PRINT ISSN 1119-8362
Electronic ISSN 2659-1499

Full-text Available Online at
https://www.ajol.info/index.php/jasem
https://www.bioline.org.br/ja

J. Appl. Sci. Environ. Manage.
Vol. 29 (4) 1261-1267 April 2025

Serum Uric Acid Levels and Renal Function in Hypertensive Patients Visiting a General Outpatient and Cardiology Units of a Government Hospital in Warri, Delta State, Nigeria

***[1]EGUVBE, AO; [2]AGBOGE, OR; [1]GEORGE, EB**

**[1]Department of Chemical Pathology, [2]Department of Radiology, Delta State University, Abraka, Delta State, Nigeria*

**Corresponding Author Email: augustine.eguvbe@delsu.edu.ng*
**ORCID ID: https://orcid.org/ 0000-0002-2722-2186*
**Tel: +2348153685021*

Co-Authors Email: ejetavwo.george@delsu.edu.ng; raygbo@yahoo.com

ABSTRACT: Elevated serum uric acid has been linked to a decline in estimated glomerular filtration rate (eGFR) in men and women. Hence, the objective of this paper was to evaluate the Serum Uric Acid Levels and Renal Function in Hypertensive Patients visiting a General outpatient and Cardiology Units of a Government Hospital in Warri, Delta State, Nigeria using appropriate standard methods. The mean serum uric acid (SUA) levels were significantly higher in the hypertensive group (0.26 ± 0.18 mmol/l) compared to the control group (0.14 ± 0.03 mmol/l, $p < 0.001$). Forty (20.0%) vs. 0 (0.0%) participants had elevated SUA in hypertensive and control groups respectively. The difference in the two groups was statistically significant (P-value <0.001). The estimated glomerular filtration rate (eGFR) was significantly lower in the hypertensive group compared to the control group (0.74 ± 0.27 vs. 0.87 ± 0.20 mL/s/m^2 respectively, $p = 0.001$). Fifty-three (26.5%) of the hypertensives compared to 8 (8.0%) of the controls had impaired renal function. The difference in the two groups was statistically significant (P= 0.004). In the hypertensive group, SUA and eGFR showed a negative correlation ($r = -0.106$), but it was not statistically significant ($p = 0.251$). SUA and renal impairment were significantly higher in hypertensive patients than in controls. There was a negative correlation between SUA and renal function in the hypertensive group, but the relationship was not statistically significant.

DOI: https://dx.doi.org/10.4314/jasem.v29i4.30

Cite this Article as: EGUVBE, A. O; AGBOGE, O. R; GEORGE, E. B (2025) Serum Uric Acid Levels And Renal Function In Hypertensive Patients Visiting A General Outpatient And Cardiology Units Of A Government Hospital In Warri, Delta State, Nigeria. *J. Appl. Sci. Environ. Manage.* 29 (4) 1261-1267

Dates: Received: 28 February 2025; Revised: 29 March 2025; Accepted: 11 April 2025; Published: 30 April 2025

Keywords: cardiovascular disease; hypertension; serum uric acid; renal function; estimated glomerular filtration rate.

Cardiovascular diseases are the main causes of death globally and hypertension is the main modifiable risk factor for cardiovascular disease (Vaduganathan *et al*., 2022, Fuchs *et al*., 2019). Serum uric acid has been implicated in the pathogenesis of high blood pressure and renal disease (Sundstrom *et al*., 2005). Elevated serum uric acid has been linked to a decline in estimated glomerular filtration rate (eGFR) in men and women (Bellomo *et al*., 2010). Uric acid, the last product of purine metabolism in humans, is derived from both endogenous and exogenous sources (Atoe *et al*., 2021, Lee *et al*., 2020). Endogenously, uric acid is produced mainly in the liver, intestines, muscle, and vascular endothelium. Exogenous sources include ingestion of red meat, seafood, fatty food, alcohol, sugar-sweetened drinks (Lee *et al*., 2020). The reference intervals of serum uric acid are 3.5 to 7.2 mg/dl and 2.6 to 6.0 mg/dl in males and

**Corresponding Author Email: augustine.eguvbe@delsu.edu.ng*
**ORCID ID: https://orcid.org/ 0000-0002-2722-2186*
**Tel: +2348153685021*

females respectively (Benn *et al*., 2018). Nevertheless, at lower concentrations of 4.7 and 5.6 mg respectively, serum uric acid has been observed to increase total mortality and cardiovascular mortality (Virdis *et al*., 2020). Hyperuricaemia, defined as increased serum uric acid concentration greater than 6 mg/dl in women and greater than 7 mg/dl in men, is a risk factor for hypertension, chronic kidney disease, metabolic syndrome and its components, and nonalcoholic fatty liver disease (Du *et al*., 2024, Sanchez-Lozada *et al*., 2020). Hyperuricaemia has been reported to play a role in the pathophysiology of gout, urolithiasis, hypertension, chronic kidney disease, congestive heart failure, the metabolic syndrome, type 2 diabetes mellitus, and atherosclerosis (Gustaffsson *et al*., 2013). The suggested mechanisms linking serum uric acid to the pathogenesis of hypertension include uric acid-induced renal afferent arteriolopathy, activation of the renin-angiotensin-aldosterone system, oxidative stress, inflammation, and endothelial dysfunction (Piani *et al*., 2021).

Studies investigating the relationship between serum uric acid and renal function in hypertensive patients are scarce in this environment. Hence, the objective of this paper was to evaluate the Serum Uric Acid Levels and Renal Function in Hypertensive Patients visiting a General outpatient and Cardiology Units of a Government Hospital in Warri, Delta State.

MATERIALS AND METHODS

Research design and population: This was a cross-sectional research carried out between November 2022 and February 2024 at Central Hospital, Warri, Delta State. This research enrolled three hundred individuals made up of two hundred hypertensives and one hundred normotensive controls. Participants were selected from patients visiting the General outpatient Department and the Cardiology Unit of Central Hospital, Warri. Participants included in this research were aged eighteen years and above, hypertensive (blood pressure ≥ 140/90 mmHg) and normotensive (blood pressure below 140/90 mmHg). The exclusion criteria included the following: diabetes mellitus, renal disease, malignancy and critically ill patients. Central Hospital Warri is one of the biggest secondary health facilities in Delta State. They render general and specialized health services to the population of Warri and Delta State.

Sample size and sampling procedure: The sample size of this research was calculated with the aid of the formula for cross-sectional studies: $n = (Z^2Pq)/d^2$, where n = sample size, Z = standard deviation, P = prevalence of hypertension (15%) (Akinlua *et al*., 2015), q = 1-P and d = degree of precision to be used (0.05). The research population was selected consecutively from patients visiting the General outpatient and Cardiology unit of Central Hospital, Warri. A written informed consent was gotten from each study participant.

Demographic and baseline characteristics: Direct interviews, using a structured questionnaire, was employed to obtain data such as identification number, age, gender, weight, height, waist circumference, medical history and laboratory results. Measurements of body weight, height and waist circumference were taken and body mass index (BMI) was calculated from the weight (kg) divided by the height (meter) squared. Averages of two blood pressure (BP) measurements were taken with a mercury sphygmomanometer after at least ten minutes of rest by the patient. Obesity was defined as BMI that is 30 kg/m^2 or greater.

Blood and urine samples: About five to ten milliliters (5-10 ml) of venous blood and spot urine were taken from each participant. The blood was collected into a plain bottle and allowed to clot. The specimen was centrifuged to obtain the serum component which was kept at -20℃ until analysis. The urine was collected into a universal container and stored in the refrigerator at 2 to 8℃ . Serum and urine creatinine concentrations were assayed on a spectrophotometer using the kinetic modification of the Jaffe procedure. Serum uric acid was assayed using enzymatic colorimetric method by Fortress Diagnostics. Estimated glomerular filtration rate (eGFR) was calculated from serum creatinine using the Modification of Diet for Renal Disease formula based on age, sex, race and serum creatinine (Levey *et al*., 2000).

Statistical analysis: Data analysis was performed using SPSS version 23. Continuous variables were examined for normality. Normally distributed continuous variables were presented as mean, standard deviation, and ranges while skewed variables were presented as median and interquartile ranges. Categorical data were summarized using frequencies and percentages. The differences in means of continuous variables between the hypertensive group and controls were compared using the student T test. Chi square test was used for univariate analysis. The Pearson's correlation coefficient was used to correlate serum uric acid with eGFR. Statistical significance was set at <0.05.

Ethical Clearance: This study was approved by the ethics and research committee (protocol number:

CHW/ECC VOL 1/286) of the Delta State Central Hospital, Warri.

RESULTS AND DISCUSSION

This present research recruited three hundred participants comprised of two hundred hypertensive and one hundred normotensive individuals. Demographic and social parameters of the participants are presented in Table 1. Notably, the age distribution differed significantly ($p < 0.001$) between the hypertensive and control groups. The most frequent age groups in the hypertensives were 50-59 years and 60-69 years while 40-49 years was the most frequent age group in the controls. In both groups, the age ranges were 19-92 years and 22-66 years respectively. The hypertensive group had a significantly higher mean age (58.23 ± 13.07 years) than the control group (43.78 ± 10.68 years, $p < 0.001$). Females predominated in the hypertensive group (73.5% vs. 26.5%, $p = 0.007$).

Anthropometrics: Waist circumference was significantly higher in the hypertensive group (98.78 ± 13.17 cm vs. 91.25 ± 8.14 cm, $p < 0.001$) (Table 1). No significant differences were observed in weight, height, or BMI (Table).

Social history: Alcohol consumption was more prevalent among hypertensive patients. Thirty-three (16.5%) of the hypertensive participants compared to 7 (7.0%) of the controls drink alcohol. The difference in both groups was not significant ($p = 0.063$) (Table 1). Smoking habits did not differ significantly between the two groups ($p = 0.246$) (Table 1).

Table 1: Demographic and Social Parameters

Variable	Hypertensive N=200 n (%)	Control N=100 n (%)	Statistics	P –values
Age			37.184[f]	<0.001*
< 30 yrs	4 (2.0)	12 (12.0)		
30 -39	5 (2.5)	18 (18.0)		
40-49	43 (21.5)	38 (38.0)		
50-59	53 (26.5)	25 (25.0)		
60-69	53 (26.5)	7 (7.0)		
≥70yrs	42 (21.0)	0 (0.0)		
Mean ± SD	58.23 ± 13.07	43.78 ± 10.68	54.903[t]	<0.001*
Modal age	67 years	42 years		
Median age	58yrs	45yrs		
Range	92-19= 73yrs	66-22= 44yrs		
Sex			7.200[c]	0.007*
Male	53 (26.5)	53 (53.0)		
Female	147 (73.5)	47 (47.0)		
Weight (kg) (Mean ± SD)	76.13 ± 17.88	76.19 ± 9.21	0.005[t]	0.946
Height (m) (Mean ± SD)	1.61 ± 0.07	1.63 ± 0.08	1.600[t]	0.208
BMI (Kg/m^2)			3.866[f]	0.276
Underweight	3 (1.5)	2 (2.0)		
Normal	57 (28.5)	18 (18.0)		
Overweight	68 (34.0)	48 (48.0)		
Obese	72 (36.0)	32 (32.0)		
Mean ± SD	28.88 ± 6.47	28.56 ± 4.67	0.114[t]	0.736
Waist circumference (cm) (Mean ± SD)	98.78 ± 13.17	91.25 ± 8.14	16.454[t]	<0.001*
Alcohol	33 (16.5)	7 (7.0)	3.462[c]	0.063
Smoking	0 (0.0)	1 (1.0)	1.345[c]	0.246

f= Fisher's exact, t= Student's *T*-test, c= Chi-square,BMI= Body mass index, SD= standard deviation, *=statistically significant ($p<0.05$)

Fig. 1 below shows the mean systolic and diastolic blood pressures in both groups.

Renal Function: Table 2 presents the biochemical variables of the study participants. The mean serum creatinine levels were higher in the hypertensive group compared to the control group, but the difference was not statistically significant (106.1 ± 94.6 vs. 84.9 ± 15.0 µmol/l respectively, $p = 0.095$). Notably, the hypertensive group exhibited a wide range of serum creatinine values (35.4-831 µmol/l), whereas the control group had a narrower range (53.0-114.9 µmol/l). The estimated glomerular filtration rate (eGFR) was significantly lower in the hypertensive group compared to the control group (0.74 ± 0.27 vs. 0.87 ± 0.20 mL/s/m^2 respectively, $p = 0.001$). The eGFR values in the hypertensive group ranged from 0.05 to 1.23 mL/s/m^2, whereas the control group ranged from 0.53 to 1.20 mL/s/m^2.

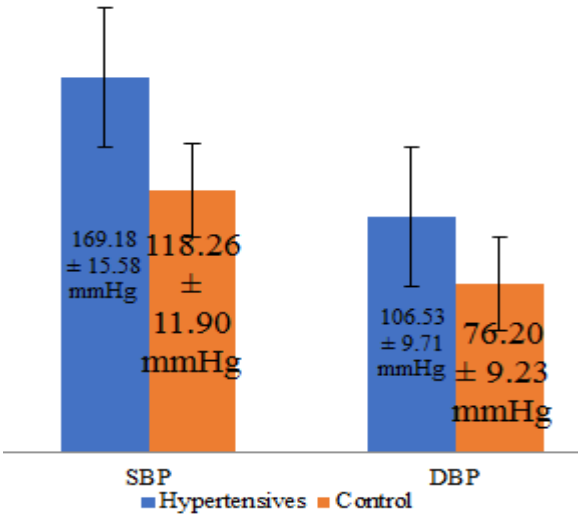

Fig. 1: Systolic and diastolic Blood pressure between the two groups
SBP= Systolic blood pressure, DBP = Diastolic blood pressure SBP (Student T-test= 495.139, P< 0.001), DBP (Student T-test= 438.814, P< 0.001)

Figure 3 shows that 147 (73.5%) hypertensives had normal renal status, while 53 (26.5%) had impaired renal function. In contrast, 92 (92.0%) controls had normal results, while 8 (8.0%) had impaired renal function. The difference in the two groups was statistically significant (P= 0.004).

Serum uric acid: The mean serum uric acid (SUA) levels were significantly higher in the hypertensive group (0.26 ± 0.18 mmol/l) compared to the control group (0.14 ± 0.03 mmol/l, p < 0.001). The SUA values in the hypertensive group ranged from 0.08 to 1.78 mmol/l, whereas the control group ranged from 0.11 to 0.24 mmol/l. Figure 2 shows that 160 (80.0%) vs. 100 (100.0%) had normal SUA in hypertensive and control groups respectively, while 40 (20.0%) vs. 0 (0.0%) had elevated SUA in hypertensive and control groups respectively. The difference in the two groups was statistically significant (P-value <0.001).

Table 2: Biochemical variables of study participants

Variable	Hypertensive N=200 n (%)	Control N=100 n (%)	Statistics	P – values
Serum Creatinine (µmol/l)				
Mean ± SD	106.1 ± 94.6	84.9 ± 15.0	2.809[t]	0.095
Minimum	35.4	53.0		
Maximum	831.0	114.9		
eGFR (mL/s/m^2)				
Mean ± SD	0.74 ± 0.27	0.87 ± 0.20	10.465[t]	0.001*
Minimum	0.05	0.53		
Maximum	1.23	1.20		
SUA (mmol/L)				
Mean ± SD	0.26 ± 0.18	0.14 ± 0.03	26.020[t]	<0.001*
Minimum	0.08	0.11		
Maximum	1.78	0.24		

*eGFR= Glomerular filtration rate, SUA = Serum uric acid, t= Student's T-test, SD= standard deviation, *=statistically significant (p<0.05)*

The Proportion of hypertensive and controls with normal and elevated Serum Uric Acid is shown in Fig. 2. A bar chart comparing the frequency of renal impairment in hypertensive and control groups is shown in Fig. 3. The correlation analysis between serum uric acid (SUA) and estimated glomerular filtration rate (eGFR) revealed in Table 3. In the hypertensive group, SUA and eGFR showed a weak negative correlation (r = -0.106), but it was not statistically significant (p = 0.251). In the control group, SUA and eGFR showed a weak positive correlation (r = 0.162), but it was also not statistically significant (p = 0.216). This present research documented significantly higher levels of SUA and renal impairment in hypertensive patients than in controls, implying a higher risk of cardiovascular and renal diseases in hypertensive patients.

Table 3: Correlation between SUA with eGFR in hypertensive patients and controls

		SUA	eGFR
Hypertensive group			
SUA	Pearson Correlation	1	- 0.106
	Sig. (2-tailed)		0.251
	N	200	200
eGFR	Pearson Correlation	- 0.106	1
	Sig. (2-tailed)	0.251	
	N	200	200
Control group			
SUA	Pearson Correlation	1	0.162
	Sig. (2-tailed)		0.216
	N	100	100
eGFR	Pearson Correlation	0.162	1
	Sig. (2-tailed)	0.216	
	N	100	100

SUA = Serum uric acid, eGFR= Estimated Glomerular Filtration Rate, Correlation is significant at < 0.05 (*)

There was a negative correlation between SUA and renal function in the hypertensive group, but the relationship was not statistically significant.

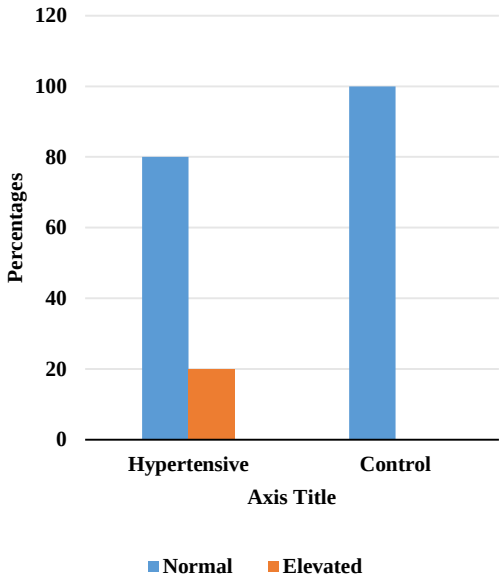

Fig. 2: Proportion of hypertensives and controls with normal and elevated Serum uric acid
X^2= 13.846, P< 0.001
Elevated Serum Uric acid (Hyperuricemia) is defined as uric acid level greater than 7 mg/dl in males and greater than 6 mg/dl in females

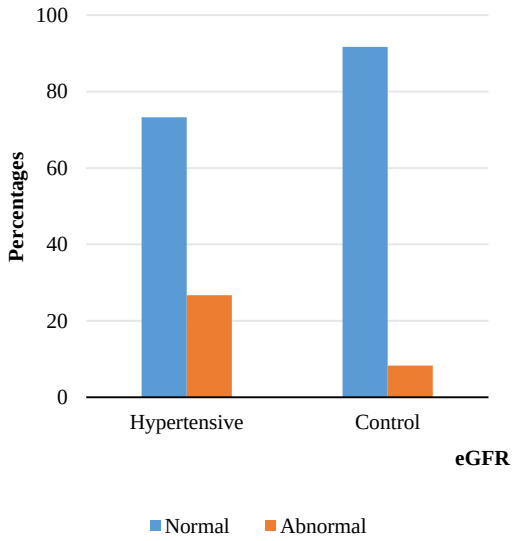

Fig. 3: Proportion of hypertensive and controls with impaired renal function using eGFR
X^2= 8.233, P= 0.004, eGFR= Estimated Glomerular Filtration Rate

The prevalence of hyperuricaemia in hypertensive patients in this present study was 20%, which was significantly higher than in the controls. However, a wide variation of prevalence has been reported by several studies. A research carried out in western Nigeria among hypertensive patients reported a prevalence of 36.7% (Fasae *et al*., 2018). Adewuya *et al*. (2020) reported a prevalence of 46.9% in a cross-sectional study of 271 hypertensive patients (Adewuya *et al*., 2020). In another Nigerian study, Ofori *et al*. (2015) reported a prevalence of 46.9% among newly diagnosed hypertensives (Ofori *et al*., 2015). A prevalence of 18.2% was reported by Zhang *et al*. (2022) in a research among 3505 hypertensive individuals aged 50-79 years (Zhang *et al*., 2022). In a hospital-based cross-sectional study among 168 hypertensive patients, the prevalence was 25.0% (Mehta *et al*., 2021). Mishra *et al*. (2017) reported a prevalence of 26.0% in a case control study involving 100 hypertensive adults (Mishra *et al*., 2017). The prevalence was 31.8% in a community-based cross-sectional study in Cameroon, among newly diagnosed hypertensive patients (Kamdem *et al*., 2016). The disparity in the prevalence of hyperuricaemia could be attributed to variations in diet, alcohol ingestion, body weight, time of day, and state of hydration (Shaffer *et al*., 2016).

This present study recorded a 26.5% prevalence of renal impairment (eGFR < 60 mls/min/1.73 m^2) in the hypertensive group, which was significantly higher than in the controls. Various studies have investigated the prevalence of renal impairment in hypertensive patients. The prevalence was 50.0% in a cross-sectional study involving 204 hypertensive patients in western Nigeria (Oluwademilade *et al*., 2020). The Berlin Initiative Study (BIS) reported a prevalence of 38.0 to 62.0% depending on the estimation equation in older adults (Ebert *et al*., 2016). The prevalence of renal impairment in a population of diabetic hypertensive patients was 35.5% (Sweileh *et al*., 2009). The differences in the prevalence of renal impairment in the various studies could be ascribed to the many factors that are associated with renal insufficiency. They include age, diabetes, hypertension, hyperlipidaemia, hyperuricaemia, use of nephrotoxic drugs, coronary heart disease and history of chronic kidney disease (Liu *et al*., 2012).

Also documented in this present study was a negative correlation between SUA and renal function in the hypertensive group, but the relationship was not statistically significant. Many studies have reported an indirect relationship between SUA and renal function. In a research among 2601 adult

hypertensives, it was documented that increase in SUA was associated with a decline in renal function (Sedaghat *et al*., 2013). In a cohort study of over 5000 participants, higher levels of SUA were associated with renal impairment (Chonchol *et al*., 2007). Also, in a study of uric acid and renal impairment in hypertensive patients, it was reported that hyperuricaemia was associated with a decline in renal function (Hung *et al*., 2022). Furthermore, a research to investigate SUA and arterial hypertension reported an indirect link between SUA levels and eGFR values (Buzas *et al*., 2018). Uric acid can cause renal insufficiency through mechanisms such as precipitation and obstruction in tubules, endothelial dysfunction, activation of the renin-angiotensin-aldosterone system, inflammation, and oxidative stress (Srivastava *et al*., 2017).

The limitation of this research is that being a hospital-based study, it may not truly represent the population.

Conclusion: SUA and renal impairment were significantly higher in hypertensive patients than in controls, implying a higher risk of cardiovascular and renal diseases in hypertensive patients. There was a negative correlation between SUA and renal function in the hypertensive group, but the relationship was not statistically significant. For hypertensive patients, regular determination of SUA and renal function is required for proper assessment of cardiovascular risk and renal insufficiency.

Declaration of Conflict of Interest: The authors declare no conflict of interest.

Data Availability: Data are available upon request from the first author or corresponding author or any of the other authors

REFERENCES

Adewuya ,OA; Ajayi, EA; Adebayo, RA; Ojo, OE; Olaoye, OB (2020). Serum uric acid and left ventricular hypertrophy in hypertensive patients in Ad0-Ekiti. *Pan Afr. Med. J.* 36(1).

Akinlua, JT; Meakin, R; Umar, AM; Freemantle, N (2015). Current prevalence pattern of hypertension in Nigeria: a systematic review. *PLoS. One*. 10:e0140021.

Atoe, K; Eguvbe, AO (2021). Assessment of serum uric acid levels in patients with cervical intraepithelial neoplasia in Benin City. *Annals Clin. Biomed. Res.* 2:99. doi:10.4081/acbr.2021.99.

Bellomo, G; Venanzi, S; Verdura,C; Saronio, P; Esposito, A; Timio, M (2010). Association of uric acid with change in kidney function in healthy normotensive individuals. *AJKD*. 56(2):264-272.

Benn, CL; Dua, P; Gurrell, R; London, P; Pike, A; Storer, RI; Vangjeli C (2018). Physiology of hyperuricemia and urate-lowering treatments. *Front. Med*. 5:160.

Buzas, R; Tautu, OF; Dorobantu, M; Ivan, V; Lighezan, D (2018). Serum uric acid and arterial hypertension- data from Sephar III Survey. *PLoS. ONE.* 13(7):e0199865.

Chonchol, M; Shlipak, MG; Katz, R; Sarnak, MJ; Newman, AB; Siscovick, DS; Kestenbaum, B; Carney, JK; Fried, LF (2007). Relationship of uric acid with progression of kidney disease. *AJKD.* 50(2):239-247.

Du, L; Zong, Y; Li, H; Wang, D; Xie, L; Yang, B; Pang, Y; Zhang, C; Zhong, Z; Gao, J (2024). Hyperuricaemia and its related diseases: mechanisms and advances in therapy. *Sig. Transduct. Target*. Ther. 9:212.

Ebert, N; Jacob, O; Gaedeke, J; Giet, M; Kuhlman, MK; Markus, P; Mielke, N; Schuchardt, M; Tolle, M; Wenning, V; Schaeffner, ES (2016). Prevalence of reduced kidney function and albuminuria in older adults: the Berlin Initiative Study. *Nephrol. Dial. Transplant.* 0:1-9.

Fasae, AJ; Busari, OA; Oluyombo, R; Yusuf, M; Opadijo, OG; Omotoso, AB (2018). Hyperuricemia and its correlation with target organ damage and electrocardiographic changes in newly diagnosed adult Nigerian hypertensive patients. *C. M. & R.* 7(1):1-7. doi: 10.11648/j.cmr.20180701.11.

Fuchs, FD; Whelton, P (2019). High blood pressure and cardiovascular disease. *Hypertens*. 75(2).Gustafsson, D; Unwin, R (2013). The pathophysiology of hyperuricaemia and its possible relationship to cardiovascular disease morbidity and mortality. *BMC. Nephrol*. 14:164.

Hung, Y-H; Huang, C-C; Lin, L-Y; Chen, J-W (2022). Uric acid and impairment of renal function in non-diabetic hypertensive patients. *Front. Med.* 8:746886.

Kamdem, F; Doualla, M-S; Lekpa, FK; Temfack, E; Nouga, YN; Donfack, OS; Ozudie, A; Kingue, S

(2016). Prevalence and factors associated with hyperuricaemia in newly diagnosed and untreated hypertensives in Sub-Saharan African setting. *Arch. Cardiovasc. Dis.* 109(10):527-532.Lee, SJ; Oh, BK; Sung, K-C (2020). Uric acid and cardiometabolic diseases. *Clin. Hypertens.* 26:13.

Levey, AS; Greene, T; Kusek, J; Beck, G (2000). A simplified equation to predict glomerular filtration rate from serum creatinine. *J. Am. Soc. Nephrol.* 11:155A.

Liu, Q; Li, Z; Wang, H; Chen, X; Dong, X; Mao, H; Tan, J; Luo, N; Johnson, RJ; Chen, W; Yu, X; Chen, w (2012). High prevalence and associated risk factors for impaired renal function and urinary abnormalities in a rural population from southern China. *PLoS. One.* 7(10):e47100.

Mehta, R; Koirala, P; Mallick, RL; Parajuli, S; Jha, R (2021). Prevalence of hyperuricemia and its association with sociodemography among hypertensive patients of Birat Medical College Teaching Hospital. *BJHS*. 6(1)14.1388-91.

Mishra, A; Gupta, P; Gupta, A; Verna, SK; Chaurasia, AK; Sharma, D (2017). Prevalence and association of hyperuricemia in patients of newly diagnosed essential hypertension. *IJCMR*. 4(2).

Ofori, SN; Akpa, M (2015). Relationship between uric acid and left ventricular mass and geometry in Nigerian patients with untreated essential hypertension. *Niger. J. Cardiol.* 12(2):129-35.

Oluwademilade, OJ; Ajani, GO; Kolawole, FT; Obajolowo, OO; Olabinri, EO; Agboola, SM (2020). Burden of chronic kidney disease in hypertensive patients in medical outpatient clinic of a rural tertiary hospital. *J. Hypertens. Manag.* 6(1).

Piani, F; Cicero, AFG; Borghi, C (2021). Uric acid and hypertension: prognostic role and guide for treatment. *J. Clin. Med.* 10(3):448.

Sanchez-Lozada, LG; Rodriguez-Iturbe, B; Kelly, EE; Nakagawa, T; Madero, M; Feig, DI; Borghi, C; Piani, F; Cara-Fuentes, G; Bjornstad, P; Lanaspa, MA; Johnson, RJ (2020). Uric acid and hypertension: an update with recommendation. *Am. J. Hypertens*. 33(7):583-594.

Sedaghat, S; Hoorn, EJ; van Rooij, FJA; Hofman, A; Franco, OH; Witterman, JCM (2013). Serum uric acid and chronic kidney disease: the role of hypertension. *PLoS. ONE.* 8(11):e76827.

Shaffer, A; Rahn, E; Saag, K; Mudano, A; Gaffo, A (2021). Variation in serum urate levels in the absence of gout and urate lowering therapy. *BMC. Rheumatol.* 5:32.

Srivastava, A; Kaze, AD; McMullan, CJ; Isakova, T; Waikar, SS (2017). Uric acid and the risks of kidney failure and death in individuals with CKD. *Kidney Dis.* 71(3):362-370.

Sundstrom, J; Sullivan, LD; Agostino, BR; Levy, D; Kannel, WB; Vasan, RS (2005). Relations of serum uric acid to longitudinal blood pressure tracking and hypertension incidence. *Hypertens*. 45:28-33.

Sweileh, WB; Sawalha, AF; Zyoud, SH; Al-Jabil, SW; Shraim, NY (2009). Prevalence of reduced renal function among diabetic hypertensive patients. *Int. J. Physiol. Pathophysiol. Pharmacol.* 1:41-47.

Vaduganathan, M; Mensah, GA; Turco, JV; Fuster, V; Roth, GA (2022). The global burden of cardiovascular diseases and risk: a compass for future health. *JACC*. 80(25):2361-2371.

Virdis, A; Masi, S; Casiglia, E; Tikhonoff, V; Cicero, AFG; Ungar, A; Rivasi, G; Salvetti, M; Barbagallo, CM; Bombelli, M, et al (2020). Identification of the uric acid thresholds predicting an increased total and cardiovascular mortality over 20 years. *Hypertens.* 75:302-308.

Zhang, Y; Nie, FQ; Huang, XB; Tang, W; Hu, R; Zhang, WQ; Liu, J-X; Xu, RH; Liu, Y; Wei, D; Wang, T-D; Fan, X (2022). High prevalence and low awareness of hyperuricemia in hypertensive patients among adults aged 50-79 years in Southwest China. *BMC. Cardiovasc. Disord.* 22(2).