50%^{3,4}. The degree of functional impairment in those who eventually develop CKD is highly variable and this has been attributed to individual variability of risk. The need to also predict those who may eventually develop CKD has further led to the search for risk factors which in concert with hypertension significantly impact on renal function and ultimately leading to ESRD.

Factors implicated as being responsible for CKD progression in hypertensives have been classified into initiating factors such as age, race (African Americans), gender, dyslipidaemias and obesity while the perpetuating factors

African Health Sciences

© 2019 Gbadegesin et al. Licensee African Health Sciences. This is an Open Access article distributed under the terms of the Creative commons Attribution License (https://creativecommons.org/licenses/BY/4.0), which permits unrestricted use, distribution, and reproduction in any medium, provided the original work is properly cited. which drive the process include; smoking, anaemia, oligonephroma, level of proteinuria, and the systolic blood pressure level⁵.

In a follow up study of 500 patients with essential hypertension, Pereira reported that proteinuria was found in 42%, while 18% developed chronic renal insufficiency over a period of time⁶. Cigarette smoking, analgesic consumption in significant quantity amongst others were seen in patients with hypertension induced chronic renal failure in Nigeria in an earlier report by Akinsola et al⁷.

The multiple risk factor interventional trial (MRFIT) study had also reported that the absolute risk of end stage renal disease was highest for those with a baseline blood pressure in the highest category of systolic blood pressure and diastolic blood pressure respectively. The relationship of blood pressure and renal damage was also positive and continuous through blood pressure range⁸.

Strategies for prevention of hypertension induced CKD must have a holistic approach based on the identification of renal risk factors that can in concert with hypertension promote the development of CKD, and its sustainability.

Risk profiling is a quantitative analysis of factors accentuating disease progression and its characteristics, the early identification of high risks should therefore form the basis for instituting preventive interventions. This becomes imperative in view of the rising global incidence and prevalence of hypertension induced kidney damage in developing countries. This study was thus carried out to determine the occurrence and pattern of renal risk factors among adult Nigerians with newly diagnosed hypertension and also aimed at detecting the relationship between the risk factors and kidney function.

Methods

This prospective study was performed at the Ladoke Akintola University Teaching Hospital(LAUTECH), Osogbo, Nigeria and at the General hospital, Osogbo over an 18 month period from Jan 1st 2009 till August 30th, 2010. Ethical approval was obtained for this study from the ethical board of LAUTECH with protocol number LTH/EC/2009/12/56.

Adults aged \geq 35 years with newly diagnosed hypertension from General Outpatient units of both centres were enrolled for the study.

Study population

This consisted of 250 consecutive adults who fulfilled the inclusion criteria for this study and who were newly diagnosed to have hypertension and another 250 age- and sex-matched healthy controls within the same community were also recruited for the study. The participants were \geq 35 years of age, yet to commence antihypertensive drugs, and gave informed consent to be included in the study. Primary hypertension was defined as systolic blood pressure \geq 140mmHg and/ or diastolic pressure \geq 90mmHg, with the absence of red cells and granular casts from the urine and a protein/creatinine ratio \leq 2.0 at baseline.

Patients with biochemical and clinical evidence suggestive of glomerulonephritis were also excluded.

A structured questionnaire was administered seeking information on socio-demographics such as age, sex, marital status and social class as established by income and occupation. History of established renal risk factors such as smoking, alcohol intake, use of salt, use of herbal medicines or remedies, analgesic use, protein intake, family history of hypertension, diabetes, or salient symptomatology of chronic kidney disease which included nocturia, passage of frothy urine and facial swelling amongst others were also obtained.

Alcohol intake was assessed with separate questions about the type, amount of alcohol consumed per week and the duration of alcohol intake prior to study enrolment.

Study participants also answered questions on smoking habits, current smoking status and the average number of cigarettes they smoked per day. The duration of use of herbal medications were also accessed. Questions were also asked concerning use of salts including adding salt to food at table and frequency of consumption of canned salted foods.

A waist circumference of ≥ 102 cm for male and ≥ 88 cm for female were taken as indicative of central/truncal obesity.Urinalysis was done using dipstick (Combi 9®) for the presence of protein, red blood cells, white blood cells, nitrite in urine samples.

Albumin: creatinine (ACR) ratio was done using clinitekR 50 (Bayer Diagnostics urine chemistry analyser, Bayer corporation, Elkhart, USA) for urine samples that were negative for proteinuria (macroproteinuria) with Combi 9 dipsticks.

Microalbuminuria was defined as an ACR of 30 mg/g to

299mg/g while macroalbuminuria was defined as an ACR of \geq 300mg/g.

Sample for lipid profile, blood glucose and haemoglobin concentration were determined automatically using an auto analyzer machine (SysmexInc, IL 60060, USA). Serum creatinine was measured by a modified kinetic Jaffe reaction. (Alkaline picric acid method, kits from RANDOX Laboratory Ltd. US), and serum electrolytes (sodium, potassium and uric acid) were analysed using flame-photometer.

Diabetes was defined as fasting blood glucose \geq 7.0mmol/l (126mg/dl).

The abbreviated Four Variable Modification of Diet in Renal Disease (MDRD) study formulawas used to estimate the glomerular filtration rate;

 $eGFR (ml/1.73m2) = 186 \times PCr (mg/dl)-1.154 \times Age(yrs)$ -0.203 x 0.742 (if female) x 1.210 (if black).

Where eGFR is the estimated glomerular filtration rate, PCr is the plasma creatinine.

The tests (Haemogram, renal function tests, uric acid, fasting blood sugar and lipid profile) were carried out on all subjects and controls.

Statistical analysis

Analysis was done using Statistical Package for Social Sciences (SPSS) version 15.

Summary statistics such as means, medians and standard deviation were used to summarize quantitative variables while qualitative data were summarized using frequencies and proportions.

The frequency of each risk factor was compared between the hypertensive patients and controls. The correlation between quantitative variables was tested using the Pearson correlation analysis while chi square test was used for two qualitative variables. The t-test and Analysis of Variance were used to compare means of two and three categories respectively. Bar charts were used to represent qualitative variables.

The risk profile of each patient with newly diagnosed hypertension was determined by combining nine selected risk factors for CKD namely smoking, alcohol intake, herbal concoction use, excessive salt intake, obesity, analgesic use, fasting hyperglycaemia, proteinuria and family history of chronic kidney disease. The number of risk factors was then grouped into 3 categories- 0 - 3 risks, >3-6 risks, > 6 risks representing mild, moderate and severe degrees respectively. The magnitude of the risk factors mild, moderate, or severe (depending on presence of 0-3, >3-6, > 6 risks) was compared between subjects and controls using chi-square test or student t-test for quantitative and qualitative variables respectively.

The independent predictors of outcomes such as GFR were identified using multiple linear regression analysis while the multiple logistic regression was used for dichotomous outcomes such as CKD. For a variable to be entered into the multiple logistic regression model, the p-value on the chi-square tests must have been less than 0.1 (10%). Hence the logistic regression analysis was carried out for predictors which were significantly associated with the outcome at p < 0.1 on chi square test. Odd ratios and their 95% confidence intervals were reported. The level of significance for all tests was 5% (p < 0.05).

Results

There were 250 study participants with newly diagnosed hypertension and 250 age and sex matched apparently healthy normotensive controls. The mean age was 55.7 ± 11.3 years for the hypertensive patients and 55.2 ± 12.5 years for the controls. There were 121 male subjects and 115 male controls respectively.

Anthropometric	Hypertensives	Controls	P value
measurement	Mean± SD	Mean± SD	
Weight (kg)	67.8±15.0	65.8±13.3	0.124
Height (m)	1.59±0.09	1.58±0.07	0.466
Body Surface Area (m ²)	1.69±0.19	1.67±0.15	0.142
BMI (Kg/m ²)	27.0±6.3	26.4±5.6	0.272
BMI Class			
Underweight	4(1.6)	6(2.4)	0.298
Normal	99(39.9)	105(42.2)	
Overweight	79(31.9)	89(35.7)	
Obesity	66(26.6)	49(19.7)	
Waist Circumference			
Normal	159(63.9)	183(73.2)	0.025
Abnormal	90(36.1)	67(26.8)	

Table 1: Comparison of anthropometric indices between hypertensive patients and controls

Table 1 shows the means and standard deviation of selected anthropometric indices among the hypertensive patients and controls. There was no statistically significant difference in the mean values of weight (p = 0.124), height (p = 0.466), body mass index (p = 0.272), waist circumference (p = 0.163) and body surface area (p = 0.142)

of the hypertensive patients and the controls. When the anthropometric indices were categorized there were significant differences in the waist circumference. A higher proportion of the hypertensive patients had abnormal waist circumference compared to controls (p = 0.025). Body mass index remained non- significant when categorized (p = 0.298).

Risk Factors	Hypertensives %	Controls %	P-value
Analgesic Use	86.4	41.6	<0.001*
Smoking	2.8	4.4	0.333
Alcohol Use	20.8	12.0	0.008*
Herbal Use	51.6	26.1	< 0.001*
Use of salted food	18.8	8.4	< 0.001*
Family history of DM	7.2	8.0	0.736
Family history of hypertension	35.2	10.2	<0.001*
Obesity (BMI \ge 30kg/m ²)	26.6	19.7	0.289
Central obesity	36.1	26.8	0.025*
Fasting Hyperglycaemia	3.3	1.2	0.021*
	5 6	0.7	<0.001*
Macroalbuminuria Microalbuminuria	5.6 42.0	0.7 18.8	<0.001* 0.001*

Table 2: Prevalence of risk factors for CKD amongst hypertensive patients and controls

Key: BMI – Body mass index * = Statistically significant

Table 2 shows that newly diagnosed hypertensives had significantly higher prevalence of analgesic use (86.4% vs. 41.6%, p<0.001), alcohol use (20.8% vs. 12%, p = 0.008), herbal use (51.6% vs. 26.1%, p<0.001), use of canned salted food (18.8% vs. 8.4%, p = 0.001), and central obesity (36.1% vs. 26.8%, p = 0.025).

The mean serum creatinine among the hypertensive patientswas $91.31\pm15.37\mu$ mol/L (range $65-129\mu$ mol/L) and the mean serum creatinine among the controls was $91.2\pm15.9 \mu$ mol/L (range $64-128\mu$ mol/L). There was however no significant statistical difference in the mean serum creatinine values between the two groups (t =0.051, p =0.959). Table 3 also shows the indicators of renal function compared between newly diagnosed hypertensives and controls. Macroalbuminuria and microalbuminuria were significantly commoner among subjects compared to controls (p<0.001). Eighty one (42%) hypertensive patientscompared to 26 (18.8%) of controls had microalbuminuria, while 14(5.6%) of the hypertensive patients compared with 1 (0.7%) control had macroalbuminuria. Seventy (28%) of the hypertensive patients and ten (4%) of the controls had estimated GFR less than 60mls/min/1.73m² respectively.

Table 3: Comparison of indicators of reduced renal function between hypertensive patients and controls.

Indicator	Hypertensives n (%)	Controls n (%)	P value
Serum creatinine >120(µml/l)	9(6.0)	3(2.0)	0.079
Microalbuminuria (mg/g)	81(42.0)	26(18.8)	<0.001
Macroalbuminuria (g/l)	14(5.6)	1(0.7)	<0.001

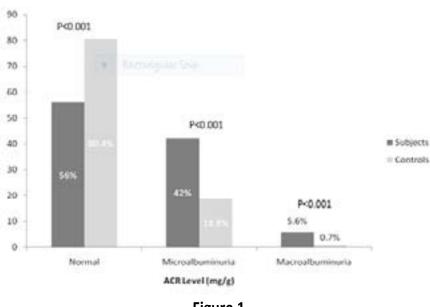




Figure 1 shows the distribution of study population according to albumin- creatinine ratio (ACR). The prevalence of microalbuminuria in the hypertensive patients was 42.4% compared to 18.8% in the controls. This was statistically significant p < (0.001). Macroalbuminuria was also commoner among the hypertensive patients when compared to the controls (5.6% vs. 0.7%, p = 0.001) The output from the logistic regression of impaired kidney function (GFR < 60ml/min/m2) on variables is

shown in Table 4. After adjusting for the various risk factors identified in this study, the major predictors of impaired kidney function were gender – with females more likely to develop the disease compared to male participants (OR =0.244, 95% CI = 0.108 - 0.551); age which indicates that the hypertensive patientswere more likely to develop CKD (OR = 1.064, 95% CI = 1.043-1.085), and fasting hyperglycaemia (OR = 4.799, 95% CI = 1.602 - 14.374).

Variable	Regression	95% Confidence	P value
	coefficient	interval	
Age	-1.00	-1.11 to -0.89	<0.001*
Gender (male vs female)	11.74	9.16 to 14.32	<0.001*
Waist circumference	0.16	0.02 to 0.30	0.030*
MABP	0.22	-0.002 to 0.44	0.052
Smoking	-1.56	-12.84 to 9.72	0.786
BMI	1.12	0.81 to 1.44	<0.001*
Systolic blood pressure	-0.16	-0.34 to 0.018	0.078
Herbal concoction use	-0.893	-4.370 to 2.585	0.614

Table 4: Regression coefficients and confidence intervals from regression

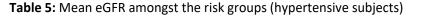
 of estimated GFR on selected risk factors for CKD among subjects

*Significant at 5% level

The comparison of mean eGFR among the risk factor categories is shown in Table 5. The risk factor categories were grouped into 3: less than 3 risks, 3-6 risks and greater than 6 risks. The eGFR decreased with the number of risk factors and the difference was statistically significant (p < 0001). Also, the newly diagnosed hypertensives with

risk factors have significantly lower mean eGFR when compared with newly diagnosed hypertensive without risk (59.80 \pm 17.55 vs 74.68 \pm 20.92mls/min, p = (0.001). Figure 2 shows a significant negative correlation between eGFR and magnitude of risk factors (r = -0.597, p < 0.001).

Risk Group	Mean eGFR ± SD	F	P-value
<3risks (n = 97)	84.6 (14.9)	12.182	<0.001
3-6 risks (n = 101)	77.2 (19.9)		
> 6 risks (n=52)	68.9 (16.8)		



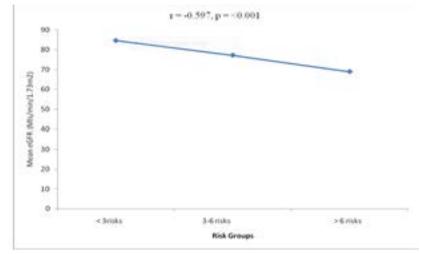


Figure 2 showing the multiple regression analysis of the risk factors on renal function

Nine risk factors namely smoking, alcohol intake, herbal concoction use, excessive salt intake, proteinuria, obesity, fasting hyperglycaemia, analgesic use and family history of CKD were selected in the categorization of the level of CKD risk. These risk factors were used in classifying patients based on the number of risk factors they possess. A higher number of controls had less than 3 risk factors compared to the hypertensive patients. About 165 (66.0%) of the hypertensive patients had greater than 6 risk factors while 42(16.8%) had between 3-6 risk factors. These were higher when compared to controls. (Table 6)

RISK GROUP	Frequency in hypertensives n (%)	Frequency in controls n (%)
< 3risk factors	43(17.2)	177 (70.8)
3–6 risk factors	42 (16.8)	30 (12.0)
>than 6 risk factors	165 (66.0)	43 (17.2)
Total	250(100)	250 (100)

Table 6: Risk profiling amongst hypertensive patients and controls

Discussion

In this study, renal function was assessed by estimated glomerular filtration rate (GFR) using the Modification of Diet in Renal Disease (MDRD) formula and kidney damage assessed by albuminuria9. Seventy (28%) of the newly diagnosed hypertensives had estimated GFR < 60ml/min and 42.4% had microalbuminuria, compared with 18.8% with microalbuminuria amongst the controls. The prevalence of the microalbuminuria in the hypertensive patients in this study is similar to the 45.5% prevalence reported by Nwankwo et al in newly diagnosed hypertensives¹⁰. This figure was also higher than figures found by Olatunde et al who reported microalbuminuria of 17.4% in a similar study done at Ile- Ife, Nigeria¹¹. The prevalence observed in the control population in this study is higher than that found by Coresh et al and the Dutch PREVEND study (9.3% and 7.2%) respectivelv^{12,13}.

The relatively significant presence of microalbuminuria in this study amongst newly diagnosed hypertensives may indicate early endothelial damage and this portends future glomerular damage and CKD. Specifically, the severity of blood pressure and increased systemic permeability to albumin possibly due to endothelial dysfunction had been found to play an important role in its development. Additional factors such as lipid abnormalities, prothombotic factors, increased activities of RAS and systemic inflammation have also been implicated^{13,14}. The goal of drug treatment of hypertension should include amelioration/ reduction of associated microalbuminuria from the onset , particularly among population pre disposed to chronic renal disease.

The prevalence of CKD in this study(as defined by GFR < 60ml/min/1.73m²) is 28% in the newly diagnosed hypertensives is similar to what was found in a study by Ayodele et alwho reported a prevalence of 23.1% among newly diagnosed hypertensive patients¹⁵. The prevalence was however higher than 4.7%, 5.7% and 8% reported in NHANES¹⁶, EPIRCE¹⁷ and Democratic Republic of Congo¹⁸. The high prevalence found may not be unconnected to the background hypertension in our subjects which confers higher risk of kidney damage in this population when compared to the other studies which largely consisted of untargeted population.

The mean serum creatinine among the newly diagnosed hypertensives was higher among subjects compared to

controls. The prevalence of impaired kidney function using a cut off value of serum creatinine > 120μ mol/L was 6.0% among the newly diagnosed hypertensives. The Kidneys have strong compensatory function and it may take about 50% loss of its function before serum creatinine starts to rise. Therefore, measurement of serum creatinine alone may under-diagnose CKD. It is advocated by NKF that renal function be assessed using estimated GFR and other markers of kidney damage which provide a more sensitive representation of kidney function or allows for early diagnosis of CKD for prompt institution of management¹⁹.

The study found significant association between increasing age and impaired kidney function in both populations studied. As reported in other studies, which shows increasing prevalence of low GFR with age, elderly participants have highest prevalence of CKD and the relationship remains significant even after regression analysis^{20,21}. This is in agreement with findings from other studies. Rowe et al and Linderman had reported a natural decline of kidney function with age^{22,23}. Possible reasons adduced by the latter for this association are reduction in the number of nephron, renal mass and presence of nephrosclerosis in ageing. The mean age of the participants, increased prevalence of risk factors and ethnicity (black race) are likely to have contributed to the higher prevalence in this study than some of the other studies. As impaired kidney function is commoner in the older age group, the recommendation to screen people in this age group would be an important strategy for detection of chronic kidney disease.

It was observed that values suggestive of reduced renal reserve (eGFR <60mls/min) was commoner in the female (32.9%) than male (20.8%). Female gender independently predicts CKD in this study. AusDiab²⁰, NHANES16 and China21 studies showed significant higher female gender prevalence of CKD. Data regarding the role of gender in determining renal risk in humans are somewhat contradictory. In general, the prevalence of CKD was greater in male than female regardless of age and also in various ethnic groups. In this study, the female subjects were older, had higher SBP and DBP (although these values were not statistically significant) and these findings may explain the higher prevalence of eGFR < 60mls/min and albuminuria in females compared to males. However, many studies have shown higher prevalence of ESRD in males than females^{22,23,24}.

Obesity represents a state of excess storage of body fat and could be defined as an excess body weight for height. With prolonged obesity, there is resultant further impairment of renal-pressure natriuresis, leading to a more severe hypertension and a gradual loss of kidney function²⁵. Aryee et al in a case control study involving 241 participants noted that hypertensives with chronic kidney disease had higher anthropometric indices, it was observed that body adiposity index and abdominal volume index were associated with corresponding incremental changes in the bloodpressure²⁶. In this study, a significant number of study participants (newly diagnosed hypertensives) had central obesity.

Smoking as a risk factor to renal damage was also noted in this study, as it may contribute to renal damage via sympathetic nervous activation, sustained hypertension via promotion of renal atherosclerosis and effects on endothelial functions²⁷. Generally it shortens the interval from microalbuminuria to overt nephropathy and accelerates progression of nephropathy and loss of glomerular filtration rates²⁷.

The habitual consumption of alcohol was seen in 20.8% of the subjects. It causes an increase in sympathetic activity, stimulation of the renin angiotensin-aldosterone system and an increase of intracellular calcium levels with a subsequent increase in vascular reactivity²⁹. However these seems to be speculative.

Excessive salt consumption was seen in 18.8% of the subjects compared with 8.4% among the controls (P< 0.05). In patients with hypertension, an increase in salt intake often increases the GFR, vascular resistance, calculated intraglomerular capillary pressure and protein excretion³⁰. Studies such as DASH and INTERSALT have all suggested a reduction in salt quantities to reduce cardiovascular morbidity. The KDIGO recommends a sodium intake of less than 90mmol/day(2g) which corresponds to 5g of sodium chloride⁹.

Analgesic consumption was also significant among 86.4% of the subjects compared with the controls. In Nigeria the pattern of analgesic consumption and its contribution to renal disease is not fully known. Abioye-kuteyi found a prevalence of 74.7% in a rural area consisting of unskilled workers31. Hypertension, pre-existing renal disease are known risk factors for renal failure after ingestion of NSAIDS³².

Chronic analgesic nephropathy is a slowly progressive renal disease resulting from daily use for many years of mixture containing at least two analgesics (e.g aspirin, paracetamol, phenacetin) and caffeine, codeine, and/or barbiturates, which may lead to psychological dependence and overuse. The cumulative dose of analgesics required to cause renal impairment is about 4kg over 2 years³¹.

When the risk factors were grouped into three: less than 3(mild risk), 3-6 risk (moderate risks), \geq 6 risk (high risks), there was significant difference in the prevalence of renal risk in the newly diagnosed hypertensives when compared to the controls. Occurrence of multiple risks was associated with reduction in renal function, there was an inverse relationship between occurrence of multiple risks and decrease in eGFR. Also, the mean eGFR was found to be higher in subjects with no added risk compared to those with risk(s). The above findings may be explained by the multi-hit hypothesis which states that multi-factors interact to overcome renal reserve and provoke progressive nephron loss³³. The magnitude of the risk factors in newly diagnosed hypertensives therefore plays an important role in the subsequent reduction in renal reserve.

Conclusion

The study reports a high prevalence of modifiable renal risk factors. These include analgesic abuse, herbal concoction and obesity amongst the newly diagnosed hypertensives. Markers suggestive of early impairment of kidney function as evident by microalbuminuria and reduced GFR were also present in a sizeable number of patients and they stand a higher risk of faster disease progression and of having cardiovascular complications. This finding underscores the need to improve the awareness of hypertension in the population since this will lead to early detection.

Analgesic abuse and undefined herbal remedy usage should be discouraged. In conclusion, methods that could be used to stem down the risk factors especially in developing countries and indeed globally is through continuous promotion and advocacy of healthy behaviours and habits via education and awareness.

Financial support and sponsorship Nil.

Conflicts of interest

The authors have no conflict of interest to declare concerning this research work.

References

1. Kearney PM, Whelton M, Reynolds K, Muntner P, Whelton PK, He J . Global burden of hypertension: analysis of worldwide data. *Lancet* 2005; 365:217-223

2. McCullough PA, Jurkovitch CT, Pergola PE, Mc-Gill JB, Brown NW, Collins AJ et al. Independent components of chronic kidney disease as a cardiovascular risk state: results from the Kidney Early Evaluation Program (KEEP). *Arch Intern Med* 2007; 167:1122-1129.

3. Okunola O, Akinsola A and Ayodele O. Kidney diseases in Africa: Aetiological considerations, peculiarities and burden. *Afr J Med Med.* 2012;41:119-133

4. Osafo C, Mate-Kole M, Affram K, Adu D. Prevalence of chronic kidney disease in hypertensive patients in Ghana. *Ren. Fail.* 2011; 33(4) 388-92.

5. Taal MW and Brenner BM. Predicting initiation and progression of chronic kidney disease: developing renal risk scores. *Kidney Int* 2006;70:1694-1705

6. Pereira G: Hypertensive vascular disease: description and natural history. *J. Chronic Dis* 1955; 1:33 – 42.

7. Akinsola A and Adelekun TA. Hypertension induced chronic renal failure: clinical features, management and prognosis. *WAJM*, 1998; 17(2): 104-108

8. Klag MJ, Whelton PK, Randall BL, Neaton JD, Brancati FL, Ford CE et al. Blood pressure and end stage renal disease in men. *N Engl J Med* 1996, 334; 13-18

9. Kidney Disease Improving Global Outcomes (KDIGO) CKD work Group 2012. Clinical practice guideline for the evaluation and management of chronic kidney disease. *Kidney Int Supplement* 2013;3(1):1-150

10. Nwankwo EA, Wudiri WW. Akinsola A: Risk factor for development of chronic kidney disease among Nigerians with essential hypertension. *J. Med. Sci* 2007; 7(1): 579-584

11. Olatunde LO, Arogundade FA, Balogun MO. Microalbuminuria and its clinical correlates in essential hypertension. *Nig J Health Sci*, 2002; 2: 25–29.

12. Coresh J, Stevens LA. Kidney function estimating equations: where do we stand? *Curr Opin Nephrol Hypertens* 2006; 15:276-284.

13. Verhave JC, Gansevoort RT, Hillege HL, Bakker SJ, De Zeeuw D, De Jong PE; PREVEND Study Group: An elevated urinary albumin excretion predicts de novo development of renal function impairment in the general population. *Kidney Int Suppl.* 2004; 66: S18 –S21.

14. Giuseppe D'Amico and Claudio Bazzi. Pathophysiology of proteinuria. *Kidney. Int.* 2003:63:809-205 15. Ayodele OE, Egbewale BE and Alebiosu CO. Kidney function and clinical correlates in newly diagnosed hypertensives attending a University Teaching Hospital in Southwest Nigeria. *African J. Med. med.sc.* 2007; 36 : 95 -101

16. United States Renal Data System. USRDS 2002 Annual Data Report. 2002; Atlas of End-Stage Renal Disease in the United States, Bethesda, National Institutes of Health, National Institute of Diabetes and Digestive and Kidney Diseases, 2002.

17. Otero A, Gayoso P. Epidemiology of chronic renal disease in the Galician population: Results of the pilot Spanish EPIRCE study. *Kidney Int.* 2005; 68: S16 – S19

18. Sumaili EK, Krzesinski JM, Zinga CV: Prevalence of chronic kidney disease in Kinshasa: Results of a pilot study from the democratic republic of Congo. *Nephrol Dial Transport* 2009; 24:117-122

19. National Kidney Foundation K/DOQI Clinical Practice Guidelines for Chronic Kidney Disease: Evaluation, Classification and Stratification. *Am J Kidney Dis* 2002; 39 (2 Suppl 1): S1 – S266.

20. Chadban SJ, Briganti EM, Kerr PG, Dunstan DW, Welborn TA, Zimmet PZ et al. Prevalence of chronic kidney damage in Australian adults. The AusDiab Kidney Study. *J. Am Soc Nephrol.* 2003; 14(7 suppl 2): S131-S138.

21. Hallan SI, Coresh J, Astor BC, Asberg A, Powe NR, Romundstad S et al: International comparison of the relationship of chronic kidney disease prevalence and ESRD risk *J. Am SocNephrol* 2006:17:2275-2284

22. Rowe JW, Andres R, Tobin JD, Norris AH, Shock NW et al. The effect of ageing in creatinine clearance in men: a cross sectional and longitudinal study. *J. Gerontol* 1976; 31(2):155-163.

23. Lindeman RD, Tobin J, Shock NW: Longitudinal studies on the rate of decline in renal function with age. *J. Am Geriatr Soc* 1985; 33: 278 – 285.

24. Eriksen BO, Ingebretsen OC: The progression of chronic kidney disease: a 10 year population based study of the effects of gender and age. *Kidney Int* 2006; 69:375 – 382.

25. Hall JE. Pathophysiology of obesity hypertension. *Curr Hypertens Rep* 2000;2:139-147

26. Aryee C, OwireduWK,Osei-yeboahJ,Owusu-Dabo E, Laing E et al. An analysis of anthropometric indicators and modifiable lifestyle parameters associated with hypertensive nephropathy. *Int. J. Hypertens.* 2016. Doi. org/10.1155/2016/6598921 27. Ritz E, Benck U, Franek E, Keller C, Seyfarth M, Clorious J. Effect of smoking on renal heamodynamics in healthy volunteers and in patients with glomerular disease. *J Am Soc Nephrol* 1998;(10):1798-804

28. Orth SR, Ogata H, Ritz E. Smoking and the kidney. *Nephrol Dial Transplant*. 2000;15:1509-1511

29. Savdie E, Grosslight GM, Adena MA. Relation of alcohol and cigarette consumption to blood pressure and serum creatinine levels. *J chronic Dis* 1984;37:617-23

30. Mallamaci F, Leonardis D, Bellizzi V. Does high

salt intake cause hyperfiltration in patients with essential hypertension. *J Hum Hypertens*1996;10:157-161

31. Abioye-Kuteyi EA, Akinsola A, Ezeoma IT. Renal disease: The need for community-based screening in rural Nigeria. *Afr J Med Pract* 1999; 6(5):198-201

32. De Broe ME, Elseviers MM: Analgesic nephropathy. *N Engl J Med* 338: 446–452, 1998

33. Kasiske BL, Ma JZ, Louis TA, Swan SK : Longterm effects of reduced renal mass in humans. *Kidney Int.* 1995; 48(3): 814 - 819.