Tropical Journal of Nephrology Vol.4 No1 June, 2009 21-29

Urinary Abnormalities, Blood Pressure and Anthropometric Profiles Among Students in a Nigerian University

[+]Ayodele OE, [++] Akanbi OO, [+]Akinwusi PO, Hassan AO and *Ogunro PO

*+Department of Medicine, *Department of Chemical Pathology, College of Health Sciences, Ladoke Akintola University of Technology, Osogbo, ++Department of Surgery, Ladoke Akintola University of Technology Teaching Hospital, Osogbo, Osun State Nigeria. Ladoke Akintola University of Technology Teaching Hospital, Osogbo, Osun State*

ABSTRACT

Reports from Nigeria indicate that young adults bear a disproportionate significant burden of end-stage renal disease (ESRD). Hypertension and chronic glomerulonephritis which account for e" 70% of the causes of ESRD in Nigeria can be diagnosed at an early stage by measurement of blood pressure and urine examination. Early detection of these diseases will allow early initiation of appropriate therapies and educational strategies, which will in turn impact positively on kidney outcome. This study was embarked upon to determine the frequency of urinary abnormalities, blood pressure and anthropometric profiles in university students. We conducted a cross sectional study in 131 (68 males, 63 females) 400L medical students of Ladoke Akintola University of Technology to determine the prevalence of urinary abnormalities, prehypertension, hypertension and overweight/obesity. The prevalence of prehypertension and hypertension in our cohort was 48% and 5% respectively. Overweight and obesity were found in 10.7% and 3.0% participants respectively. None of our study participants had significant urinary abnormality. In conclusion, our study shows that prehypertension and hypertension thus occur among university students. Thus, there is need to promote the initiation and sustenance of lifestyle modifications at an early age since this may help in the prevention of hypertension or progression of prehypertension to hypertension.

INTRODUCTION

Chronic kidney failure (CKF) represents the end of the continuum of chronic kidney disease (CKD) and is a devastating medical, social and economic burden for the patients, their families and the country as a whole [1,2]. Available reports indicate that the incidence and prevalence of CKF are on the increase globally [3-5] . The annual growth rate of patients with end-stage renal disease (ESRD) requiring renal replacement therapy (RRT) is between 5.0 % and 8.0% which far exceeds the world's population growth rate of 1.2% – 1.3% [5,6]. In addition, the average annual cost of RRT per patient is far in excess of the gross national income (GNI) per capita of most developing countries (economies) [7-9]. Thus, Nigeria may not be able to offer RRT to patients with ESRD. Reports from Nigeria also indicate that young adults bear a disproportionate significant burden of ESRD [10-12]. For example, 52% of the patients dialyzed between 1990 and 1995 at the University College Hospital, Ibadan were < 35 years of age.10 Thus, it is imperative that the nation must pursue a vigorous preventive program to reduce the burden of CKF.

Hypertension and chronic glomerulonephritis account for e" 70% of the causes of ESRD in Nigeria [10-12]. These diseases can be diagnosed at an early stage by measurement of blood pressure and examination of the urine. Early detection of these diseases will allow early initiation of appropriate

***Corresponding author :* Dr O.E Ayodele**

Department of Medicine, College of Health Sciences, Ladoke Akintola University of Technology, Osogbo, Osun State, Nigeria.

therapies and educational strategies, which may in turn impact positively on kidney outcome.

A continuous graded relationship exists between blood pressure (BP), cardiovascular disease (CVD) and CKD [13]. This relationship is present even at BP levels considered to be within non-hypertensive range [13]. This finding necessitated the introduction of prehypertension, defined as office systolic blood pressure (SBP) of 120 – 139 mm Hg and/or diastolic blood pressure (DBP) of 80 – 89 mm Hg in the Seventh Report of the Joint National Committee on Prevention, Detection, Evaluation and Treatment of High Blood Pressure (JNC 7) [14]. Many reports have shown that prehypertension is associated with increase in all cause and CVD mortality [15-17]. Prehypertension and hypertension have been associated with overweight /obesity suggesting that the prevalence of this condition will increase over time if the obesity epidemic continues to grow [15]. Obesity has also been associated with type 2 diabetes mellitus, coronary artery disease, stroke, arthritis, sleep apnoea syndrome and certain cancers [18]. The diagnosis of prehypertension and obesity should prompt the institution of lifestyle modifications at an earlier stage to prevent progressive rise in BP and related CVD.

In view of the high prevalence of ESRD in young adults in Nigeria and the opportunity that blood pressure measurement and urine examination offers in the early detection of hypertension and glomerulonephritis, this study was embarked upon to determine the prevalence of urinary abnormalities, hypertension, prehypertension and overweight/obesity in university students.

SUBJECTS AND METHODS

This was a cross sectional study involving 400 level clinical year students of Ladoke Akintola University of Technology (LAUTECH), Osogbo, Osun State, Nigeria. The study population consisted of 135 students who agreed to be part of the study after the aim and protocol had been explained to them. Female students were instructed to submit their urine after completion of menstruation in those undergoing menstruation. Excluded from the study were students who refused to be part of the study. The study protocol was approved by Research Ethics Committee of the Ladoke Akintola University of Technology.

A structured questionnaire was used to obtain information such as age, gender, marital status, current history of smoking, alcohol intake, oral contraceptive use, participation in formal exercise for e" 3 days per week, personal and family history of hypertension, diabetes and chronic kidney disease.

The weight (in kg) of each participant was taken in light clothing with the shoes off and the height (in metres) was done using a stadiometer. The body mass index (BMI) was calculated from weight/ height2 (kg/m2). Overweight and obesity were defined as BMI of 25 – 29.9 kg/m2 and e"30 kg/m2 respectively [14]. The waist circumference [WC] (in centimetres) was taken midway between the lowest rib and the iliac crest. The hip circumference [HC] (in centimetres) was taken at the level of the greater trochanters. Abdominal obesity was defined as abdominal circumference e" 102 cm in males and e" 88 cm in females [14]. The waist-hip ratio was determined by dividing the WC by the HC.

Each participant was instructed about urine sample collection. A mid-stream morning urine specimen was collected from each of the participants. Urinalysis was done using Combi 9 MultistixR by Bayer immediately after voiding. A 10 ml urine aliquot was then centrifuged at 3000 revolutions per minute (rpm) for 5 minutes and the supernatant was poured into a separate tube [19]. A drop of the urine sediment was placed on a glass slide and covered with cover-slip. At least 10 microscopic fields of the sample were examined at low and high magnification [19]. Urine microscopy was done within 2 hours of specimen collection. Persistent dipstick proteinuria of e" 1+, persistent dipstick haematuria e" 1+ in the absence of urinary tract infection, menstruation or strenuous exercise, presence of dysmorphic red blood cells, > 5 white blood cells per high power field (hpf), > 2 red blood cells /hpf, cellular casts and large quantity of hyaline cast were taken as important finding(s) [19]. All the urine microscopy examination was done by one of the authors (HAO).

The blood pressure (BP) of the participants was done using a mercury sphygmomanometer with appropriate cuffs and standardized protocols [20]. Each participant was allowed five minutes rest before BP measurement. Attempts were made also to ensure that participants had not taken coffee or smoked cigarette 30 minutes before BP was taken. Korotkoff's sounds I and V were taken as the systolic blood pressure (SBP) and diastolic blood pressure (DBP) respectively [20]. Three blood pressure readings were taken and the average of the last two readings was used for analysis. Blood pressure readings were classified according to the JNC 7 report [14]. When SBP and DBP fall into different categories, the higher category was selected to classify individual's BP.14 Normal blood pressure was defined as SBP < 120 mm Hg and DBP < 80 mm Hg. Prehypertension was defined as SBP between 120 and 139 mm Hg and/or DBP between 80 and 89 mm Hg [14]. Participants were classified as having hypertension if SBP was persistently e" 140mm Hg and / or DBP was e" 90 mm Hg or if on treatment with antihypertensive medication [14]. Participants who had BP in the hypertension range had their BP taken on two other separate occasions within two weeks to confirm persistent elevation of BP [14]. Pulse pressure was determined from difference between SBP and DBP. Conventional mean arterial blood pressure (MAP) was calculated using the formula:14. Mean arterial pressure (mm Hg) = DBP (mm Hg) + {1/3 (SBP [mm Hg] – DBP [mm Hg])}

STATISTICAL ANALYSIS

Data analysis was performed using SPSS software, version 15 (SPSS Inc., Chicago, Illinois, USA). Continuous and categorical variables were displayed as means ± standard deviation (S.D) and percentages respectively. The student's t test was used to assess differences between means. Differences between categorical variables were analyzed by Chi-square test with Fisher's exact correction applied as appropriate. Differences between groups were analyzed by analysis of variance (ANOVA). A p <

Table 1: Baseline demographic and clinical characteristics of the study population

Participants' characteristics	Male (%)	Female (%)	Total (%)
Gender	68 (52)	63 (48)	131 (100.0)
Mean age (years)	25.4 ± 2.5	24.7 ± 1.4	25.1 ± 2.1
Family history of hypertension			
Yes	20 (29.4)	19 (30.2)	39 (29.8)
No	40 (58.8)	40 (63.5)	80 (61.1)
Don't know	8 (11.8)	4 (6.3)	12 (9.1)
Family history of DM			
Yes	10 (14.7)	12 (19.0)	22 (16.8)
No	55 (80.9)	50 (79.4)	105 (80.2)
Don't know	3 (4.4)	1 (1.6)	4 (3.0)
Family history of CKD			
Yes	1 (1.5)	1 (1.6)	2 (1.5)
No	66 (97.0)	61 (96.8)	127 (97.0)
Don't know	1(1.5)	1 (1.6)	2 (1.5)
Personal history of hypertension			
Yes	1 (1.5)	0 (0.0)	1 (0.8)
No	67 (98.5)	63 (100.0)	130 (99.2)
Personal history of DM			
No	68 (100.0)	63 (100.0)	131 (100.0)
Formal exercise			
Yes	1 (1.5)	9 (14.3)	0 (7.6)
No	67 (98.5)	54 (85.7)	121 (92.4)
Current alcohol intake			
Yes	2 (2.9)	0 (0.0)	2 (1.5)
No	66 (97.1)	63 (100.0)	129 (98.5)
Current smoking			
No	68 (100.0)	63 (100.0)	131 (100.0)

0.05 for 2 tailed tests was used to determine statistical significance.

RESULTS

The study population consisted of 131 students [68 males (51.9%) and 63 females (48.1%)]. Table I showed the baseline demographic and clinical characteristics of the study population. The mean age of the study population was 25.1 ± 2.1 years (range 21 – 34 years). Family history of hypertension, diabetes mellitus and CKD was obtained in 39 (29.8%), 22 (16.8%) and 2 (1.5%) participants respectively. Only 10 participants formally undertook exercise. None of the participants currently smokes and the frequency of alcohol consumption was also quite low. None of the female participants was on oral contraceptive pills.

None of the participants had significant proteinuria (defined as e"1+) though 18 (13.7%) had trace proteinuria. None of the participants had haematuria, glycosuria, leucocyturia or significant cellular elements on urinary microscopy.

Table 2 showed the blood pressure and anthropometric profiles of the study population. The mean SBP and DBP of the study population were 118.7 ± 12.6 mm Hg and 74.7 ± 8.0 mm Hg respectively. Compared to females, males had a significantly higher mean DBP (78.8 ± 7.3 vs. 72.3 ± 8.1mm Hg, p = 0.001, 95% CI 1.8 – 7.2), higher MAP (91.4 ± 8.0 vs. 87.1± 8.9 mm Hg, 95 % CI = 1.4 – 7.2, p = 0.004), as well as a higher mean WHR (0.87 ± 0.05 vs. 0.80 ± 0.05, 95% CI = 0.05 – 0.08, p < 0.001) [Table 2]. Males were also significantly taller (1.72 ± 0.07 vs. 1.62 ± 0.06, 95% CI = 0.08 – 0.12, p < 0.001) and heavier than females (64.3 ± 7.3 vs. 58.2 ± 9.8 kg, 95% CI = 3.2 – 9.1, p < 0.001).

However, there was no statistically significant difference in the BMI of male and female participants (21.8 ± 2.0 vs. 22.2 ± 3.7 kg/m2, 95 % CI = 0.6 – 1.4, p = 0.43). Females on the other hand had a statistically significant higher mean HC than males. Ten participants (9 females, 1 male) engaged in formal exercise. Participants who engaged in formal exercise had significantly lower DBP (68.8 ± 7.2 vs. 75.1 ± 7.9, 95% CI= - 1.23 to -11.42, p = 0.015) and comparable SBP (120.2 ± 17.3 vs. 118.6 ± 12.3, 95% CI = -6.68 to 9.28, p = 0.707) when compared to those who did not engage in formal exercise. After

Table 2: Blood pressure and anthropometric profiles of study participants.

Participants' characteristics	Male (n=68)	Female (n=63)	Total (n=131)	95% CI	p value
Age (years)	25.4 ± 2.5	24.7 ± 1.4	25.1±2.1	0.03 - 1.43	0.042
SBP (mm Hg)	120.6 ±12.1	116.7 ±13.0	118.7±12.6	-0.44 - 8.24	0.078
DBP (mm Hg)	78.8 ± 7.3	72.3 ± 8.1	74.7±8.0	1.84 - 7.16	0.001
PP (mm Hg)	43.8 ± 9.6	44.4 ± 9.8	44.1±9.7	-3.96 - 2.76	0.724
MAP (mm Hg)	91.4 ± 8.0	87.1 ± 8.9	89.3±8.7	1.39 - 7.21	0.004
Weight (kg)	64.3 ± 7.3	58.2 ± 9.8	61.4±9.1	3.19 - 9.12	<0.001
Height (m)	1.72 ± 0.07	1.62 ± 0.06	1.67±0.08	0.08 - 0.12	<0.001
BMI (kg/m2)	21.8 ± 2.0	22.2 ± 3.7	22.0±2.9	-1.43 - 0.61	0.431
WC (cm)	75.2 ± 5.5	75.5 ± 8.0	75.3±6.8	-2.70 - 2.21	0.780
HC (cm)	86.6 ± 5.3	94.1 ± 8.6	90.2±8.0	- 9.99 - -5.08	<0.001
WHR	0.87 ± 0.05	0.80 ± 0.05	0.84±0.06	0.05 - 0.08	<0.001

Key: *Values for continuous variables are mean ± SD. CI – confidence interval, M – male, F – female, SBP – systolic blood pressure, DBP – diastolic blood pressure, MAP – mean arterial pressure, PP – pulse pressure, BMI – body mass index, WC – waist circumference, HC – hip circumference, WHR – waist hip ratio.*

correcting for the effect of gender, females engaged in regular formal exercise still had significant lower DBP (67.28 ± 5.60 vs. 73.15 ± 8.19 mmHg, 95% CI = -10.52 to -0.22, p = 0.017) when compared with their counterparts who did not engage in formal exercise. When compared to those without family history of hypertension, participants with family history of hypertension had a higher, though insignificant mean SBP (119.7 ± 8.7 vs. 118.0 ± 14.2 mm Hg, p = 0.496) and DBP (75.4 ± 6.2 vs. 73.9 ± 8.3, p = 0.313).

Table 3: Blood pressure classification (according to JNC 7), body mass index, abdominal obesity categories and engagement in formal exercise of the study population

Participants' characteristics	Male n = 68 (%)	Female n = 63 (%)	p value
Systolic blood pressure (mm Hg)			
<120	31 (45.6)	40 (63.5)	0.117
120 - 139	33 (48.5)	20 (31.7)	
140 - 159	3 (4.4)	2 (3.2)	
e” 160	1 (1.5)	1 (1.6)	
Diastolic blood pressure (mm Hg)			
<80	38 (55.9)	50 (79.4)	0.017
80 - 89	28 (41.2)	11(17.4)	
90 - 99	2 (2.9)	1 (1.6)	
e” 100	0 (0.0)	1 (1.6)	
Body mass index (kg/m2)			
<25	63 (92.6)	50 (79.4)	0.04
25.0 - 29.9	5 (7.4)	9 (14.3)	
e” 30	0 (0.0)	4 (6.3)	
Abdominal obesity			
Yes	0 (0.0)	4 (6.3)	0.035
No	68 (100.0)	59 (93.7)	
Formal exercise			
Yes	1 (1.5)	9 (14.3)	0.007
No	67 (98.5)	54 (85.7)	

Table 3 showed the BP, BMI and abdominal obesity distribution of the study population. The frequencies of overweight and obesity in the participants were 10.7 % and 3.0 % respectively with more female participants having BMI e” 25 kg/m2 (21% vs. 7%, p = 0.027). Also, abdominal obesity was found in 4 females and none of the males (p = 0.035).

Normal BP, prehypertension and hypertension were found in 47%, 48% and 5% of the participants respectively (Table 4). More males had prehypertension compared to females (60% vs. 35%, p = 0.002) [Table 4]. Majority of the participants (71%) with hypertension had stage 1 hypertension. When compared with participants with normal BP, participants with prehypertension had statistically significant higher mean weight (65.3 ± 8.9 vs. 57.2 ± 7.9 kg, p < 0.001), higher mean BMI (22.9 ± 3.3 vs. 21.0 ± 2.4, p < 0.001), higher mean WC (77.2 ± 7.2 vs. 73.4 ± 6.0 cm, p = 0.002) and higher WHR (0.85 ± 0.06 vs. 0.82 ± 0.06, p = 0.02) [Table IV]. There was no statistically significant difference in the gender population, weight, BMI, WC, HC and the WHR of participants with prehypertension and those with hypertension (Table 4).

DISCUSSION

The prevalence of obesity in our study was 3.0% which is comparable to 3.4% in the study by Odili *et al.* in undergraduates at Delta State University.21 However, the prevalence of overweight of 10.7% in our study was much lower than 23.9% by Odili *et al* and 27% by Huang et al in USA college students.22 Also, the prevalence of obesity and overweight in our cohort were less than 14.8% and 7.4% respectively obtained in urban Nigerian women aged 25 to 34 years.23 The reasons for this difference is not obvious may relate to caloric intake, level of physical exertion and family history of obesity.

Male participants in this study had higher SBP and DBP compared to females, a finding that was also shown by Odili *et al.* [21]. This gender difference in BP can be explained by the fact that during the teenage years, average BP is consistently higher for boys than for girls such that by age 18 years, boys have average systolic and diastolic blood pressures that are almost 10 mm Hg and 5 mmHg higher, respectively, than the corresponding values for girls.24 The frequency of hypertension in our cohort was 5% which was slightly higher than 3.4% obtained by Odili *et al.* However, our value is much lower than 9.9%
51, 7 (in women) and 10.5% (in men) aged 20 – 29 years obtained for sub-Saharan Africa in the analysis of worldwide data of hypertension by Kearney et al.25

Table 4: Demographic, anthropometric and clinical characteristics of participants with prehypertension as compared with participants with normal blood pressure and hypertension

Participants' characteristics	Normotensive (n=61)	Prehypertensive (n=63)	pa	Hypertensive (n=7)	pb
Gender (M/F)	23/38	41/22	0.002	4/3	0.327
Age (years)	24.8 ± 1.6	25.2 ± 2.2	0.27	26.4 ± 3.3	0.192
SBP (mm Hg)	109.6 ± 5.9	123.7 ± 7.6	<0.001	153.0 ± 7.3	<0.001
DBP (mm Hg)	69.3 ± 4.9	78.4 ± 5.6	<0.001	87.4 ± 13.6	0.001
Conventional					
MAP (mm Hg)	82.7 ± 4.7	93.5 ± 4.5	<0.001	109.2 ± 10.5	<0.001
PP (mm Hg)	40.3 ± 5.1	45.3 ± 9.5	0.001	65.6 ± 11.8	<0.001
Weight (kg)	57.2 ± 7.9	65.3 ± 8.9	<0.001	59.6 ± 5.8	0.108
Height (m)	1.65 ± 0.07	1.69 ± 0.09	0.008	1.64 ± 0.07	0.131
BMI (kg/m2)	21.0 ± 2.4	22.9 ± 3.3	<0.001	22.2 ± 1.5	0.583
WC (cm)	73.4 ± 6.0	77.2 ± 7.2	0.002	75.1 ± 5.8	0.472
HC (cm)	89.3 ± 6.4	91.3 ± 9.4	0.170	87.7 ± 7.1	0.329
WHR	0.82 ± 0.06	0.85 ± 0.06	0.02	0.86 ± 0.08	0.592

Key: *Values for continuous variables are mean ± SD. M – male, F – female, SBP – systolic blood pressure, DBP – diastolic blood pressure, MAP – mean arterial pressure, PP – pulse pressure, BMI – body mass index, WC – waist circumference, HC – hip circumference, WHR – waist hip ratio. Pa – post-hoc p value for comparison between participants with normal blood pressure and those with prehypertension. Pb - post-hoc p value for comparison between participants with prehypertension and those with hypertension.*

Males had a larger WHR than females, an observation primarily due to females having larger HC compared to males. Females were more obese and showed significant truncal obesity, a dimension of obesity that has been shown to have more adverse health consequences such as impaired glucose tolerance, diabetes mellitus, hypertension, heart disease and stroke than peripheral obesity.

Our finding of lower diastolic BP in participants who engaged in formal physical exercise was consistent with findings from observational epidemiological and randomized controlled studies that demonstrated an inverse relationship between physical activity and blood pressure.26-28 In a meta-analysis by Whelton *et al* involving 54 randomized controlled trials, pooled estimates showed that aerobic exercise was associated with a significant reductio in SBP (-3.84 mmHg) and DBP (-2.58 mmHg).28 The mechanisms by which physical exercise reduce BP include reduction in weight; improvement in insulin sensitivity with subsequent reduction in insulin levels, a factor that has been implicated in the pathogenesis of hypertension; and enhancement of endothelium-dependent vasodilatation which is mediated in part by increased nitric oxide production.29 In addition, regular physical activity is associated with improvement in high density lipoprotein cholesterol (HDL-C), and reduction in levels of total cholesterol, low density lipoprotein cholesterol (LDL-C), triglycerides and markers of inflammation such as C – reactive protein though we did not investigate for these in our study.29 Thus, the beneficial effects of physical activity on cardiovascular disease risk reduction go beyond BP lowering.

We found the prevalence of prehypertension in our cohort to be 48%, a value that is higher than 32% found in those aged 18 to 39 years in the US.30 This represents individuals in whom early intervention by adoption of healthy lifestyles could reduce BP, reduce the rate of progression of BP to hypertensive levels with age, or prevent hypertension entirely.14 We did not find any person with significant urinary abnormalities. This may relate to the relatively small number of our cohort. In a study by Topham et al,31 only 1% of 3570 university students had persistent urinary abnormality. The low prevalence of alcohol consumption and the absence of smoking in our study population may be due to the awareness of the participants of the adverse health implications of these social habits.

Our study had some limitations. First, the study cohort being our students may be regarded as a vulnerable one. However, the students were made to realize that no form of punishment will be meted out to those who refused to be part of the study and the entire cost of the study was borne by the investigators. Second, we did not investigate for the prevalence of microalbuminuria which may be an early marker of kidney disease.1 However, there is likelihood that most of our participants with trace proteinuria have microalbuminuria.[32, 33] Sam *et al* [32] and Konta *et al* [33] in separate studies demonstrated that trace proteinuria could be a useful indicator of microalbuminuria. Single episode dipstick positive proteinuria of e”1+ was chosen in this study because this level of proteinuria had been shown to be associated with increased risk for the development of end-stage renal disease (ESRD)[34, 35]. For example, Tozawa *et al* [34] demonstrated that baseline proteinuria of e”1+ was associated with a relative risk for the development of ESRD of 11.29 in men and 12.5 in women. Third, we cannot completely rule out “white coat effect” in our cohort and there is likelihood that the use of ambulatory blood pressure measurement (ABPM) may give lower prevalence of prehypertension and hypertension in this cohort.14

In conclusion, our study shows that prehypertension and hypertension were fairly prevalent in our university students. Thus, there is need to promote the initiation and sustenance of lifestyle modifications at an early age since this may help in the prevention of hypertension or progression of prehypertension to hypertension.

REFERENCES

1. National Kidney Foundation. K/DOQI clinical practice guidelines for chronic kidney disease: evaluation, classification and stratification: Am J Kidney Dis 2002; 39 (Suppl 1): S1 – S266.
2. Schoolwerth AC, Engelgau MM and Hostetter TH: A public health action plan is needed in chronic kidney disease. Advances in Chronic kidney disease 2005; 12 (4): 418 – 423.
3. Alebiosu CO and Ayodele OE. The global burden of chronic kidney disease and the way forward. Ethn Dis 2005; 15: 418 – 423.
4. El Nahas AM and Bello AK: Chronic kidney disease: the global challenge. Lancet 2005; 365: 331 – 340.
5. Lysaght MJ: Maintenance dialysis population dynamics: current trends and long-term implications. J Am Soc Nephrol 2002; 13: S37 – S40
6. World Population Prospects. The 2006 revision. Available at www.un.org/esa/population/publications/wpp 2006/WWP 2006_Highlights_red.pdf
7. Moosa MR and Kidd M: The dangers of rationing dialysis treatment: the dilemma

facing a developing country. Kidney Int 2006; 70: 1107 – 1114.

8. Elhassan EAM, Kaballo B, Fedail H *et al:* Peritoneal dialysis in the Sudan. Perit Dial Int 2007; 27: 503 – 510.

9. White SL, Chadban SJ, Jan S, Chapman JR and Cass A: How can we achieve global equity in provision of renal replacement therapy? Bull World Health Organization 2008; 86: 229 – 237.

10. Salako BL: Managing chronic renal disease in Nigeria. Niger J Med 2001; 40(3): 75 – 77.

11. Akinsola W, Odesanmi WO, Ogunniyi JO and Ladipo GOA. Diseases causing chronic renal failure in Nigerians –A prospective study of 100 cases: Afr J Med med Sci 1989; 18: 131 – 135.

12. Alebiosu CO, Ayodele OO, Abbas A and Aina Olutoyin A: Chronic renal failure at Olabisi Onabanjo University Teaching Hospital, Sagamu, Nigeria. African Health Sciences 2006; 6 (3): 132 – 138.

13. MacMahon S, Peto R, Cutler J *et al.* Blood pressure, stroke and coronary heart disease. Part 1, prolonged differences in blood pressure: prospective observational studies corrected for the regression dilution bias. Lancet 1990; 335: 765 – 774.

14. Chobanian AV, Bakris GL, Black HR *et al* for the National High Blood Pressure Education Program Coordinating Committee. Seventh Report of the Joint National Committee on Prevention, Detection, Evaluation and Treatment of High Blood Pressure. Hypertension 2003; 42: 1206 – 1252.

15. Svetkey LP. Management of prehypertension. Hypertension 2005; 45: 1056 – 1061.

16. Toprak A, Wang H, Chen W *et al.* Prehypertension and black-white contrasts in cardiovascular risk in young adults: Bogalusa Heart Study. J Hypertens 2009; 27: 243 – 250.

17. Greenhund KJ, Croft JB and Mensah GA. Prevalence of heart disease and stroke risk factors in persons with prehypertension in the United States, 1999 – 2000. Arch Intern Med 2004; 164: 2113 – 2118.

18. Haslam DW and James WPT. Obesity. Lancet 2005; 366: 1197 – 209.

19. Kouri T, Fogazzi G, Gant V *et al.* European urinalysis guidelines. Scan J Clin Lab Med 2000; 60 (Suppl. 231): 1 – 96.

20. Beevers G, Lip GYH and O'Brien E. ABC of hypertension: Blood pressure measurement. BMJ 2001; 322: 1043 – 1047.

21. Oghagbon KE, Odili VU, Nwangwa EK, Pender KE. Body mass index and blood pressure pattern of students in a Nigerian University. International Journal of Health Research 2009; 2: 177 – 182.

22. Huang T T-K, Kempf AM, Strother ML *et al.* Overweight and components of the metabolic syndrome in college students. Diabetes Care 2004; 27: 3000 – 3001.

23. Rotimi CN, Cooper RS, Ataman SL *et al.* Distribution of anthropometric variables and the prevalence of obesity in populations of West African origin; The International Collaborative Study on Hypertension in Blacks (ICSHIB). Obesity Research 1995; 3 (Suppl. 2): 95S – 106S.

24. Buzzano LA, He J and Whelton PK. Blood pressure in westernized and isolated population. In: Comprehensive Hypertension (eds. Lip GYH and Hall JE) 2007; 21 – 30, Mosby Elsevier, Philadelphia.

25. 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.

26. Hayashi T, Tsumura K, Suematsu C, Okada K, Fujii S and Endo G. Walking to work and the risk of hypertension in men: the Osaka Health Survey. Ann Intern Med 1999; 131: 21 – 26.

27. Pereira MA, Folsom AR, McGovern PG *et al.* Physical activity and incident hypertension in black and white adults: the Atherosclerosis Risk in Communities Study. Prev Med 1999; 28: 304 – 312.

28. Whelton SP, Chin A, Xin X and He J. Effects of anaerobic exercise on blood pressure: a meta-analysis of randomized controlled trials. Ann Intern Med 2002; 136: 493 – 503.

29. Stewart KJ. Exercise training and the cardiovascular consequences of type 2 diabetes and hypertension: plausible mechanisms for improving cardiovascular health. J Am Med Assc 2002; 288: 1622 – 1631.

30. Wang Y and Wang QJ. The prevalence of prehypertension and hypertension among US adults according to the New Joint National Committee Guidelines. New Challenges of the Old Problem. Arch Intern Med 2004; 164: 2126 – 2134.

31. Topham PS, Jethwa A, Watkins M, Rees Y and Feehally J. The value of urine screening in a young adult population. Family Practice 2004; 21: 18 – 21.

32. Sam R, Shaykh MS, Pegoraro AA *et al.* The significance of trace proteinuria. Am J Nephrol 2003; 23: 438 – 441

33. Konta T, Hao Z, Takasaki S *et al.* Clinical utility of trace proteinuria for microalbuminuria screening in the general population. Clin Exp Nephrol 2007; 11: 51 – 55

34. Tozawa M, Iseki K, Iseki C, Kinjo K, Ikemiya Y and Takishita S. Blood pressure predicts risk of developing end-stage renal disease in men and women. Hypertension 2003; 41: 1341 – 1345.

35. Ishani A, Grandits GA, Grimm RH *et al.* for the MRFIT Research Group. Association of single measurements of dipstick proteinuria, estimated glomerular filtration rate, and haematocrit with 25-year incidence of end-stage renal disease in the Multiple Risk Factor Intervention Trial. J Am Soc Nephrol 2006; 17: 1444 – 1452.