Research article

Prevalence and risk factors for hyperuricemia among patients with hypertension at Moi Teaching and Referral Hospital, Eldoret, Kenya

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Abstract

Objective: Uric acid, a mediator of high blood pressure, is an inexpensive easy-to-obtain indicator of cardiovascular risk (stroke, myocardial infarction and renal disease). This study was conducted to determine the prevalence and risk factors for hyperuricemia among patients with hypertension in western Kenya.

Methods: This cross-sectional study conducted at the Moi Teaching and Referral Hospital in western Kenya, enrolled randomly selected adults (≥ 18 years) with hypertension, attending medical outpatients' clinic. Clinical (age, gender, stroke history, Body Mass Index, antihypertensive drugs and duration of illness) and laboratory (fasting lipid profile, blood sugar, uric acid and serum creatinine) data were collected. Data were keyed into Microsoft excel database and analyzed using STATA© version 13. Descriptive statistics were summarized using means, frequencies and proportions. Risk factors for hyperuricemia were analysed using two-sample t-tests, two-sample Wilcoxon rank sum tests and Pearson's Chi Square tests.

Results: Of the 275 participants enrolled, 182 (66%) were female, mean age 54 (sd 12.5) years, mean Body Mass Index 28.9 (sd 4.9) and median duration of illness 6 months. Overall prevalence of hyperuricemia was 44%; with 37.6% and 47.3% in males and females respectively.Factors associated with hyperuricemia included high Body Mass Index (p 0.036), low Glomerular Filtration Rate (P<0.0001) and dyslipidemia (p<0.0001).

Conclusion: There is a high prevalence of hyperuricemia among patients with hypertension in western Kenya. Risk factors associated with hyperuricemia include high Body Mass Index, dyslipidemia and low glomerular filtration rate. The use of losartan and calcium channel blockers is recommended in patients with hyperuricemia and subsequent longitudinal studies to be done to determine utility of uric acid monitoring in blood pressure control.

Key words: Hypertension, Hyperuricemic Lipid Profile, Body Mass Index, Estimated Glomelular Filtration Rate (eGFR).

Introduction

Hypertension is currently the most common cardiovascular problem in Africa, affecting an estimated 20 million people.The prevalence of hypertension in Africa ranges from 25% to 35% in adults aged 25 to 64 years and increases with advancing age[1]. In Kenya the prevalence of hypertension is 21%[2]. In sub-Saharan Africa, hypertensive end organ damage including haemorrhagic and atherothrombotic strokes, hypertensive heart disease, hypertensive nephrosclerosis and coronary artery disease is a major cause of morbidity and mortality[1,3]. There is a drastic shift from increase in communicable diseases to non- communicable diseases[4].

Uric acid has been associated with development of hypertension in several reports.[5-8]. Studies have shown that hyperuricemia is closely associated with an increased risk for hypertension, impaired fasting glucose, Type II diabetes and the metabolic syndrome[9-11]. In the Framingham Heart Study, each increase in serum uric acid levels by 1.3mg/dl was associated with the development of hypertension with an odd ratio of 1.17[12]. In the First National Health and Nutrition Study (NHANES I) study, for every 1.01 mg/dl increase in the serum uric acid levels, the hazard ratio for total mortality and for cardiovascular mortality were 1.09 and 1.19 for men and 1.26 and 1.3 for women, respectively[13].

Hyperuricemia is one of the important risk factors for coronary artery disease in patients with hypertension. It has been associated with end-organ damage for instance, left ventricular

hypertrophy, carotid atherosclerosis, microalbuminaemia, cardiovascular events and mortality in patients with hypertension[14, 15].

Uric acid has been considered a causative factor for hypertension and atherosclerosis[16]. Elevated serum uric acid lowers endothelial nitric oxide levels, reducing neuronal nitric oxide synthase in the macula densa of the kidney and stimulates the rennin-angiotensin system. This mechanism was demonstrated in animal studies where rats developed high blood pressure in about 3 to 5 weeks after raised uric acid levels was induced by the administration of oxonic acid which is an inhibitor of Uricase[17].

Evidence from a number of studies suggests that uric acid be added to the list of conventional risk factors of hypertension like obesity, age, renal disease, and diabetes mellitus[18-21]. In Kenya, the current prevalence of hyperuricemia among patients with hypertension is unknown. Old data from Kenyatta National Hospital found a prevalence of hyperuricemia among untreated patients with essential hypertension to be 27.5% and among those on treatment to be 58%[22]. This study sought to determine the prevalence and associated risk factors for hyperuricemia among patients with hypertension at the Moi Teaching and Referral Hospital in western Kenya.

Materials and Methods

This study was conducted in the medical outpatient clinics of the Moi Teaching and Referral Hospital (MTRH). The hospital is located in Eldoret town, which is 350 Kilometers northwest of the Kenyan capital, Nairobi. MTRH is a tertiary (level 6) health facility serving as a teaching hospital for Moi University School of Medicine. It is the second referral hospital in Kenya and serves a catchment population of approximately 13 million people in western Kenya. The hospital has various specialist clinics, including medical outpatient clinics where patients with hypertension are seen twice weekly.

This was a cross-sectional descriptive study. The study enrolled adults (≥18 years) with hypertension for a period not exceeding 2 years since diagnosis. Hypertension was defined as a Systolic Blood Pressure (SBP)>140 mmHg and/or a Diastolic Blood Pressure (DBP) >90mmHg on two occasions, or being on antihypertensive medications. Participants with malignancies, those on chemotherapy, who were critically ill and pregnant women, were excluded.

The sample size was computed using the Cochran formula (Cochran, 1963). In order to have 95% confidence interval of the proportion with hyperuricemia of 58%[22] with a margin of error of 5%, a sample of 375 was required. However, after correcting for the finite population size of about 170 per month for 6 months, a sample size of 275 was computed.

Potential participants were identified through simple random sampling using computer generated random numbers. The clinic booking register served as the sampling frame. Participants who met the study eligibility criteria were consented and then recruited. Those who were not fasted were rescheduled for another day. Patients' self-reported bio data and a comprehensive medical history of current and past illnesses, including but not limited to: duration of hypertension, history of diabetes, stroke, alcohol use and cigarette smoking as well as medication use including hypertension medication, drugs for diabetes, and lipid lowering agents being used was collected.

Body Mass Index (BMI) was computed in the standard manner and the degree of obesity was classified based on National Institute of Health (NIH) cut offs[1]. Blood pressure measurement was done after a patient had rested for at least 5 minutes. Two readings were taken and the average was recorded in the questionnaire. Patients who reported history of diabetes, was verified from the records. The patients who were not known hypertensives but with BP readings of systolic above 140mmHg and diastolic of more than 90mmHg had their charts reviewed and if they had high blood pressure during their last visit they were considered hypertensive.

Fasting blood samples were drawn for lipid profile (total cholesterol, LDL, HDL and triglycerides); uric acid serum creatinine. The estimated Glomerular Filtration Rate (eGFR) was calculated using the Chronic Kidney Disease-Epidemiology Consortium (CKD-EPI) equation.

Data were collected between January and September 2015, using interviewer administered structured questionnaire. Medical records were also reviewed and relevant clinical and laboratory data were obtained and entered into the data collection form. The variables collected included demographic characteristics such as age, gender, history of smoking, alcohol use, diet and occupation. Medical and family history of hypertension, diabetes and kidney disease were also obtained. Other variables collected included; laboratory parameters (FBS, creatinine and lipid profile). The dependent/outcome variables are the levels of uric acid for the hypertensive patients. The data were de-identified, encrypted, double-entered into a Microsoft Excel© database and pass-word protected. All data were kept confidential in lockable cabinets.

Data were analyzed using STATA© version 13 special edition. Categorical variables were summarized as frequencies and corresponding percentages. Continuous variables that assumed Gaussian distribution were summarized as means and the corresponding Standard Deviation (SD). Continuous variable that violated the Gaussian assumptions were summarized as median and the corresponding Inter Quartile Range (IQR). Inferential statistics were used to draw conclusions about the population. Significance tests such as the two-sample t-test for comparison of two normally distributed continuous variables, two-sample Wilcoxon rank sum test (Mann Whitney U test) for non-Gaussian distributed continuous variables, and Pearson's Chi Square test for

categorical variables were used. Gaussian assumptions were assessed empirically using Shapiro Wilk test.

This study was carried out with the approval of the Institutional Research and Ethics Committee (IREC) of MTRH and Moi University School of Medicine and permission from MRTH management. A signed written informed consent was obtained for each participant who was included in this study. Confidentiality was maintained throughout the study by pass-word protecting database and limiting its access only to principal investigator and research assistants. Interviews were carried out in a consultation room to ensure privacy and convenience. All participants including those who declined consent received the same level of care awarded to all other patients irrespective of their participation. There were very minimal anticipated risks to the participants attributable to this study except the physical pain and discomfort associated with sample collection. Questionnaires will be shredded after three years of publication of the study findings. There was no conflict of interest in this study and no incentives were used to recruit patients. Patients were informed of their results and the same availed to their primary clinicians.

Results

Out of a total of 505 patients with hypertension screened at the medical outpatient clinics, 275 were enrolled as shown in Figure 1.Of the 275 who were enrolled 182(66%) were female. The mean age was 54.2 years (SD 14.3). More than two thirds, 215(78.2%) were un-employed. Thirty five (12.7%) had a history of smoking, 59(21.5%) had a history of alcohol use (Table 1).The median duration of illness after diagnosis was 6 months (IQR 2, 18). Twenty nine (10.6%) had a history of stroke and another 42(15.3%) had a history of diabetes. Treatment history showed that 40(14.6%) patients were on furosemide, 114(41.5%) were on hydrochlorothiazide, 29(10.6%) were on amlodipine, and 125(45.5%) were on nifedipine. Eighty four, 30.6%, were using enalapril while 10(3.6%) were on beta blockers (carvedilol-5 or propranolol-2 or atenolol-3).Twenty three 23(8.4%) were on atorvastatin for treatment of dyslipidemia (Table 2).

Figure 1: Enrollment Schema

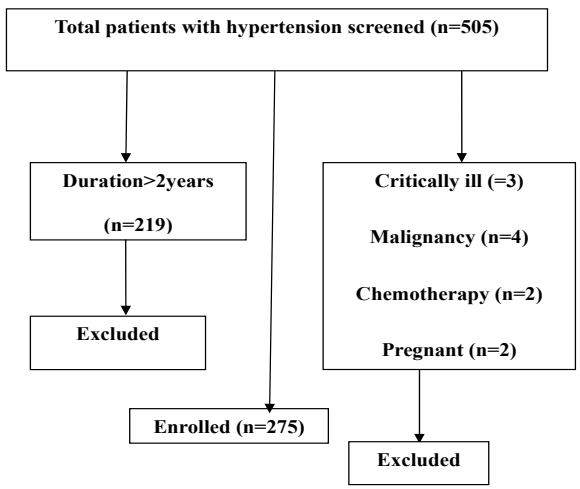

Table 1: Socio demographic characteristics

Variable	Normal uric acid (n=154)	Hyperuricemia (n=121)	p-value
Mean age (years)	55.0 (46.0, 61.0)	55.0 (45.0, 66.0)	0.739
Gender			
Male	58(37.7%)	35(28.9%)	0.128
Female	96(62.3%)	86(71.1%)	
History of smoking			
Yes	22(14.3%)	13(10.7%)	0.382
No	132(85.7%)	108(89.8%)	
Alcohol use			
Yes	32(20.8%)	27(22.3%)	0.758
No	122(79.2%)	94(77.7%)	

Table 2: Medical history of patients

Variable	Normal uric acid (n=154)	Hyperuricemia (n=121)	p-value
Reported history of stroke			
Yes	14(9.1%)	15(12.4%)	0.376
No	140(90.9%)	106(87.6%)	
Reported history of diabetes			
Yes	20(13.0%)	22(18.2%)	0.235
No	134(87.0%)	99(81.8%)	
Reported family history of hypertension			
Yes	74(48.1%)	55(45.5%)	0.668
No	80(51.9%)	45(54.5%)	
Reported family history of kidney disease			
Yes	6(3.9%)	9(7.4%)	0.199
No	148(96.1%)	112(92.6%)	
Hypertension control agents			
Hydrochlorothiazide	58(37.7%)	56(46.3%)	0.150
Furosemide	19(12.3%)	21(17.4%)	0.241
Amlodipine	14(9.2%)	15(12.4%)	0.376
Nifedipine	74(48.1%)	51(42.2%)	0.329
ACEI	47(30.5%)	37(30.6%)	0.992
ARB	29(18.8%)	21(17.4%)	0.753
Beta blockers	6(3.9%)	4(3.4%)	0.531[f]
Diabetes control agents			
Yes	21(13.6%)	17(14.1%)	0.921
No	133(86.3%)	104(85.9%)	
Lipid lowering agents			
Yes	7(4.6%)	16(13.2%)	0.010
No	147(95.4%)	105(86.8%)	

The median BMI was 28.9 (IQR: 26.1, 33.4) kgs/m^2 with a median Systolic Blood Pressure (SBP), and Diastolic Blood Pressure (DBP) of 154.0 (IQR: 139.0, 166.0) mm Hg, and 94.0 (IQR: 85.0, 102.0) mm Hg respectively. More than 70% had SBP> 140 mm Hg, and more than half had DBP>90 mm Hg. This gives the proportion of patients with uncontrolled blood pressure as 214 (77.8%). Other clinical characteristics are summarized in Table 3.

Table 3: Clinical characteristics

Variable	Normal uric acid (n=154)	Hyperuricemia (n=121)	p-value
Median duration of illness	6.0 (2.0, 12.0)	6.0 (2.0,18.0)	0.402
Mean BMI (kg/m^2)	28.4 (25.6, 32.6)	30.2 (26.5, 33.9)	0.033
Mean SBP (mm/Hg)	156.0(141,164.0)	150.0(133, 169)	0.540
Mean DBP (mm/Hg)	93.5(87.0,102.0)	96.0(82.0,104.0)	0.943
Mean pulse rate (beats/ min)	84.1±13.7	84.3±12.6	0.164
BMI			
Underweight (<18.5)	0(0%)	0(0%)	0.036
Normal (18.5-25)	34 (22.1%)	15 (12.4%)	
Overweight (25-30)	63 (40.9%)	45 (37.2%)	
0bese (>30)	57 (37.0%)	61 (50.4%)	
SBP			
>140mmHg	116 (75.3%)	83 (68.6%)	0.312[f]
<140mmHg	38(24.7%)	17(31.4%)	
DBP			
>90mmHg	92 (59.7%)	67 (55.4%)	0.184
<90mmHg	62(40.3%)	54(44.6%)	

The median serum uric acid was 338.5 (IQR: 270.0, 415.0) mmol/l. The prevalence of hyperuricemia in this cohort was 44.0% (95% CI: 28.0%, 50.0%). The median total cholesterol, HDL, LDL, and triglycerides were 4.9 (IQR: 4.0, 5.7) mmol/l; 1.0 (IQR: 0.8, 1.3) mmol/l; 2.9 (IQR: 2.1, 3.7) mmol/l, and 1.3 (IQR: 1.1, 1.8) mmol/l respectively. There were 248(90.2%) participants with dyslipidemia; elevated total cholesterol, 114(41.5%); decreased HDL, 207 (75.3%), elevated LDL, 125(45.5%), and elevated triglycerides, 87(31.6%). The median creatinine was 66.0 (IQR: 53.0, 82.2) mmol/l, and median eGFR was 110.5 (IQR: 88.7, 122.7) ml/min per 1.73m^2 (Range3.3-164.3 ml/ min per 1.73m^2). An estimated 24(8.7%) were in chronic kidney disease stage 3 or 4. Other clinical and laboratory characteristics are summarized in Table 4.

In the univariate model of factors associated with hyperuricemia, higher Body Mass Index (p=0.036); use of lipid lowering drugs (p=0.010); lower HDL (median 0.9 (IQR: 0.7, 1.2) vs. 1.1 (IQR: 0.9, 1.4) mmol/l, p=<0.0001) and elevated triglycerides 49(40.5%) vs. 38(24.7%), p=0.005) were positively associated with hyperuricemia. Dyslipidemia, defined as either elevated total cholesterol, Low-Density Lipoprotein (LDL), triglycerides or low levels of High-Density Lipoprotein (HDL), was associated with the presence of hyperuricemia, 118 (97.5%) vs. 130 (84.4%), p<0.0001. Lower eGFR levels were also associated with the presence of hyperuricemia (median eGFR: 103.4 (IQR: 74.5, 134.8) vs. 121.7 (108.0, 148.5), p<0.0001). Chronic kidney disease stage 3 or worse was associated with the presence of hyperuricemia, 20 (16.5%) vs. 1 (0.7%), p<0.0001. (Tables 3 and 4).

In the adjusted multivariate model, factors associated with increased risk of hyperuricemia included female gender (OR: 2.18 (95% CI: 1.15, 4.15); higher BMI (every 5 units higher) (OR: 1.50 (95% CL: 1.13, 1.99); dyslipidemia (OR: 4.40 (95% CI: 1.12, 17.19) and use of

Table 4: Laboratory characteristics

Variable	Normal uric acid (n=154)	Hyperuricemia (n=121)	p-value
Mean total cholesterol	4.9 (4.1, 5.6)	4.9 (3.8, 5.8)	0.919
Mean HDL	1.1 (0.9, 1.4)	0.9 (0.7, 1.2)	<0.0001
Median LDL	2.9 (2.3, 3.5)	2.9 (2.0, 3.8)	0.964
Median triglycerides	1.3 (1.1, 1.7)	1.4 (1.2, 2.1)	0.061
Median eGFR	116.6 (102.3, 127.4)	101.6 (73.9, 118.5)	<0.0001
LDL			
>2.50mmo/l	68(44.1%)	57 (47.1%)	0.626
<2.50mmo/l	86(55.84%)	64(52.9%)	
Total cholesterol			
>5.17mmol/l	59 (38.3%)	55 (45.5%)	0.233
<5.17mmol/l	95(61.7%)	66(54.5%)	
Triglycerides			
>1.8mmol/l	38 (24.7%)	49 (40.5%)	0.005
<1.8mmo/l	116(75.3%)	72(59.5%)	
HDL*			
Low	91 (59.1%)	99 (81.8%)	0.001
High	63(40.9%)	22(18.2%)	
Dyslipidemia	122 (79.2%)	116 (95.9%)	<0.0001
eGFR			
above 60	150(97.4%)	101(83.5%)	<0.0001[f]
30-60	3(2%)	6(5%)	
15-30	0(0%)	5(4.1%)	
<15	1(0.7%)	9(7.4%)	
Stage 3 or worse CKD	4(2.6%)	20(16.5%)	<0.0001

Table 5: Factors associated with presence of hyperuricemia

Variable	Odds Ratio (95% CL)
Age (per 10 years increase)	0.71 (0.56, 0.90)
Female	2. 18 (1.15, 4.15)
BMI (per 5 units increase)	1.50 (1.13, 1.99)
eGFR (per 30 units increase)	0.32 (0.22, 0.48)
Dyslipidemia	4.40 (1.12, 17.19)
Lipid drugs (ATOV)	4.63 (1.42, 15.13)
Drugs for diabetes mellitus	0.23 (0.06, 0.95)
Nifedipine	0.44 (0.24, 0.82)
ARB	0.39 (0.17, 0.92)
HCTZ	1.57 (0.88, 2.81)
Aspirin	3.01 (0.68, 13.32)
History of diabetes mellitus	2.75 (0.73, 10.35)

lipid lowering drugs (OR: 4.63 (95% CL: 1.42, 15.13). Factors associated with reduced risk of hyperuricemia included use of diabetes mellitus drugs (OR: 0.23 (95% CL: 0.06, 0.95); use of nifedipine (OR: 0.44 (95% CL: 0.24, 0.82); use of ARB (OR: 0.39 (95% CL: 0.11, 0.92); older age (every 10 years older) (OR: 0.71 (95% CL: 0.56, 0.90) and higher eGFR (every 30 units higher) (OR: 0.32 (95% CL: 0.22, 0.48) (Table 5).

Discussion

Nearly half (44%) of patients with hypertension in western Kenya have hyperuricemia, with prevalence of 37.6% and 47.3% among males and females respectively. In the last decade interest in uric acid has resurfaced after a long period of inertia, largely reflecting results from experimental studies that show detrimental effects of uric acid on blood pressure and kidney function.

Other reports have also found a high prevalence of hyperuricemia among patients with hypertension. For instance, an older study from Kenya found an incidence of 27.5% among untreated hypertensive patients and 58.3% among the treated hypertensive patients[23]. Studies from West Africa including Nigeria, Cameroon and Mali documented prevalence of hyperuricemia ranging between 49% and 67%[8,11,24]. Higher prevalence in these studies could be explained by the lower cutoff uric acid level (> 5.5mg/dl for both sexes and in our study the cut off was >5.7mg/dl for female patients and >7mg/dl for the male patients)[11]. In addition, those reporting the highest prevalence of hyperuricemia also had longer duration of hypertension (> 40 months) compared to ours which was 24 months[11]. Studies have shown that hyperuricemia increases with duration of hypertension[25].

Studies from Asia and the US have also reported similarly high prevalence of hyperuricemia among patients with hypertension. For instance, in Taiwan a prevalence of 35% among male patients and 45% among female patients was reported[26]. In the US a prevalence of 41.7% was reported[27]. In another study, hyperuricemia was present in 25% of untreated hypertensive subjects, in 50% of subjects taking diuretics, and in >75% of subjects with malignant hypertension[28]. These findings are generally comparable to our results.

The increase in serum uric acid in hypertension may be due to the decrease in renal blood flow that accompanies the hypertensive state, since a low renal blood flow will stimulate urate reabsorption[29]. Hypertension also results in microvascular disease, and this can lead to local tissue ischemia[30]. In addition to the release of lactate that blocks urate secretion in the proximal tubule, ischemia also results in increased uric acid synthesis. In ischemic conditions, ATP is degraded to adenine and xanthine, and there is also increased generation of xanthine oxidase. The increased availability of substrate (xanthine) and enzyme (xanthine oxidase) results in increased uric acid generation as well as oxidant (O_2^-) formation[31].

In our study, factors associated with increased risk of hyperuricemia included higher BMI, dyslipidemia, use of lipid lowering drugs and female gender. A large study from China including 5235 hypertensive patients showed that with an increase of Body Mass Index (BMI), serum uric acid level increased significantly in both sexes [BMI < 25, > or = 3032]. An even larger study including 31,473 participants from the National Health and Nutrition Examination Survey from 1999–2012 in the US demonstrated that the combined effect of hyperuricemia and overweight/obesity on the risk of hypertension is much stronger than any separate one[33].

Other factors associated with hyperuricemia from our study included low eGFR levels and chronic kidney disease stage 3 or worse. Other studies have reported similar findings[8,23,24,34]. Since serum uric acid is eliminated principally by the kidneys its levels increase as the GFR falls. According to Froehlich and Susic demonstrated that an increasing serum uric acid level may be a useful biomarker of hypertension and its consequently deranged renal haemodynamics[33].

An estimated 90% of our participants had dyslipidemia, with only 8.4% being on statins. Other studies have shown a significant correlation between uric acid and dyslipidemia[9,24,35,36]. Triglycerides have been linked to insulin resistance which promotes hypertension through renal tubular sodium re-absorption, augmentation of the sympathetic nervous system reactivity and activation of the renin–angiotensin system[17]. Given that uric acid can also induce the Renin-angiotensin system it is possible that they both have an additive effect on the blood pressure response.

Older age was associated with lower risk of hyperuricemia. This is in line with a study done in Cameroon by Nguedia *et al*[24] and Lin *et al*[26] where they found a significant negative correlation between uric acid and age. However, Teng *et al*[38] reported a contrary result wherein uric acid was associated with the risk of hypertension in the elderly. The differences with our study might be explained by differences in population characteristics.

Other risk factors for hyperuricemia reported in other studies include smoking, alcohol use and diuretic use[24,38,39]. Our study did not show any significant association between hyperuricemia and alcohol or smoking probably due to under-reporting by patients in our study. In our study, diuretic (furosemide and hydrochlorothiazide) use was associated with hyperuricemia but did not reach statistical significance, this could be due to the fact that most patients >95% were on multiple antihypertensive agents, since none was on uricosuric drugs. Use of Calcium Channel Blockers (CCB) and Angiotensin Receptor Blockers (ARB) were associated with a 51% and 61% reduced risk of hyperuricemia respectively. This has been found in other studies as well. The hypouricemic effect of losartan may be due to losartan targeting the urate anion exchange and diminish urate reabsorption in the proximal convoluted tubule[40]. ACE inhibitors and CCBs increase uric acid excretion but the effect is modest[41].

Our study did not establish an association between blood pressure and hyperuricemia, despite the fact that majority (77.8%) of patients in our study had blood pressure of >140/90 mm/hg. This was a cross-sectional study, blood pressure was taken only once therefore some patients might have been misclassified due to white coat effect. This is similar to findings from other studies that

showed that serum uric acid at baseline did not predict the longitudinal changes in both SBP and DBP[34]. This suggests that uric acid level does not influence response to pharmacotherapy for hypertension. Other studies have however found a significant independent association between uric acid with both systolic and diastolic blood pressure[8,24]. An increase in both systolic and diastolic blood pressure was also marked by a corresponding increase in serum uric acid concentration. In the Framingham Heart Study including 3329 participants, it was found that serum uric acid levels was an independent predictor of hypertension incidence and longitudinal BP progression at short-term follow-up[42].

Studies have shown that hyperuricemia independently predicts the risk of stroke[12,13,42,43]. A systematic review and meta-analysis of 16 prospective cohort studies found that the elevated serum uric acid level in adults is associated with a modest but statistically significant increased risk of stroke incidence and mortality[44]. In addition, higher serum urate levels predicted poor outcome at 90 days among patients with stroke independently of other prognostic factors[45]. In patients with diabetes, elevated serum uric acid is thought to play a role, along with obesity, blood pressure, and insulin resistance, in the metabolic syndrome that may be responsible for endothelial dysfunction. We found a higher proportion of strokes among patients with hyperuricemia but the difference was not statistically significant.

One limitation of this study was that blood pressure was only measured during one visit. It is possible that some individuals were misclassified owing to the white coat effect. However, the study had a number of strengths, it was relatively easy and quick to conduct the study, data on all variables were collected at once, we were able to measure the prevalence of all factors under investigation and our findings can be analyzed to generate hypothesis for other in depth research.

In summary, there was a high prevalence (44%) of hyperuricemia among this population in western Kenya. Since there is a significant role of uric acid in development of hypertension and its complications, serum uric acid levels can be used as a marker of disease progression and existence of metabolic syndrome among these patients. Known risk factors for hyperuricemia such as higher BMI, dyslipidemia, female sex, and low eGFR were prevalent in this population. Therefore, we recommend the use of losartan and calcium channel blockers in patients with hyperuricemia and subsequent longitudinal studies to be done to determine utility of uric acid monitoring in blood pressure control.

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