Journal of Medical and Biomedical Sciences (2012) 1(3): 1-12


# ORIGINAL ARTICLE

## Metabolic syndrome among Ghanaian patients presenting with chronic kidney disease

W.K.B.A Owiredu[1], R.K.D. Ephraim[2], B.A. Eghan Jnr.[3], N. Amidu[4] and E.F. Laing[1]

*[1]Department of Molecular Medicine, [3]Department of Medicine, School of Medical Sciences, College of Health Sciences, Kwame Nkrumah University of Science and Technology, Kumasi, Ghana; [2]Department of Laboratory Technology, University of Cape Coast, Ghana [3]Department of Medical Laboratory Science, School of Medicine and Health Sciences, University for Development Studies, Tamale, Ghana*

**Metabolic syndrome (MetS) is a general risk factor for cardiovascular and chronic kidney disease (CKD) in Western populations. This study assessed the relationship between MetS and its components in Ghanaian patients presenting with CKD. The study population comprised of 146 non-dialysed individuals with CKD with mean age of 50.2±1.1 years. Eighty (80) age and sex matched healthy participants without kidney pathology were used as controls. Estimated GFR (eGFR) was calculated using the 4-variable Modification of Diet in Renal Disease (*4v*-MDRD) and CKD was defined as eGFR<60 ml/min/1.73m². MetS was defined as the presence of three or more of the following risk factors according to the National Cholesterol Education Program Adult Treatment Panel III (NCEP-ATP III) criteria: elevated blood pressure (BP), low high density lipoprotein cholesterol (HDL-C), high triglycerides (TG), elevated plasma glucose and abdominal obesity. The prevalence of MetS in this study was 30.1% and a significant relationship was observed between the number of MetS components and the presence CKD. The CKD group are about 3 times at risk of developing MetS as compared to the control group (95% CI=0.9-8.8). Female participants with CKD are 9 fold at risk of developing MetS as compared to the male counterparts (95% CI=1.7-47.9). The CKD patients were about 2 fold at risk of developing hypertension (95% CI=1.7-3.3) and diabetes (95% CI=1.2-2.6), about 3 times at risk of developing hypertriglyceridaemia (95% CI=1.1-5.5) and approximately 4 times at risk of developing proteinuria (95% CI=2.7-7.0). Increased WC, TG and SBP are components of the metabolic syndrome which contribute to the initiation and progression of CKD.**
*Journal of Medical and Biomedical Sciences (2012) 1(3), 1-12*

**Keywords***:* Metabolic syndrome, diabetes, dyslipidaemia, obesity, chronic kidney disease

### INTRODUCTION

Chronic kidney disease (CKD) has become a global public health concern due to its increasing prevalence (Coresh *et al.*, 2003) and the associated increase in risk of end-stage kidney disease (ESKD), cardiovascular disease (CVD) and untimely deaths (Muntner *et al.*, 2002; National Kidney Foundation, 2002). Identifying and treating risk factors for development of CKD may therefore be the best approach to preventing and/or delaying adverse outcomes (National Kidney Foundation, 2002).

MetS, characterized by a clustering of abdominal obesity, hypertriglyceridaemia, low high-density lipoprotein cholesterol (HDL-C), elevated blood pressure (BP), and high fasting blood glucose (FBG), has been associated with an increased risk for the development of diabetes and CVD as well as an increased mortality from CVD and all causes (Ford, 2005; Reynolds and He, 2005). The National Cholesterol Education Program Adult Treatment

Correspondence: *W.K.B.A. Owiredu, Department of Molecular Medicine, School of Medical Sciences, Kwame Nkrumah University of Science and Technology, Kumasi, Ghana. E*-mail: wkbaowiredu@yahoo.com

Panel (NCEP-ATP III) criteria defines MetS as having at least three of the following: abdominal obesity; high triglyceride levels; low high-density lipoprotein (HDL) cholesterol; hyperglycaemia; and hypertension (NCEP, 2001).

MetS is important for several reasons: (a) it is one of the causes of CKD (Kambham *et al.*, 2001), (b) it can be treated at lower cost if detected early and (c) it is a predictor of CVD (Iseki *et al.*, 2004). A few epidemiological studies among the global adult population especially in the United States of America have reported that MetS is associated with CKD and microalbuminuria (Chen *et al.*, 2004; Kurella *et al.*, 2005). Growing economic development over the years has led to changes in lifestyle and diet, and consequently an increased prevalence of obesity in Ghana. Thus, MetS with its association to obesity is expected to be even more prevalent now and in the future. However, there is paucity of data on the relationship between MetS and CKD. The aim of the present study therefore was to establish the relationship between MetS and CKD in the Ghanaian population.

## MATERIALS AND METHODS

### Study area and subjects

This study was carried out at the Komfo Anokye Teaching Hospital (KATH), Kumasi and the Tamale Teaching Hospital (TTH) between August 2007 and September 2009. One hundred and forty six (146) patients comprising eighty (80) females and sixty-six (66) males within the age range of 20-80 years were recruited into the study after the objectives of the study had been clearly explained to them in English and/or the local dialect. Patients with clinically diagnosed CKD who were yet to commence dialysis were randomly selected for the studies with patients on any form of dialysis being excluded from the study.

The aetiology of the CKD ranged from diabetic nephropathy, 90(61.6%) patients; chronic glomerulonephritis, 12(8.2%) patients; adult polycystic kidney disease, 1(0.7%) patient; hypertensive nephropathy, 10(6.8%) patients and chronic kidney disease of unknown aetiology, 33(22.6%) patients. Eighty (80) healthy volunteers of similar age and sex distribution were studied as controls. The participation of the respondents who are all indigenes of Ghana was voluntary and informed consent was obtained from each of them. The study was approved by the School of Medical Sciences and the Komfo Anokye Teaching Hospital Committee on Human Research, Publication and Ethics (SMS/KATH/CHRPE).

### Sample collection

Venous blood samples were collected after an overnight fast (12–14 hours), between 7 am and 10 am. About 5 ml of venous blood was collected out of which three 3 ml was dispensed into vacutainer® plain tubes and 2 ml into fluoride oxalate tubes. After centrifugation at 500 g for 15 min, the serum and plasma were stored at - 80°C until assayed.

### Biochemical assays

Serum Biochemistry was performed with ATAC® 8000 Random Access Chemistry System (Elan Diagnostics, Smithfield, RI, USA). Parameters that were determined include; fasting blood glucose (FBG), serum creatinine (CRT), total cholesterol (TC), triglycerides (TG) and high density lipoprotein cholesterol (HDL-C). Serum low density lipoprotein cholesterol (LDL-C) was calculated using the Frederickson-Friedewald's formula (Friedewald *et al.,* 1972). The methods adopted by the automated instrument for the estimation of the above parameters was according to the instructions provided by the reagent manufacturer-JAS™ diagnostics, Inc. (JAS Diagnostics, Inc. Miami Florida, USA). TC determination was according to the method described by Trinder (Trinder, 1969). TG determination employed a modified Trinder method (Trinder, 1969; Barham and Trinder, 1972). LDL-C determination: LDL-C (mmol/l) was calculated according to Friedwald's formula in accordance with the manufacturer's instructions i.e. $LDL_C$=TC - TG⁄2-$HDL_C$.

### Urine protein estimation

Early morning urine was collected in plastic containers from the respondents and urine protein was determined using the dip-stick qualitative method

(CYBOW™ DFI Co Ltd, Gimhae-City, Republic of Korea).

**Anthropometric variables**

Anthropometric measurements included height to the nearest 0.5 cm without shoes and weight to nearest 0.1 kg in light clothing. Subjects were weighed on a bathroom scale (Zhongshan Camry Electronic Co. Ltd, Guangdong, China) and their height measured with a wall-mounted ruler. Body mass index (BMI) was calculated by dividing weight (kg) by height squared ($m^2$). Waist circumference (WC) (to the nearest centimetre) was measured with a Gulick II spring-loaded measuring tape (Gay Mills, WI) midway between the inferior angle of the ribs and the suprailiac crest. Blood pressure was measured by trained personnel using a mercury sphygmomanometer and a stethoscope. Measurements were taken from the left upper arm after subjects had been sitting for >5 minute in accordance with the recommendations of the American Heart Association (Kirkendall et al., 1967). Duplicate measurements were taken with a 5 minute rest interval between measurements and the mean value was recorded to the nearest 2.0 mmHg.

**Estimation of GFR**

The 4-variable Modification of Diet in Renal Disease (4v-MDRD) equation was used to estimate the GFR of both participants with CKD and controls using serum CRT. This equation has been found to be the most accurate among the renal function equations in CKD applicable to Ghanaians (Owiredu et al., 2008). The eGFR result from the equations was used to stratify the study population into five categories corresponding with the five stages of CKD in the Kidney Disease Outcome Quality Initiative (K/DOQI) classification (NKF/KDOQI™, 2002). The staging classified GFR ≥ 90 ml/min/1.73 $m^2$ as stage 1; 60-89 ml/min/1.73 $m^2$ as stage 2; 30-59 ml/min/1.73 $m^2$ as stage 3; 15-29 ml/min/1.73 $m^2$ as stage 4; and < 15 ml/min/1.73 $m^2$ as stage 5.

**Definitions**

CKD defined as eGFR<60 ml/min/1.73m2.

MetS was defined according to the criteria of the National cholesterol education program, adult treatment panel III (NCEP ATP III) to include individuals with three or more of the following five components: (1) abdominal obesity- (waist circumference > 102 cm for men, or > 88 cm for women); (2) high TG ≥ 1.7 mmol/L (150 mg/dl); (3) low HDL-C : men < 0.9 mmol/L (< 40 mg/dl) or women < 1.0 mmol/L (< 50 mg/dl); and (4) High BP (systolic BP ≥ 130 mmHg or diastolic BP ≥ 85 mmHg or treatment of hypertension); and (5) high fasting glucose ≥ 6.1 mmol/l (NCEP, 2001).

**Statistical analysis**

The results are expressed as Means ± SEM. Unpaired t-test was used to compare mean values of continuous variables and $\chi^2$ was used to compare discontinuous variables. A level of $p<0.05$ was considered as statistically significant. MetS (or its components) and other known risk factors for CKD were included in the model. Odds ratio (OR) (with 95% CI) of CKD by the number of metabolic risk factors were calculated. GraphPad Prism version 5.00 for windows was used for statistical analysis (GraphPad software, San Diego California USA, www.graphpad.com).

## RESULTS

**General characteristics of the study population**

Table 1 represents the general characteristics of the study population. Participants with CKD had significantly higher levels of urine protein, serum creatinine and lower levels of estimated GFR as compared to the control subjects; however there was no significant difference between the ages of the cases and controls. The mean values of most components of the metabolic syndrome were significantly higher when the CKD group were compared to the control group i.e. the CKD group had significantly higher WC, had higher blood pressure [systolic blood pressure (SBP) and diastolic blood pressure (DBP)], higher fasting blood glucose (FBG) and had higher lipid levels (i.e. TG and TC) than the control group (Table 1). When CKD patients were stratified according to the presence or absence of the MetS, those with MetS were significantly older, had higher SBP, and higher levels of TG compared to those without MetS. The mean value of HDL-C was significantly lower among those with MetS

**Table 1: General characteristics of study population with and without metabolic syndrome**

| Parameters | Control (n=80) | CKD (n=146) | MetS | | Gender | |
|---|---|---|---|---|---|---|
| | | | MetS+CKD (n=44) | MetS-CKD (n=102) | CKD-Female (n=80) | CKD-Male (n=66) |
| Age (yrs) | 46.3 ± 1.9 | 50.2 ± 1.1 | 61.0 ± 2.6 | 44.0 ± 1.6†† | 46.2 ± 2.3 | 48.1 ± 1.7 |
| BMI (kg/m²) | 24.6 ± 0.8 | 24.4 ± 0.4 | 27.6 ± 1.3 | 24.8 ± 0.5† | 26.2 ± 0.9 | 24.3 ± 0.6 |
| WC (cm) | 74.1 ± 1.7 | 85.0 ± 1.4* | 89.4 ± 3.1 | 82.3 ± 1.6† | 84.6 ± 2.2 | 84.0 ± 1.9 |
| SBP (mmHg) | 120.7 ± 1.8 | 140.4 ± 3.8*** | 154.5 ± 4.3 | 135.6 ± 2.4† | 144.7 ± 3.5 | 136.5 ± 2.8 |
| DBP (mmHg) | 70.4 ± 1.2 | 90.3 ± 2.6*** | 98.2 ± 2.7 | 87.3 ± 1.7† | 93.4 ± 2.5 | 87.7 ± 1.8 |
| PRT (g/l) | 0.04 ± 0.02 | 1.2 ± 0.2*** | 0.7 ± 0.2 | 1.1 ± 0.2 | 1.2 ± 0.4 | 1.2 ± 0.3 |
| CRT (µmol/l) | 105.9 ± 3.9 | 268.0 ± 25.6*** | 371.2 ± 82.6 | 353.9 ± 47.5 | 221.8 ± 25.0 | 325.3 ± 47.4 |
| FBG (mmol/l) | 5.3 ± 0.2 | 8.7 ± 0.3*** | 7.8 ± 0.5 | 6.9 ± 0.3 | 6.8 ± 0.5 | 7.2 ± 0.6 |
| HDL-C (mmol/l) | 1.3 ± 0.05 | 1.6 ± 0.2 | 1.1 ± 0.1 | 1.4 ± 0.1†† | 1.4 ± 0.1 | 1.3 ± 0.1 |
| TG (mmol/l) | 1.5 ± 0.1 | 1.8 ± 0.1* | 2.7 ± 0.1 | 1.9 ± 0.1† | 1.8 ± 0.2 | 2.2 ± 0.3 |
| TC (mmol/l) | 4.5 ± 0.1 | 5.3 ± 0.3* | 5.6 ± 0.2 | 5.3 ± 0.2 | 5.4 ± 0.4 | 5.3 ± 0.4 |
| eGFR (ml/min/1.73 m²) | 92.4 ± 5.7 | 57.6 ± 4.1*** | 99.7 ± 13.4 | 89.3 ± 6.9 | 50.2 ± 4.1 | 66.8 ± 7.6§ |
| Prevalence of MetS | 3 (3.75%) | 44 (30.1%) | | | 29(36.2%) | 15 (22.7%) |

***BMI = Body mass index, WC= Waist circumference, SBP = Systolic blood pressure, DBP = Diastolic blood pressure, PRT = Proteinuria, CRT = Creatinine, TC = Cholesterol, HDL-C = High density lipoprotein, TG = Triglyceride, FBG = Fasting blood glucose, eGFR = estimated glomerular filtration rate, MetS = Metabolic syndrome. *p<0.05, **p<0.01, ***p<0.001; †p<0.05, ††p<0.01; §p<0.05 when the groups were compared.***

compared to those without MetS. Furthermore, when the CKD patients were classified by gender, the female subjects had significantly lower estimated GFR compared to the control group. The risk of developing MetS is similar among both sexes (Table 1).

**Relative risk of developing MetS risk factors**

Table 2 represents the odds ratios of MetS risk factors in CKD stratified by the presence or absence of MetS and gender. When compared with the control subjects, the CKD patients were about 9 fold at risk of developing hypertension (95% CI = 3.1- 25.1) and diabetes (95% CI = 4.7-18.2), about 2 times at risk of developing hypertriglyceridaemia (95% CI = 1.3-4.2) and approximately 4 times at risk of developing low HDL (95% CI= 1.5-13.4). The risk of developing proteinuria is several folds in the CKD patients compared to the controls (OR=409; 95% CI = 24.7-6759).

When the CKD patients were stratified based on the presence or absence of metabolic syndrome, those with MetS were about 7 times at risk of developing hypertension (95% CI = 2.9-16.8), obesity (95% CI = 2.8-16.0) and proteinuria (95% CI = 3.0-16.4) and 3 times at risk of developing diabetes (95% CI = 1.2-6.4) (Table 2). Furthermore, the risk of developing hypertriglyceridaemia is several folds among those with MetS compared to those without MetS (OR = 18.2; 95% CI = 5.2-63.6). The risk of developing obesity (OR = 0.2; 95% CI = 0.1-0.6) and proteinuria (OR = 0.4; CI = 0.2-0.8) is less pronounced in the males compared to the females (Table 2).

**Comparison between patients with increasing number of comorbidities**

The comparison between patients with increasing comorbidities is shown in Figure 1. Comorbidity was defined as the presence of one or more risk factors of MetS. Participants with greater number of comorbidities (≥3) also had higher WC ($F_{3,46}$ = 2.878; p =0.046), BMI ($F_{3,46}$ = 4.112; p=0.010) and SBP levels ($F_{3,43}$ =2.546; p=0.048). For those having zero, one or two comorbidities, the WC levels were 68.1±4.7 m, 86.4±2.5 m and 86.6±5.3 m respectively. The BMI levels were 19.2±1.0 kgm$^{-2}$, 27.3±1.2

**Table 2: Odds Ratios of MetS risk factors in CKD stratified by presence/absence of MetS or gender**

| Variables | Raised BP | Raised FG | Obesity | Raised TG | Reduced HDL-C | Proteinuria |
|---|---|---|---|---|---|---|
| Control (n=80) | 4/80(5.5%) | 14/80(17.5%) | 13/80(16.2%) | 22/80(27.5%) | 4/80(5.0%) | 0/80(0.0%) |
| CKD (n=146) | 45/146(30.8%) | 97/146(66.4%) | 36/146(24.6%) | 69/146(47.2%) | 28/146(19.2%) | 105/146(72.0%) |
| OR(95% CI) | 8.9(3.1- 25.1)*** | 9.3(4.7-18.2)*** | 1.7(0.8-3.4)ns | 2.3(1.3-4.2)** | 4.5(1.5-13.4)** | 409(24.7-6759)*** |
| ***Stratified based on metabolic syndrome*** | | | | | | |
| CKD-MetS (n=102) | 31/113(27.4%) | 43/113(38.0%) | 17/113(15.0%) | 40/113(35.3%) | 30/113(26.5%) | 28/113(24.8%) |
| CKD+MetS (n=44) | 24/33(72.8%) | 21/33(63.6%) | 18/33(54.5%) | 30/33(90.9%) | 12/33(36.3%) | 23/33(69.7%) |
| OR(95% CI) | 7.0(2.9-16.8)*** | 2.8(1.2-6.4)* | 6.8(2.8-16.0)*** | 18.2(5.2-63.6)*** | 1.6(0.7-3.6) | 7.0(3.0-16.4)*** |
| ***Stratified by gender*** | | | | | | |
| CKD+Female (n=80) | 25/80(31.2%) | 52/80(65.0%) | 28/80(35.0%) | 45/80(56.2%) | 16/80(20.0%) | 64/80(80.0%) |
| CKD-Male (n=66) | 20/66(30.3%) | 45/66(68.2%) | 8/66(12.1%) | 34/66(51.5%) | 16/66(24.2%) | 40/66(60.6%) |
| OR(95% CI) | 0.9(0.5-1.9)ns | 1.1(0.6-2.3)ns | 0.2(0.1-0.6)** | 0.8(0.4-1.6) | 1.6(0.7-3.5) | 0.4(0.2-0.8)* |

***HDL-C = High density lipoprotein cholesterol, CKD = Chronic kidney disease, OR = Odds ratio, CI = Confidence interval, BP = Blood pressure, FG = Fasting glucose, TG = triglyceride, CKD+MetS=CKD patients with metabolic syndrome, CKD-MetS=CKD patients without metabolic syndrome and ns=not significant *p<0.05, **p<0.01, ***p<0.001.***

kgm-2, 25.3±1.6 kgm-2 for those with zero, one or two comorbidities respectively. The SBP levels for those with zero, one, two comorbidities were 124.0±4.0 mmHg, 131.4±5.7 mmHg and 143.4±7.7 mmHg respectively. However, DBP showed no significant difference (p=0.128).

From figure 2, serum creatinine (CRT) ($F_{3,44}$ = 0.7791; p = 0.512) and eGFR ($F_{3,42}$ = 0.1953; p = 0.899) showed no significant difference for trend. For those having zero, one, two or at least three or more comorbidities, the eGFR levels were 108.3±28.4 ml/min/ 1.73 m2, 87.5±20.6 ml/ min/1.73 m2, 86.4±17.7 ml/min/1.73 m2 and 99.7±24.2 ml/min/1.73 m2 respectively. The serum CRT levels were 216.6±8.1 μmolL-1, 311.6±103.7 μmolL-1, 485.8±159.9 μmolL-1 and 263.3±122.3 μmolL-1 for those with zero, one, two and at least three or more comorbidities respectively.

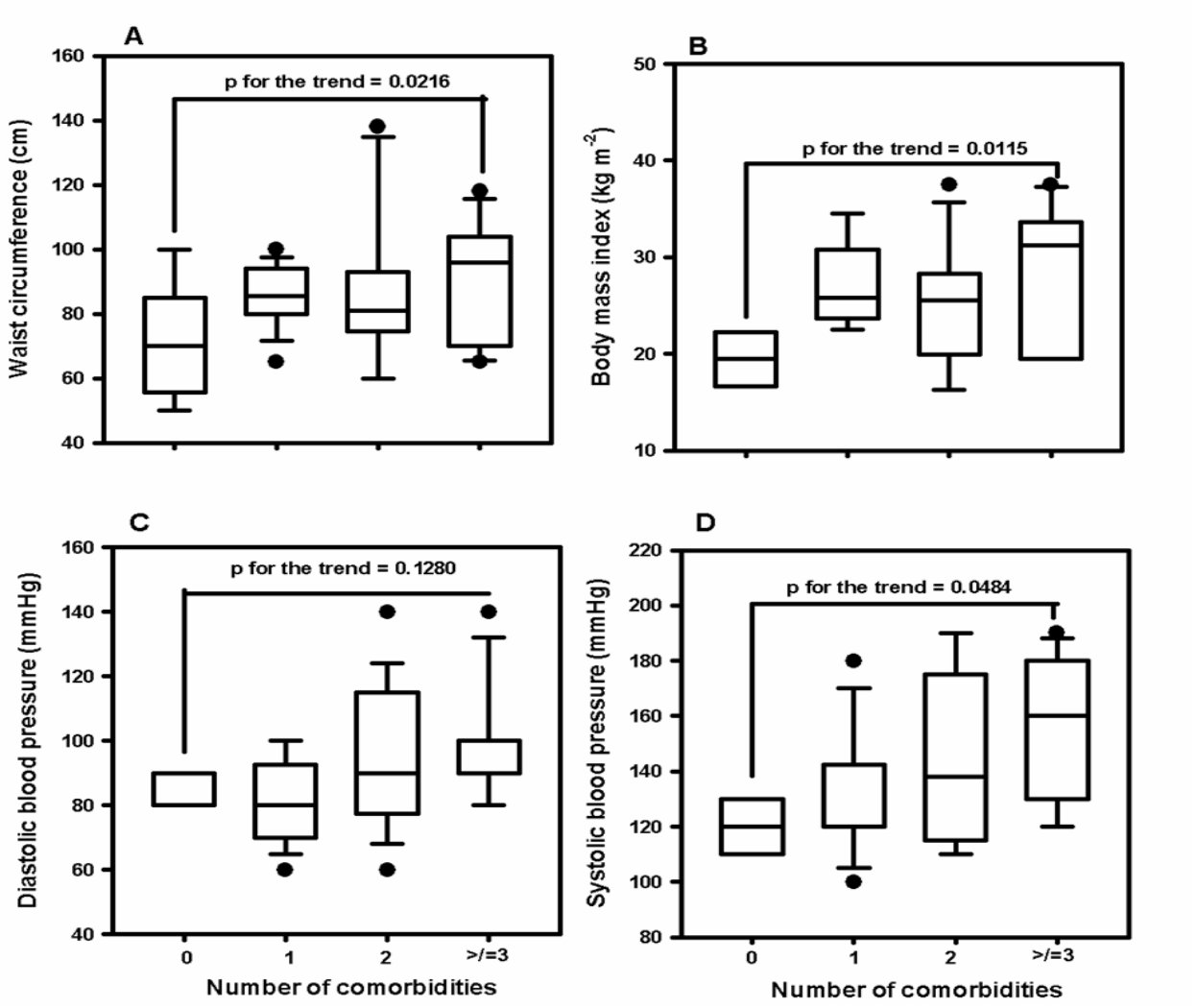


**Figure 1: Comparisons of BMI, DBP, SBP and WC between participants with a different number of comorbidities of the MetS in CKD. The lower and upper margins of the box represent the 25th and 75th percentiles, with the extended arms representing the 10th and 90th percentiles, respectively. The median is shown as the horizontal line within the box. Outlying points are shown individually**.

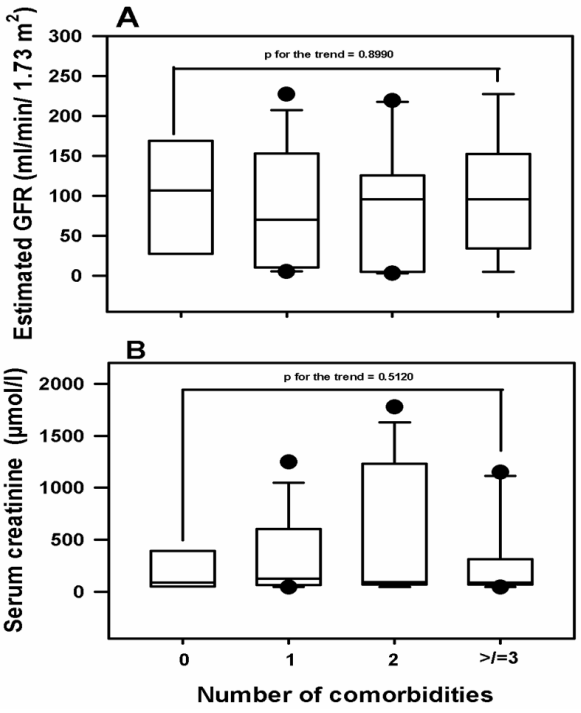


**Figure 2: Comparisons of eGFR and serum Creatinine between participants with different number of comorbidities of MetS in CKD. The lower and upper margins of the box represent the 25th and 75th percentiles, with the extended arms representing the 10th and 90th percentiles, respectively. The median is shown as the horizontal line within the box. Outlying points are shown individually**.

Many of the participants had multiple comorbidities; and those with a greater number of comorbidities also had higher TG ($F_{3,45}$ = 3.593; p = 0.027) and lower HDL-C ($F_{3,46}$ = 5.573; p = 0.002). However, FBG ($F_{3,44}$= 1.533; p = 0.219) and TC ($F_{3,46}$ = 0.403; p = 0.751) showed no significant difference for trend. The TG levels were 1.2±0.5 $mmolL^{-1}$, 1.4±0.2 $mmolL^{-1}$, 2.4±0.4 $mmolL^{-1}$ or 2.7±0.3 $mmolL^{-1}$ for those with zero, one, two, and at least three or more comorbidities respectively. The low HDL-C levels for those with zero, one, two or and least three or more comorbidities were 1.6±0.3 $mmolL^{-1}$, 1.8±0.2 $mmolL^{-1}$, 1.1±0.1 $mmolL^{-1}$ or 1.0±0.1 $mmolL^{-1}$ respectively (Figure 3).

**Risk factors of developing MetS among the various CKD group**

Table 3 represents the odds ratios of MetS risk factors at various stages of CKD. When participants with CKD were classified into the various stages, the risk of developing hypertension decreased from about 10 times in stage 1 to about 7 times in stage 2 before increasing to about 9 times for stage 3, decreased to 6 times in stage 4 and increased to about 14 times in stage 5. The risk of having hyperglycaemia also increased from stage 1 to stage 3, and then decreased in stage 4 and 5, whereas the risk of developing obesity remained fairly stable throughout the various stages (1-5). The risk of developing low HDL-C decreased from stage 1 to stage 2 before increasing in stage 3, with a further decrease in stage 4, and finally increasing again at stage 5. The risks of developing hypertriglyceridaemia slightly increased progressively reaching the highest value at stage 5. MetS risk increased and reached a peak at stage 3, and decreased at stage 4 before finally increasing again at stage 5. The risk of developing proteinuria from this study fluctuated through the stages reaching a value greater than the initial value at stage 5 (Table 3).

## DISCUSSION

This randomized case-controlled study sought to determine the prevalence of MetS and the relationship between the components of MetS and CKD in a Ghanaian population presenting with various stages of CKD. This study indicated the prevalence of MetS as defined by the NCEP ATP III criteria to be 30.1% of the participants. This finding is consistent with studies done in Australia (31%), Thailand (30.1%) and 34.1% in over 40 year olds in China but slightly lower than what was reported in Bangladesh (37%) (Johnson *et al.*, 2007; Zhang *et al.*, 2007; Satirapoj *et al.*, 2011; Nath *et al.*, 2012). This could be attributed to differences in the selection of participants, the MetS definition used and also the fact that MetS is an independent factor for CKD development. The current study also observed a high prevalence of MetS in female CKD participants compared to male CKD participants. This is consistent with observations made in numerous studies including the Virgem das Graças

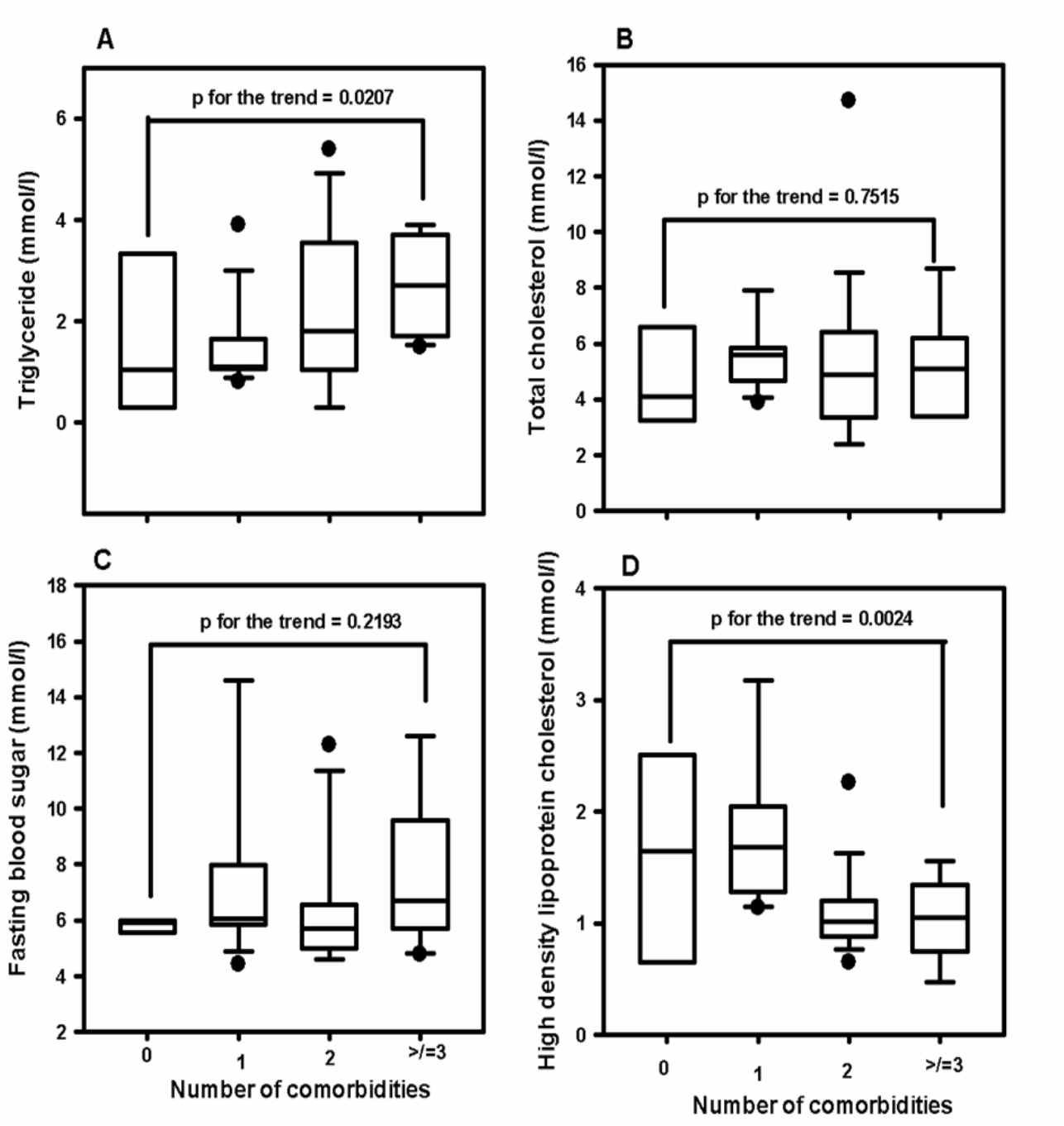


***Figure 3: Comparisons of FBG, TG, TC and HDL-C between participants with different number of comorbidities of MetS in CKD. The lower and upper margins of the box represent the 25th and 75th percentiles, with the extended arms representing the 10th and 90th percentiles, respectively. The median is shown as the horizontal line within the box. Outlying points are shown individually.***

**Table 3: Odds ratios of MetS risk factors at various stages of CKD**

| Parameter | Stage 1 (n=24) | OR (95% CI) | Stage 2 (n=35) | OR (95% CI) | Stage 3 (n=37) | OR (95% CI) | Stage 4 (n=25) | OR (95% CI) | Stage 5 (n=24) | OR (95% CI) |
|---|---|---|---|---|---|---|---|---|---|---|
| Hypertension | 8(33.3%) | 9.5(2.5-35.4) | 9(25.7%) | 6.6(1.8-23.2) | 12(32.4%) | 9.1(2.7-30.8) | 6(24.0%) | 6.0(1.5-23.4) | 10(41.6%) | 13.6(3.7-49.4) |
| FGB | 13(54.1%) | 5.5(2.1-15.0) | 26(74.3%) | 13.6(5.2-35.3) | 28(75.6%) | 14.6(5.7-37.8) | 18(72.0%) | 12.1(4.2-34.5) | 12(50.0%) | 4.7(1.7-12.6) |
| Obesity | 5(20.8%) | 1.3(0.4-4.3) | 8(22.8%) | 1.5(0.5-4.1) | 9(24.3%) | 1.6(0.6-4.3) | 10(40.0%) | 3.4(1.2-9.3) | 4(16.7%) | 1.0(0.3-3.5) |
| TG | 10(41.6%) | 1.8(0.7-4.8) | 18(51.4%) | 2.8(1.2-6.4) | 18(48.6%) | 2.5(1.1-5.6) | 10(40.0%) | 1.7(0.7-4.5) | 13(54.1%) | 3.1(1.2-8.0) |
| Low HDL | 4(16.7%) | 3.8(0.8-16.5) | 5(14.3%) | 1.9(0.8-12.6) | 11(29.7%) | 8.0(2.3-27.4) | 5(20.0%) | 4.7(1.2-19.3) | 7(29.1%) | 7.8(2.0-29.8) |
| Proteinuria | 5(20.8%) | 45.0(2.4-857) | 12(48.0%) | 149(8.3-2671) | 12(32.4%) | 79(4.5-1381) | 10(40%) | 109(6.0-1961) | 7(29.1%) | 69(3.7-1266) |
| MetS | 6(25.0%) | 8.5(1.9-37.5) | 13(37.1%) | 15.1(3.9-58.0) | 13(35.1%) | 14.0(3.6-52.9) | 4(16.0%) | 4.8(1.0-23.5) | 8(33.3%) | 12.8(3.0-53.7) |

***Stage 1=eGFR ≥90 mL/min/1.73m²; stage 2 = eGFR 60-89 mL/min/1.73m²; stage 3 = eGFR 30-59 mL/min/1.73m²; stage 4 =eGFR 16-29 mL/min/1.73m²; stage 5 = eGFR<15 mL/min/1.73m² TG=triglycerides; TC=total cholesterol; HDL=high density lipoprotein; FGB=fasting blood glucose; OR=odds ratio.***

community study (Dallongeville *et al.*, 2004) and that of Nath *et al.*, (2012) who reported prevalence rates of 32.35 and 42.5% for males and females respectively in a cross-sectional study involving 300 CKD patients in Bangladesh.

High TG and low HDL cholesterol have been identified as independent risk factors for initiation and progression of CKD (Fried *et al.*, 2001). However, in this study increased TG but not low HDL-C was predictive of CKD development as observed in earlier studies by Luk *et al.*, (2008). The processes underlying the role of lipids in the initiation of renal injury have not been fully elucidated.

In the current study, obesity was defined using the NCEP ATP III criteria for diagnosis of MetS and measured WC to determine abdominal obesity. Participants with MetS and CKD also had significantly higher WC a finding consistent with observations made in other studies (Kwan *et al.*, 2007; Chou *et al.*, 2008). The strong association between MetS and renal damage can be explained in the light of the role played by obesity related glomerulopathy. Even though the mechanism by which waist circumference increase the risk of CKD has not been well explained, it has been linked with the production of inflammatory cytokines like leptin, interlukin-6 (IL-6) tumour necrotic factor-alpha (TNF-alpha) and adiponectin (Satirapoj and Supasyndh, 2007). These cytokines, mostly produced by the adipose tissue, play a role in kidney damage in patients with MetS by activating sympathetic nervous activity, aggravating renal haemodynamics, in addition to increasing inflammatory and oxidative states (Iseki, 2008).

High systolic blood pressure is prevalent in CKD as observed among the CKD subjects with MetS in this study. High systolic blood pressure is a determinant of CKD progression and should therefore be the focus of control of antihypertensive therapy (Young *et al.*, 2002). The association of CKD with isolated systolic hypertension (and wide pulse pressure) may be explained by increased vascular stiffness. Wide pulse pressure appears to be a marker of vascular stiffness and cardiovascular calcification, a predictor of cardiovascular risk in the elderly (Bielak

*et al.*, 2004) and it is associated with increased mortality in patients with renal disease (Klassen *et al.*, 2002).

The relationship between the MetS and the incidence of CKD is that of MetS components directly causing harm to the kidneys through systemic atherosclerosis. Individual components of MetS, including glucose intolerance, hypertension and dyslipidaemia, could act directly as risk factors for renal injury through renal or systemic atherosclerosis according to previous epidemiological studies (Humphrey *et al.*, 1989; Whelton *et al.*, 1996; Hunsicker *et al.*, 1997). In the present study, it was found that clusters of these risk factors had a stronger impact on the development of CKD than individual risk factors. Additionally, the accumulation of three or more of the metabolic disorders outlined by the NCEP ATP III criteria promoted the development of CKD or progression of GFR decline. These findings support the hypothesis that clusters of atherogenic metabolic disorders induce renal vessel injury, resulting in deterioration of renal function (Ninomiya *et al.*, 2006).

## CONCLUSION

The prevalence of MetS in CKD patients was 30.1% using the NCEP ATP III criteria and increased WC, TG and SBP are components of the metabolic syndrome which contribute to the initiation and progression of CKD. A critical assessment of Met S and its components should be included in the monitoring and management scheme of CKD patients in order to reduce its prevalence and thus control the progression of CKD.

## ACKNOWLEDGEMENT

The authors are grateful to the staff of the Laboratory Department, Tamale Teaching Hospital and the Department of Clinical Biochemistry, KATH for their technical assistance.

## COMPETING INTERESTS

The authors declare that they have no competing interests.

## REFERENCES

Barham D. and Trinder P. (1972) An improved colour reagent for the determination of blood glucose by the oxidase system. *Analyst ,* 97, 142-145.

Bielak L.F., Turner S.T., Franklin S.S., Sheedy P.F., 2nd and Peyser P.A. (2004) Age-dependent associations between blood pressure and coronary artery calcification in asymptomatic adults. *J Hypertens* 22, 719-725.

Chen J., Muntner P., Hamm L.L., Jones D.W., Batuman V., Fonseca V., Whelton P.K. and He J. (2004) The metabolic syndrome and chronic kidney disease in U.S. adults. *Ann Intern Med* 140, 167-174.

Chou C.Y., Lin C.H., Lin C.C., Huang C.C., Liu C.S. and Lai S.W. (2008) Association between waist-to-hip ratio and chronic kidney disease in the elderly. *Intern Med J* 38, 402-406.

Coresh J., Astor B.C., Greene T., Eknoyan G. and Levey A.S. (2003) Prevalence of chronic kidney disease and decreased kidney function in the adult US population: Third National Health and Nutrition Examination Survey. *Am J Kidney Dis* 41, 1-12.

Dallongeville J., Cottel D., Arveiler D., Tauber J.P., Bingham A., Wagner A., Fauvel J., Ferrieres J., Ducimetiere P. and Amouyel P. (2004) The association of metabolic disorders with the metabolic syndrome is different in men and women. *Ann Nutr Metab* 48, 43-50.

Ford E.S. (2005) Risks for all-cause mortality, cardiovascular disease, and diabetes associated with the metabolic syndrome: a summary of the evidence. *Diabetes Care* 28, 1769-1778.

Fried L.F., Orchard T.J. and Kasiske B.L. (2001) Effect of lipid reduction on the progression of renal disease: a meta-analysis. *Kidney Int* 59, 260-269.

Friedewald W.T., Levy R.I. and Fredrickson D.S. (1972) Estimation of the concentration of low-density lipoprotein cholesterol in plasma, without use of the preparative ultracentrifuge. *Clin Chem* 18, 499-502.

Humphrey L.L., Ballard D.J., Frohnert P.P., Chu C.P., O'Fallon W.M. and Palumbo P.J. (1989) Chronic renal failure in non-insulin-dependent diabetes mellitus. A population-based study in Rochester, Minnesota. *Ann Intern Med* 111, 788-796.

Hunsicker L.G., Adler S., Caggiula A., England B.K., Greene T., Kusek J.W., Rogers N.L. and Teschan P.E. (1997) Predictors of the progression of renal disease in the Modification of Diet in Renal Disease Study. *Kidney Int* 51, 1908-1919.

Iseki K. (2008) Metabolic syndrome and chronic kidney disease: a Japanese perspective on a worldwide problem. *J Nephrol* 21, 305-312.

Iseki K., Ikemiya Y., Kinjo K., Inoue T., Iseki C. and Takishita S. (2004) Body mass index and the risk of development of end-stage renal disease in a screened cohort. *Kidney Int* 65, 1870 -1876.

Johnson D.W., Armstrong K., Campbell S.B., Mudge D.W., Hawley C.M., Coombes J.S., Prins J.B. and Isbel N.M. (2007) Metabolic syndrome in severe chronic kidney disease: Prevalence, predictors, prognostic significance and effects of risk factor modification. *Nephrology (Carlton)* 12, 391-398.

Kambham N., Markowitz G.S., Valeri A.M., Lin J. and D'Agati V.D. (2001) Obesity-related glomerulopathy: an emerging epidemic. *Kidney Int* 59, 1498-1509.

Kirkendall W.M., Burton A.C., Epstein F.H. and Freis E.D. (1967) Recommendations for human blood pressure determination by sphygmomanometers. *Circulation* 36, 980-988.

Klassen P.S., Lowrie E.G., Reddan D.N., DeLong E.R., Coladonato J.A., Szczech L.A., Lazarus J.M. and Owen W.F., Jr. (2002) Association between pulse pressure and mortality in patients undergoing maintenance hemodialysis. *JAMA* 287, 1548-1555.

Kurella M., Lo J.C. and Chertow G.M. (2005) Metabolic syndrome and the risk for chronic kidney disease among nondiabetic adults. *J Am Soc Nephrol* 16, 2134-2140.

Kwan B.C., Murtaugh M.A. and Beddhu S. (2007) Associations of body size with metabolic syndrome and mortality in moderate chronic kidney disease. *Clin J Am Soc Nephrol* 2, 992-998.

Luk A.O., So W.Y., Ma R.C., Kong A.P., Ozaki R., Ng V.S., Yu L.W., Lau W.W., Yang X., Chow F.C., Chan J.C. and Tong P.C. (2008) Metabolic syndrome predicts new onset of chronic kidney disease in 5,829 patients with type 2 diabetes: a 5-year prospective analysis of the Hong Kong Diabetes Registry. *Diabetes Care* 31, 2357-2361.

Muntner P., He J., Hamm L., Loria C. and Whelton P.K. (2002) Renal insufficiency and subsequent death resulting from cardiovascular disease in the United States. *J Am Soc Nephrol* 13, 745-753.

Nath R., Mridha M., Sarker K., Mollah F., SFerdousi S. and Rahman M. (2012) Metabolic Syndrome in Chronic Kidney Disease Patients. *Dinajpur Med Col J* 1, 34-38.

National Kidney Foundation (2002) K/DOQI clinical practice guidelines for chronic kidney disease: evaluation, classification, and stratification. *Am J Kidney Dis* 39, S1-266.

NCEP (2001) Executive Summary of The Third Report of The National Cholesterol Education Program (NCEP) Expert Panel on Detection. *JAMA* 285, 2486–2497.

Ninomiya T., Kiyohara Y., Kubo M., Yonemoto K., Tanizaki Y., Doi Y., Hirakata H. and Iida M. (2006) Metabolic syndrome and CKD in a general Japanese population: the Hisayama Study. *Am J Kidney Dis* 48, 383-391.

NKF/KDOQI™ N.K.F. (2002) Clinical practice guidelines for chronic kidney disease. *American Journal of Kidney Diseases*, S1 - S266.

Owiredu W.K.B.A., Ephraim R.K.D., Amidu N., Eghan Jnr B.A. and Quaye L. (2008) Predictive Performance of Renal Function Equations Among Ghanaians Presenting with Chronic Kidney Disease. *J. Med. Sci* 8, 491-497.

Reynolds K. and He J. (2005) Epidemiology of the metabolic syndrome. *Am J Med Sci* 330, 273 -279.

Satirapoj B. and Supasyndh O. (2007) Insulin resistance and the kidney. *J Nephrol Soc Thai* 13, 20-27.

Satirapoj B., Supasyndh S., Mayteedol N., Chaiprasert A. and Choovichian P. (2011) Metabolic syndrome and its relation to chronic kidney disease in a South East Asian population *South East Asian J Trop Med Public Health* 42 176-183.

Trinder P. (1969) Determination of blood glucose using an oxidase peroxidase system with a non- carcinogenic chromogen. *J Clin Pathol.* 22, 158-161.

Whelton P.K., Perneger T.V., He J. and Klag M.J. (1996) The role of blood pressure as a risk factor for renal disease: a review of the epidemiologic evidence. *J Hum Hypertens* 10, 683-689.

Young J.H., Klag M.J., Muntner P., Whyte J.L., Pahor M. and Coresh J. (2002) Blood pressure and decline in kidney function: findings from the Systolic Hypertension in the Elderly Program (SHEP). *J Am Soc Nephrol* 13, 2776-2782.

Zhang L., Zuo L., Wang F., Wang M., Wang S., Liu L. and Wang H. (2007) Metabolic syndrome and chronic kidney disease in a Chinese population aged 40 years and older. *Mayo Clin Proc* 82, 822-827.

ISSN 2026-6294