Open Science Journal of Clinical Medicine
2015; 3(4): 129-135
Published online July 10, 2015 (http://www.openscienceonline.com/journal/osjcm)

Urine Examination and Renal Function in Adult Patients Attending Outpatient Clinics in Kumasi: Cross-Sectional Study

Frank B. Micah*, Bernard C. Nkum

Department of Medicine, School of Medical Sciences, Kwame Nkrumah University of Science and Technology, Kumasi, Ghana

Email address

fbmicah@hotmail.com (F. B. Micah)

To cite this article

Frank B. Micah, Bernard C. Nkum. Urine Examination and Renal Function in Adult Patients Attending Outpatient Clinics in Kumasi: Cross-Sectional Study. *Open Science Journal of Clinical Medicine.* Vol. 3, No. 4, 2015, pp. 129-135.

Abstract

Background: Early identification and treatment of kidney diseases in adults are important in prevention of chronic kidney diseases. *Objective*: To determine the prevalence of abnormal urine dipstick findings, abnormal urine microscopy and chronic kidney diseases (CKD) among patients reporting at KomfoAnokye Teaching Hospital (KATH), Kumasi, Ghana. *Design*: Cross-sectional study *Setting*: Directorate of Medicine and Polyclinic outpatient clinics of KATH. *Methods*: A total of 424 patients, 20 years and over reporting for the first time to KATH were recruited, 414 were included in the analysis and 10 were excluded. 76 had neither diabetes mellitus (DM) nor systemic hypertension 92 had only DM, 106 had only hypertension and 140 had both. A questionnaire was filled and anthropometric measurements were taken. Serum creatinine was determined from venous blood samples and urine dipsticks and microscopy were done. The estimated glomerular filtration rate (eGFR) was calculated using the Cockcroft and Gault equation. CKD was defined and classified by The National Kidney Foundation's Kidney Diseases Outcomes Quality Initiative guidelines. *Results*: The prevalence of proteinuria was 16%, glycosuria was 16%, abnormal urine microscopy 6%, haematuria 1% and CKD 39%. There were no cases of Stage 5 CKD. The mean (sd) creatinine was 98.61 (27.30) µmol/L, mean urea was 4.41 (1.71) mmol/L and mean eGFR was 118.5 (57.5)ml/min/1.73 m^2. There were significantly more males than females and more hypertensives than normotensives with CKD. *Conclusion*: The prevalence of proteinuria and CKD in patients attending clinics in Kumasi, Ghana was high. There is the need for more and regular screening of patients especially those with hypertension and DM.

Keywords

Systemic Hypertension, Chronic Kidney Disease, Renal Failure, Proteinuria

1. Introduction

The global epidemic of chronic kidney disease (CKD) presents a challenge and a major public health problem for high-income developed countries, as well as developing countries. It is worsened by the enormous disease burden of hypertension and the high incidence of diabetes in developing countries. [1] Chronic kidney disease may be too covert for early detection and its occurrence is a worldwide health problem. [2] A major question for renal medicine in developing countries is how to define strategies that can identify early enough those subjects who are at risk of developing a renal disease later in life. This will make it possible to design population-oriented preventive measures that will limit the need for dialysis and transplantation.

The simplest and least expensive way of screening apparently healthy subjects for CKD is urinalysis and several studies have been done using reagent strips, documenting their effectiveness in detecting urinary abnormalities at relatively low cost. [3] Urine testing has been a part of medicine for many centuries, with Hippocrates having written about urine examination as early as 400 BC. Advances in chemistry allowed significant progress in urine testing during the nineteenth century, and the modern era of reagent strip (dipstick) testing began in 1956. [4] It is common to find abnormalities on urine screening tests, but in most cases, these are transient or due to a false positive reading. [5] Screening can also identify individuals who have subclinical chronic kidney disease who may potentially benefit from early identification.

Proteinuria is defined as urinary protein excretion of more

than 300 mg per day and is the hallmark of renal disease. Microalbuminuria is defined as the excretion of 30 to 300 mg of protein per day and is a sign of early renal disease. [6] Proteinuria on initial dipstick urinalysis is found in as much as 17 percent of selected populations. Asymptomatic proteinuria is associated with significant renal disease in less than 1.5 percent of patients. [7] Proteinuria can be classified as transient or persistent. [8] The pathophysiologic mechanisms of proteinuria can be classified as glomerular, tubular or overflow. [7] The presence of relatively low levels of urine protein can be an early marker of increased risk of progressive kidney disease, poor cardiovascular outcomes, and death. [9]

End stage renal disease is usually the result of slowly progressive kidney damage. Due to the asymptomatic nature of renal disease, kidney damage frequently remains undetected until late in the course, at which stage therapeutic interventions are often ineffective. In contrast, early detection and intervention may slow or halt the decline toward end stage renal disease. [10] Early identification and treatment of kidney diseases in adults are therefore important initial steps in prevention of chronic kidney diseases. The presence of kidney damage may be indicated by proteinuria, hematuria, or reduced glomerular filtration rate. [11] In screening for CKD, the current guidelines recommend using the estimated glomerular filtration rate (eGFR) after determining serum creatinine levels and albuminuria. [12]

As part of our lipids study in Kumasi, [13] we determined the renal function and urine microscopy for the participants and in this paper we report these findings. The aim of this study was to determine the prevalence of abnormal urine dipstick findings, abnormal urine microscopy and CKD.

2. Materials and Methods

The detailed methodology has been previously reported in the article on our original study. [13] This was a cross sectional study conducted at the KATH, Kumasi from April 2008 to January 2009. The recruitment of the study participants was at the Directorate of Medicine and the Polyclinic Directorate of the hospital. The inclusion criteria were all patients reporting during the study period who were 20 years and above, who were treated on outpatient basis and who consented to participate by completing the informed consent form. The exclusion criteria were refusal to give consent, liver cirrhosis and inability to stand for weight and height measurement.

One of the authors administered a questionnaire and carried out a physical examination on the participants. Height was measured to the nearest 0.5 cm and weight was measured to the nearest 0.1 kg after participants had removed their footwear using a standardized combined manual scale and stadiometer (Asimed MB 201T Plus from Aparatos Y Sistemas De Medida, S. A.). Hip and waist circumferences were measured to the nearest 0.5 cm using a plastic tape measurement. An automatic BP machine (OMRON M7 sphygmomanometer; Omron Matsusaka Co. Ltd, Matsusaka City, Mie-Ken, Japan) was used to measure the blood pressure (BP) and pulse rate. Three readings were taken 1 min apart but the mean of the last two readings was used in the data analysis having discarded the first reading.

Each participant was treated appropriately but had to report to the hospital the following day after an overnight fast for urine examination and biochemical investigations which included fasting lipid profile, liver function test, fasting blood glucose (FBG), urea, creatinine and electrolytes. The patient first provided about 10 to 20mls of urine in a sterile wide-necked leak proof urine specimen container. This was immediately tested with Multistix urine dipsticks (Siemens AG, Germany) after which urine microscopy examination was done. Ten mls of venous blood samples were then collected for the biochemistry tests, which were undertaken using a BT3000 auto analyser, manufactured by Biotechnica Instruments S.p.A. Rome, Italy. Serum creatinine was measured using a Jaffe alkaline picrate method.

The following definitions were adopted for this study. Systemic hypertension was defined as systolic blood pressure (SBP) ≥ 140 and / or diastolic blood pressure (DBP) ≥ 90 mmHg in subjects who are not taking antihypertensive medication or being on drug therapy for hypertension. [14] Diabetes mellitus (DM) as fasting venous blood glucose ≥ 6.1 mmol / L and or 2h post glucose capillary whole blood ≥ 11.1 mmol / L or being on drug or diet therapy for diabetes. [15], [16] Overall Obesity was Body Mass Index (BMI) ≥30 kg / m ^{2} and Central Obesity or High Waist Hip Ratio (WHR), WHR> 0.9 for males and > 0.8 for females. [17]

The estimated glomerular filtration rate (eGFR) was calculated using the Cockcroft and Gault equation; eGFR (ml/min) = 1.23 (140 - age) × weight (kg)/ Plasma creatinine (μmol/l) for males and eGFR (ml/min) = 1.04 (140 - age) × weight (kg)/ Plasma creatinine (μmol/l) for females. [18] The calculated eGFR were converted from ml/min to ml/min/1.73 m^2 by multiplying calculated values by 1.73, and dividing by body surface area (BSA). The BSA was calculated from the Mosteller formula [19]

$$BSA = \frac{\sqrt{\mathrm{W} - \mathrm{Ht}}}{6}$$

Here W is weight in kg and Ht is height in m.

The National Kidney Foundation's Kidney Diseases Outcomes Quality Initiative (NKF KDOQI) guidelines was used in defining and classifying CKD. This classification is based on GFR and the presence or absence of kidney damage. The reduced GFR and or kidney damage must be present for more than 90 days to establish chronicity. In the absence of past data on GFR or markers of kidney damage, chronicity is inferred from clinical presumption of kidney disease for more than 3 months. Based on this assumption CKD was classified into Stage 1 GFR > 90 ml/min/1.73 m^2 and albuminuria, Stage 2 GFR 60 - 89 ml/min/1.73 m^2 and albuminuria, Stage 3 GFR 30 - 59 ml/min/1.73 m^2, Stage 4 GFR 15 - 29 ml/min/1.73 m^2 and Stage 5 GFR < 15 ml/min/1.73 m^2. [20]

The urine dipsticks reported urine glucose results as absent, +1, +2, and +3 and for the purpose of analysis urine glucose was defined as any positive urine glucose. The urine protein

was also reported as absent, +1, +2, and +3 and for this analysis proteinuria was defined as any positive urine protein on examination. Abnormal microscopy was defined as participants with urine white blood cells >10 per high power field or red blood cells >10 per high power field or epithelia cells >10 per high power field or those with any combination of these abnormal findings.

The data was analysed using Stata version 8.0 statistical package and Microsoft Excel 2010. The mean and standard deviation were calculated for continuous variables, and were compared using the Student t-test. The results for eGFR were not normally distributed so the median with the interquartile range were additionally reported. Percentages were calculated for discrete variables and these were compared using Pearson Chi-square test. P-values of less than 0.05 were taken as statistically significant. The participants were classified into normotensives with and without DM and hypertensives with and without DM and these four subgroups were captioned as the clinical group.

The study was approved by the Committee of Human Research, Publication and Ethics of KATH and School of Medical Sciences, Kwame Nkrumah University of Science and Technology. The study was thoroughly explained to each participant after which they gave formal consent by signing or thumb printing an informed consent form.

3. Tables

Table 1. *The clinical characteristics of participants by sex.*

	Male	Female	All	P
	Mean (SD))	Mean (SD)	Mean (SD)	(t or χ2 test)
Number (%)	159 (38.4)	255 (61.6)	414	
Age (years)	48 (14)	51 (13)	50 (14)	0.11 (t)
Age range (years)	21 - 88	20 - 83	20 - 88	
Weight (kg)	68.8 (11.9)	68.3 (15.1)	68.5 (13.9)	0.71 (t)
Height (m)	1.68 (0.06)	1.58 (0.06)	1.62 (0.08)	<0.0001 (t)
BMI (kg/m2)	24.3 (3.7)	27.3 (5.6)	26.2 (5.1)	<0.0001 (t)
WHR	0.90 (0.07)	0.90 (0.11)	0.90 (0.09)	0.95 (t)
SBP (mmHg)	134 (21)	132 (22)	133 (22)	0.37 (t)
DBP (mmHg)	84 (11)	84 (12)	84 (12)	1.00 (t)
BMI≥30 (%)	9 (5.7)	84 (32.9)	93 (22.5)	<0.001 (χ2)
HIGH WHR (%)	81 (50.9)	223 (87.5)	304 (73.4)	<0.001 (χ2)
ONLY DM (%)	36 (22.6)	56 (22.0)	92 (22.2)	0.87 (χ2)
ONLY HPT (%)	36 (22.6)	70 (27.5)	106 (25.6)	0.28 (χ2)
DM-HPT (%)	54 (34.0)	86 (33.7)	140 (33.8)	0.96 (χ2)

Table 2. *Fasting blood glucose and renal function tests results by sex.*

	Male Mean (SD)	Female Mean (SD)	All Mean (SD)	P (t test)
Number (%)	159 (38.4)	255 (61.6)	414	
FBG (mmol/L)	7.89 (4.43)	8.21 (4.52)	8.09 (4.48)	0.48
Urea (mmol/L)	4.48 (1.58)	4.36 (1.77)	4.41 (1.71)	0.48
Creatinine (μmol/L)	111.10 (25.55)	90.92 (25.47)	98.61 (27.30)	<0.0001
eGFR (ml/min/1.73 m2)	62.10 (19.91)	153.11 (72.86)	118.45 (57.5)	0.12

Table 3. *Estimated glomerular filtration rate by sex.*

	Male Number (%)	Female Number (%)	All Number (%)	P (χ2 test)
Number (%)	159 (38.4)	255 (61.6)	414	
eGFR (ml/min/1.73 m2)				
> 90	4 (2.5)	115 (45.1)	119 (28.7)	<0.00001
60 - 89	79 (49.7)	95 (37.3)	174 (42.0)	0.01
30 - 59	72 (45.3)	44 (17.3)	116 (28.0)	<0.00001
15 - 29	4 (2.5)	1 (0.3)	5 (1.3)	0.05
< 15	0	0	0	N/A

Table 4. *Estimated glomerular filtration rate by clinical groups.*

	No DM No Hypertension Number (%)	DM Number (%)	Hypertension Number (%)	DM - Hypertension Number (%)	All Number (%)	P (χ2 test)
Number (%)	76 (18.4)	92 (22.2)	106 (25.6)	140 (33.8)	414	
eGFR ml/min/1.73 m2						
> 90	26 (34.2)	40 (43.5)	17 (16.0)	36 (25.7)	119 (28.7)	<0.001
60 - 89	29 (38.2)	40 (43.5)	52 (49.1)	53 (37.9)	174 (42.0)	0.30
30 - 59	21 (27.6)	12 (13.0)	35 (33.0)	48 (34.3)	116 (28.0)	<0.01
15 - 29	0 (0)	0 (0)	2 (1.9)	3 (2.1)	5 (1.3)	0.32
< 15	0 (0)	0 (0)	0 (0)	0 (0)	0 (0)	N/A

Table 5. *Urine analysis results by sex.*

	Male Number (%)	Female Number (%)	All Number (%))	P (χ2 test)
Number (%)	159 (38.4)	255 (61.6)	414	
Urine Glucose	29 (18.2)	38 (14.9)	67 (16.2)	0.37
Urine Protein	26 (16.4)	40 (15.7)	66 (15.9)	0.86
Abnormal Microscopy	6 (3.8)	17 (6.7)	23 (5.6)	0.21

Table 6. *Urine analysis results by clinical groups.*

	No DM No Hypertension Number (%)	DM Number (%)	Hypertension Number (%)	DM - Hypertension Number (%)	All Number (%)	P (χ2 test)
Number (%)	76 (18.4)	92 (22.2)	106 (25.6)	140 (33.8)	414	
Urine Glucose	0 (0)	32 (34.8)	0 (0)	35 (16.2)	67 (16.2)	<0.001
Urine Protein	11 (14.5)	15 (16.3)	14 (13.2)	26 (18.6)	66 (15.9)	0.70
Abnormal Microscopy	4 (5.3)	4 (4.4)	2 (1.9)	13 (9.3)	23 (5.6)	0.08

Table 7. *Urine analysis results by estimated glomerular filtration rate.*

	> 90 Number (%)	60 - 89 Number (%)	30 - 59 Number (%)	15 - 29 Number (%)	< 15 Number (%)	All Number (%)	P (χ^2 test)
Number (%)	119 (28.7)	174 (42.0)	116 (28.0)	5 (1.3)	0 (0)	414	
Urine Glucose	21 (21.6)	28 (16.1)	18 (15.5)	0 (0)	0 (0)	67 (16.2)	0.75
Urine Protein	20 (16.8)	20 (11.5)	21 (18.1)	5 (100.0)	0 (0)	66 (15.9)	<0.00001
Abnormal Microscopy	6 (5.0)	7 (4.0)	10 (8.6)	0 (0)	0 (0)	23 (5.6)	0.24

Table 8. *Prevalence of CKD by sex.*

	Male Number (%)	Female Number (%)	All Number (%))	P (χ^2 test)
Number (%)	159 (38.4)	255 (61.6)	414	
CKD				
Stage 1	2 (1.3)	18 (7.1)	20 (4.8)	<0.01
Stage 2	10 (6.3)	10 (3.9)	20 (4.8)	0.27
Stage 3	72 (45.3)	44 (17.3)	116 (28.0)	<0.00001
Stage 4	4 (2.5)	1 (0.3)	5 (1.3)	0.05
Stage 5	0	0	0	N/A
All Stages	88 (55.3)	73 (28.6)	161 (38.9)	<0.00001

Table 9. *Prevalence of CKD by clinical groups.*

	No DM No Hypertension Number (%)	DM Number (%)	Hypertension Number (%)	DM - Hypertension Number (%)	All Number (%)	P (χ^2 test)
Number (%)	76 (18.4)	92 (22.2)	106 (25.6)	140 (33.8)	414	
CKD						
Stage 1	1 (1.3)	7 (7.6)	2 (1.9)	10 (7.1)	20 (4.8)	0.07
Stage 2	4 (5.3)	5 (5.4)	4 (3.8)	7 (5.0)	20 (4.8)	0.95
Stage 3	21 (27.6)	12 (13.0)	35 (33.0)	48 (34.3)	116 (28.0)	<0.01
Stage 4	0 (0)	0 (0)	2 (1.9)	3 (2.1)	5 (1.3)	0.32
Stage 5	0 (0)	0 (0)	0 (0)	0 (0)	0 (0)	N/A
All Stages	26 (34.2)	24 (26.1)	43 (40.6)	68 (48.6)	161 (38.9)	<0.01

4. Results

In all 424 subjects participated in our original study, [13] however the data of 414 (97.6%) participants were included in the analysis and the results are presented in this paper. The reasons for excluding the 10 participants were unavailability of biochemistry and urine results. There were 159 (38%) males and 255 (62%) females giving a male: female ratio of 1:1.6.

The mean (sd) age for the participants was 50 (14) years with no significant age difference between the males and females (Table 1). The mean weight was 68.5 (13.9) kg, mean BMI 26.2 (5.1) while mean SBP and DBP were133 (22) and 84 (12) mmHg respectively.The prevalence of general obesity (BMI≥30 %) was 23% and that of central obesity 73%. The proportion of participants with both DM and hypertension was 34%, 18% had neither hypertension nor DM, 22% had only DM and 26% had only hypertension.

The mean FBG was 8.09 (4.48) mmol/L and there were no significant sex difference. The mean urea was 4.41 (1.71) mmol/L, mean creatinine98.61 (27.30) μmol/L and the mean

eGFR 118.5 (57.5) ml/min/1.73 m^2. The median (interquartile range) eGFR were 106.1 (93.7 - 123.8) ml/min/1.73 m^2 for males, 88.4 (77.8 - 103.0) ml/min/1.73 m^2 for females and 72.4 (56.5 - 94.5) ml/min/1.73 m^2 for all the participants. Mean creatinine was significantly higher in males but urea and eGFR were similar in the males and females (Table 2).

Tables 3 and 4 show the proportion of participants with the various classes of eGFR. There was none with eGFR< 15, 1.3% had eGFR 15 - 29, 28% had eGFR 30 - 59, 42% had eGFR 60 - 89 and 29% had eGFR> 90. There were significantly more females with eGFR> 90, while the males significantly had more eGFR15 - 89. When the results were stratified by clinical groups there statistically significant differences in the proportion of participants with eGFR 30 - 59 and eGFR> 90 in the various clinical groups but not in those with eGFR 15 - 29 and eGFR 60 - 89. Patients with only DM had the highest proportion of eGFR> 90 and the lowest proportion of eGFR 30 - 59 while those with only hypertension had the lowest proportion of eGFR> 90 and the highest proportion of eGFR 30 - 59.

The urine results were not statistically significant different in the men and women (Table 5). All the 16% of participants with urine glucose were DM patients (Table 6). Urine protein was detected in 16% of the participants, while abnormal microscopy was found in about 6%. Out of the 23 who had abnormal microscopy, one male and 4 females had haematuria (red blood cells >10 per high power field). Eight (2%) had both urine protein and abnormal microscopy, 8 (2%) had urine glucose and abnormal microscopy, 19 (5%) had urine protein and urine glucose while 4 (1%) had all the abnormalities, urine glucose, urine protein and abnormal microscopy. Table 7 shows the urine results by the various classes of eGFR. None of the participants with eGFR 0 -29 had urine glucose nor abnormal microscopy. All the 5 participants with eGFR 15 – 29 had urine protein. Twenty participants with eGFR> 90 and another 20 with eGFR 60 – 89 had urine protein and these were classified as CKD Stages 1 and 2 as shown in Tables 8 and 9.

The prevalence of both CKD Stages 1 and 2 were therefore 4.8%, while that of Stage 3 was 28% and Stage 4 was 1.3%. The overall prevalence of CKD was 38.9%. There were no participants with Stage 5 CKD. CKD was significantly common in the males than the females and in the hypertensives compared to those with normal blood pressure.

5. Discussion

This study has examined the renal function, urine dipstick findings and urine microscopy results of patients reporting at KATH. The prevalence of proteinuria was 16%, glycosuria was 16%, abnormal urine microscopy 6%, haematuria 1% and CKD 39%. There were no cases of Stage 5 CKD. There were significantly more males than females and more hypertensives than normortensives with CKD.

Chadban et al in the AusDiab kidney study determined the prevalence of reduced GFR, proteinuria and hematuria in 11,247 noninstitutionalized Australians adult population in a community based cross-sectional study. The prevalence of reduced GFR was 11.2%, hematuria 4.6% and proteinuria 2.4%. Approximately 16% of the population studied had at least one of these indicators (proteinuria, haematuria, or reduced GFR) indicating the presence of kidney damage. [11] From the Third National Health and Nutrition Examination Survey (NHANES III), Clase and others determined the prevalence of low GFR in nondiabetic black and white adult participants in the US. By the Cockcroft-Gault formula, 0.81% had GFR below 30 ml/min, 14% below 60 ml/min and 39% below 80 ml/min. Mean (sd) Cockcroft-Gault GFR was 89 (0.9) ml/min while mean serum creatinine was 94.5 (0.2) mmol/L. [21]In the 2009-2010 NHANES study, participants provided 2 untimed urine samples, an initial random urine collected in the NHANES mobile examination center and subsequently at home a first morning void. Saydah et al found the prevalence of albuminuria, defined as urine albumin/creatinine ratio ≥30 mg/g, to be 12.7% in the first morning urine and 15.2% in the random spot urine. [6] The results of these well designed national community based prevalence studies were lower than our findings though they cannot be directly compared. Such findings are expected since our study was a hospital based study with most of the participants being patients with hypertension and or DM which are recognized risk factors for renal failure.

In Kinshasa, Democratic Republic of Congo, Sumali and his team of researchers have conducted a number of nephrology studies. In a pilot study published in 2009, they reported the prevalence of CKD (Cockcroft-Gault) among the general population as follows: 1.4% had stage 1, 2.2% had stage 2, 15.0% had stage 3, 0.2% stage 4 and 0.2% had stage 5. The overall prevalence of CKD was 19.0%. [22] In a later clinical study involving patients with hypertension, DM, obesity and HIV, the prevalence of CKD (Modification of Diet in Renal Disease) was 4% for stage 1, 6% stage 2, 18% stage 3, 2% stage 4, and 6% had stage 5 CKD. The prevalence for all the stages of CKD was 36%. [23] The prevalence in these clinical cases was as expected higher than in the general population but similar to our findings in DM and hypertension patients.

We had earlier conducted a similar hospital based study in the Gambia and reported on 300 hospital patients with two thirds having hypertension and approximately 15% DM. The mean eGFR was 103.2 (80.2) ml/min/1.73 m^2 while the mean creatinine was 88.1 (54.1) μmol/L. The prevalence of CKD was 41% and the prevalence of proteinuria was 25%. In these Gambian patients there was a strong and significant association between proteinuria and CKD before and even after controlling for age, sex, hypertension and DM. [24] The prevalence of renal failure in the Gambian study was similar to the present study (41% vs. 39%, $p = 0.63$) while on the other hand the prevalence of proteinuria was significantly higher than the current study (25% vs. 16%, $p < 0.01$).

In Accra, Ghana Osafo et al screened 712 patients with known hypertension from 4 polyclinics. The prevalence of CKD stages 1-2 was 19.1%, CKD stages 3-5 was 27.8% with an overall prevalence of CKD of 46.9%. Of these patients

14.7% had pre-existing DM and their prevalence of renal failure (55%) was not significantly different from those without DM (46%) ($p = 0.13$). They found the overall prevalence of proteinuria to be 28.9%. [25] The prevalence of CKD and proteinuria in the Accra study were therefore higher than our current study.

A community based study in 12 communities in Ashanti Region measured creatinine clearance based on 2 timed 24 hour urine collections as well as estimating GFR by the Cockcroft-Gault formula in 944 participants with a hypertension prevalence of almost 30%. Mean creatinine was 79.0 (17.4) μmol/L and mean creatinine clearance GFR was 84.1 (22.6) ml/min/1.73 m^2. The prevalence of CKD by creatinine clearance was 12.7% for Stage 3, 0.5% for Stage 4 while there were no Stage 5 CKD. By Cockcroft-Gault estimation the mean eGFR was 74.7 (18.6) ml/min/1.73 m^2 while the prevalence of CKD was 20.3% for Stage 3 and 0.6% for Stage 4. By this method also there were no cases of Stage 5 CKD. [26]

Two previous studies have been conducted in KATH where our study was undertaken. In 1999 448 out-patient hypertension patients were recruited. The mean creatinine was 212 (296) μmol/L and 30.2% of the patients had creatinine> 140μmol/L while 13.1% had ≥ 400μmol/L. The prevalence of proteinuria determined by dipsticks was 25.5%. [27] In a 2007 study among DM patients, 109 out-patients participants had a prevalence of microalbuminuria of 43.1%. Patients with microalbuminuria had significantly higher serum creatinine and blood urea nitrogen than those patients without microalbuminuria. [28]

These studies indirectly confirm the findings of our study of high prevalence of renal disease, characterised by high prevalence of CKD and proteinuria. As in our current study these are findings within populations of mostly hypertension and DM patients, risk factors which are major contributors of renal disease. These patients with renal failure also have an increased risk for other adverse outcomes such as cardiovascular diseases and premature death. There is therefore the need of regular screening for renal failure among these groups of patients, to identify those with early stages of renal failure and to institute appropriate measures to prevent further progression and worsening of the renal failure. These measures may include pharmacological control of blood pressure and reduction of proteinuria using drugs such angiotensin – converting enzyme inhibitors and angiotensin II receptor antagonists, drugs which have been shown to have renoprotective effect. Other measures will include smoking cessation where applicable, lowering of blood lipids with statins and tight glucose control in DM. All these are feasible measures in our resource restrained setting and will prevent patients with early stages of renal failure from progressing to end stage renal failure where they will need the more expensive renal replacement therapies such peritoneal and haemodialysis or even renal transplant.

Our study therefore contributes to the reports on renal diseases in KATH in particular and in Ghana in general. This is one of the major strengths of this simple cross-sectional study which has simply presented the prevalence of renal failure and urine dipstick and microscopy findings. Our initial study was designed to report on lipid abnormalities among these patients so we did not determine the urinary albumin-creatinine ratio or albumin excretion rate on a morning urinary sample or a 24-hour urine sample which would have been the ideal investigation instead of using urine dipsticks on a single spot urine specimen. Proteinuria has imperfect accuracy in the diagnosis of persistent proteinuria and also has very low sensitivity. Other limitations include determining CKD status based on a single measurement of serum creatinine. Further we did not recalibrate our creatinine results against a traceable isotope dilution mass spectrometry (IDMS) enzymatic method, and so we were unable to determine eGFR using other methods like the Modification of Diet in Renal Disease (MDRD) and Chronic Kidney Disease Epidemiology Collaboration (CKD-EPI) equations. Cockcroft–GaulteGFR measurements has not been validated against a gold standard like the plasma clearance of 51Cr-EDTA in Kumasi however it was found to underestimate GFR creatinine clearance using 24-hour urine collections particularly in older age groups. [26]

6. Conclusion

The prevalence of proteinuria and CKD was high in Kumasi, Ghana. These findings suggest the urgent need to increase awareness, screening, detection, treatment and prevent progression of renal failure in patients in Kumasi, particularly among DM and hypertension patients.

Acknowledgements

This study was funded by the authors. There was no external financial assistance.

References

[1] BelloAK, Nwankwo E, El Nahas AM. Prevention of chronic kidney disease: a global challenge. Kidney Int Suppl. 2005 Sep;(98):S11-7.

[2] Park YH, Choi JY, Chung HS, Koo JW, Kim SY, Namgoong MK, Park YS, Yoo KH, Lee KY, Lee DY, Lee SJ, Lee JE, Chung WY, Hah TS, Cheong HI, Choi Y, Lee KS. Hematuria and proteinuria in a mass school urine screening test. Pediatr Nephrol. 2005 Aug;20(8):1126-30. Epub 2005 Jun 10.

[3] Plata R1, Silva C, Yahuita J, Perez L, Schieppati A, Remuzzi G. The first clinical and epidemiological programme on renal disease in Bolivia: a model for prevention and early diagnosis of renal diseases in the developing countries. Nephrol Dial Transplant. 1998 Dec;13(12):3034-6.

[4] Shajari A, Shajari H, Zade MH, Kamali K, Kadivar MR, Nourani F. Benefit of urinalysis. Indian J Pediatr. 2009 Jun;76(6):639-41. doi: 10.1007/s12098-009-0068-3. Epub 2009 Apr 23.

[5] Hajar F1, Taleb M, Aoun B, Shatila A. Dipstick urine analysis screening among asymptomatic school children. N Am J Med Sci. 2011 Apr;3(4):179-84. doi: 10.4297/najms. 2011.3179.

[6] Saydah SH, Pavkov ME, Zhang C, Lacher DA, Eberhardt MS, Burrows NR, Narva AS, Eggers PW, Williams DE. Albuminuria prevalence in first morning void compared with previous random urine from adults in the national health and nutrition examination survey, 2009-2010. Clin Chem. 2013 Apr;59(4):675-83. doi: 10.1373/clinchem.2012.195644. Epub 2013 Jan 11.

[7] Carroll MF1, Temte JL. Proteinuria in adults: a diagnostic approach. Am Fam Physician. 2000 Sep 15;62(6):1333-40.

[8] Simerville JA1, Maxted WC, Pahira JJ. Urinalysis: a comprehensive review. Am Fam Physician. 2005 Mar 15;71(6):1153-62.

[9] Boulware LE, Troll MU, Jaar BG, Myers DI, Powe NR. Identification and referral of patients with progressive CKD: a national study. Am J Kidney Dis. 2006 Aug;48(2):192-204.

[10] Chadban SJ, Atkins RC. Glomerulonephritis. Lancet. 2005 May 21-27;365(9473):1797-806.

[11] Chadban SJ, Briganti EM, Kerr PG, Dunstan DW, Welborn TA, Zimmet PZ, Atkins RC. Prevalence of kidney damage in Australian adults: The AusDiab kidney study. J Am Soc Nephrol. 2003 Jul; 14 (7 Suppl 2):S131-8.

[12] Kidney Disease: Improving Global Outcomes (KDIGO) CKD Work Group. KDIGO 2012 Clinical Practice Guideline for the Evaluation and Management of Chronic Kidney Disease. Kidney inter., Suppl. 2013;3:1–150.

[13] Micah FB, Nkum BC. Lipid disorders in hospital attendants in Kumasi, Ghana. Ghana Med J. 2012, 46:14-21.

[14] Chalmers J, MacMahon S, Mancia G, Whitworth J, Beilin L, Hansson L et al. 1999 World Health Organization-International Society of Hypertension Guidelines for the management of hypertension. Guidelines sub-committee of the World Health Organization. *Clin Exp Hypertens*. 1999 Jul-Aug; 21(5-6):1009-60.

[15] Alberti KG, Zimmet PZ. Definition, diagnosis and classification of diabetes mellitus and its complications. Part 1: diagnosis and classification of diabetes mellitus provisional report of a WHO consultation. *Diabet Med.* 1998 Jul;15(7):539-53.

[16] World Health Organisation. Definition and diagnosis diabetes mellitus and intermediate hyperglycaemia: Report of a WHO/IDF Consultation, Geneva, Switzerland. 2006.

[17] Clinical Guidelines on the Identification, Evaluation, and Treatment of Overweight and Obesity in Adults--The Evidence Report. National Institutes of Health. *Obes Res*. 1998 Sep;6 Suppl 2:51S-209S.

[18] Cockcroft DW, Gault MH. Prediction of creatinine clearance from serum creatinine. Nephron 1976;16:31–41.

[19] Mosteller RD. Simplified calculation of body-surface area. N Engl J Med 1987, 317(17):1098.

[20] Anonymous. Kidney Disease Outcome Quality Initiative. Clinical Practice Guidelines for Chronic Kidney Disease: Evaluation, Classification and Stratification. Am J Kidney Dis 2002;39 Suppl 1:S1-S246.

[21] Clase CM, Garg AX, Kiberd BA. Prevalence of low glomerular filtration rate in nondiabetic Americans: Third National Health and Nutrition Examination Survey (NHANES III). J Am Soc Nephrol. 2002 May;13(5):1338-49.

[22] Sumaili EK, Krzesinski JM, Zinga CV, Cohen EP, Delanaye P, Munyanga SM, Nseka NM. Prevalence of chronic kidney disease in Kinshasa: results of a pilot study from the Democratic Republic of Congo. Nephrol Dial Transplant 2009, 24(1):117–122.

[23] Sumaili EK, Cohen EP, Zinga CV, Krzesinski JM, Pakasa NM, Nseka NM. High prevalence of undiagnosed chronic kidney disease among at-risk population in Kinshasa, the Democratic Republic of Congo. BMC Nephrol 2009, 10:18.

[24] Nkum BC, Micah FB, Eghan BA, Ankrah TC, Nyan O. Renal function, uric acid and urine protein in adult patients attending outpatient clinics in Banjul, The Gambia. In print.

[25] 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. doi: 10.3109/0886022X.2011.565140.

[26] Eastwood JB, Kerry SM, Plange-Rhule J, Micah FB, Antwi S, Boa FG, Banerjee D, Cappuccio FP. Assessment of GFR by four methods in adults in Ashanti, Ghana: the need for an eGFR equation for lean African populations. Nephrol Dial Transplant. 2010 Jul;25(7):2178-87.

[27] Plange-Rhule J, Phillips R, Acheampong JW, Saggar-Malik AK, Cappuccio FP, Eastwood JB. Hypertension and renal failure in Kumasi, Ghana. J Hum Hypertens. 1999 Jan;13(1):37-40.

[28] EghanBA Jr, FrempongMT, Adjei-Poku M. Prevalence and predictors of microalbuminuria in patients with diabetes mellitus: a cross-sectional observational study in Kumasi, Ghana. Ethn Dis. 2007 Autumn;17(4):726-30.