



Evaluation of Serum Levels of Lipoprotein-A and Uric Acid and Their Correlation in Hypertensive Patients in Delta State, Nigeria

*¹EGUVBE, AO; ²GEORGE, EB; ³ESSIET, DF; ⁴SLATER, HE

¹Department of Chemical Pathology, Delta State University, Abraka, Delta State, Nigeria.

²Department of Chemical Pathology, Delta State University, Abraka, Delta State, Nigeria.

³ Department of Chemical Pathology, Delta State University, Abraka, Delta State, Nigeria.

⁴Department of Medicine, Central Hospital Warri, Delta State, Nigeria

*Corresponding Author Email: augustine.eguvbe@delsu.edu.ng

*ORCID ID: <https://orcid.org/0000-0002-2722-2186>

Co-Authors Email: dominic-f@delsu.edu.ng; ejetavwo.george@delsu.edu.ng; henryslater72@gmail.com

ABSTRACT: Lipoprotein (a) is an independent risk factor for cardiovascular disease. Also, elevated serum uric acid concentration is linked with an increased risk of cardiovascular disease. Therefore, the objective of this paper was to evaluate the serum levels of Lp (a) and uric acid and their correlation in hypertensive patients in Delta State, Nigeria. The study was conducted among 200 hypertensive and 100 normotensive individuals in a hospital-based cross-sectional investigation. Data obtained showed that one hundred and fifteen (57.5%) hypertensive and 15 (15%) normotensive controls had elevated plasma concentration of Lp (a). The lipoprotein (a) levels in the hypertensive had a mean of 32.8 ± 16.6 mg/dl. The controls had a mean of 16.9 ± 13.9 mg/dl. The difference in mean Lp (a) levels was statistically significant ($p < 0.001$). Uric acid level in the hypertensive had a mean of 4.1 ± 1.8 mg/dL. In the controls, the mean value was 2.7 ± 1.2 mg/dL. The difference in mean was statistically significant ($p < 0.001$). Forty-two (21.0%) of the hypertensive compared to 5 (5.0%) of the controls had hyperuricaemia ($p = 0.005$). Among the hypertensive, Lp (a) was positively associated with uric acid ($r = 0.238$, $p = 0.009$). Lp (a) and uric acid levels in hypertensive patients were significantly higher than in the controls. Among the hypertensive population, Lp (a) was positively associated with uric acid. Thus, routine screening of hypertensive patients for Lp (a) can be used to demonstrate the presence of cardiovascular risk.

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Hypertension, which is a key modifiable risk factor for cardiovascular disease, is the main cause of morbidity and mortality in Nigeria (Oguanobi *et al.*, 2021, Adelaye *et al.*, 2015). Lipoprotein (a) (Lp [a]) is an independent risk factor for cardiovascular disease and higher serum levels are found in hypertensive patients than normotensive individuals (Brosolo *et al.*, 2021, Papadakis *et al.*, 1999). Also, hyperuricemia is more prevalent in hypertensive patients and is linked with an increased risk of cardiovascular disease (Nagahama *et al.*, 2004, Sundstrom *et al.*, 2005). Increased serum

concentrations of Lp (a) in hypertensive patients is suggestive of the presence and severity of hypertensive vascular damage (Brosolo *et al.*, 2021). Increased Lp (a) concentrations has been reported as an independent and causal risk factor for atherosclerotic cardiovascular disease and calcific aortic valve disease (Ward *et al.*, 2021). The pathogenic mechanisms of Lp (a) include proatherogenic, proinflammatory and prothrombotic effects (Brosolo *et al.*, 2021). Lp (a) is made up of a low-density lipoprotein (LDL)-like portion attached

*Corresponding Author Email: augustine.eguvbe@delsu.edu.ng

*ORCID ID: <https://orcid.org/0000-0002-2722-2186>

covalently to apolipoprotein (a) (Tsimikas *et al.*, 2020). It is produced in the liver and concentrations higher than 20-30 mg/dl are linked to a two-fold risk of having coronary artery disease (Maranhao *et al.*, 2014).

Uric acid is the last product of purine metabolism in humans (Atoe *et al.*, 2021). It is a waste product that is excreted mainly by the kidney and intestine (Remedious *et al.*, 2012). Hyperuricemia is linked with an increased risk for hypertension, independent of typical risk factors of hypertension (Grayson *et al.*, 2011). Studies on animals reveal that hyperuricaemia predisposes to hypertension by mechanisms such as inflammatory and vascular changes in the renal microcirculation, activation of the renin-angiotensin system and endothelial dysfunction (Sundstrom *et al.*, 2005). Uric acid has been linked with insulin resistance, stroke, heart disease, and chronic kidney disease. Studies have reported that reducing the concentration of uric acid has been seen to lower blood pressure in hypertensive and prehypertensive patients. Thus, serum uric acid has been documented as a potentially modifiable cardiovascular risk factor (Kuwabara *et al.*, 2018). Studies correlating Lp (a) levels with serum uric acid have not been done in this environment. Hence, the objective of this paper was to evaluate the serum levels of Lp (a) and uric acid and their correlation in hypertensive patients in Delta State, Nigeria.

MATERIALS AND METHODS

Research design and population: This was a hospital-based cross-sectional study conducted between October 2022 and December 2023 at the Delta State Central Hospital, Warri. Three hundred participants consisting of two hundred hypertensive and one hundred normotensive individuals were involved in the study. The hypertensive and normotensive cases were patients attending the General outpatient and Cardiology Units of the Delta State Central Hospital, Warri. The inclusion criteria included participants who were aged eighteen years and above, diagnosed with hypertension (blood pressure \geq 140/90 mmHg) and normotensive individuals with blood pressure below 140/90 mmHg. Participants with a history of diabetes mellitus, chronic alcoholism or smoking, renal disease, liver disease, pregnancy, malignancy, hypothyroidism/hyperthyroidism, hypertriglyceridemia ($>$ 400 mg/dl), hereditary familial hypercholesterolemia/dyslipoproteinemia, women on hormone replacement therapy were excluded from the study. Patients on phenytoin, carbamazepine, metformin, pentoxifylline, methotrexate, vitamin D supplements and lipid lowering agents were also excluded. Central Hospital

Warri is a secondary health facility with 254 beds. They render general and specialized medical and surgical services.

Sample size and sampling procedure: The sample size was calculated from the formula for a cross-sectional study: $n = (Z^2Pq)/d^2$, where n = sample size, Z = standard deviation, P = prevalence of hypertension, $q = 1-P$ and d = degree of precision to be used (0.05). Consecutive sampling was used to recruit participants. Written informed consent was obtained from each study participant. The study used a structured interviewer-administered questionnaire, which included identification number, age, gender, weight, height, waist circumference, medical history and laboratory results.

Demographic and baseline characteristics: The study used a structured interviewer-administered questionnaire, which included identification number, age, gender, weight, height, waist circumference, alcohol intake and smoking history, medical history and laboratory results. A detailed history was taken including past or current co-morbidities. BP was measured using a mercury sphygmomanometer after the patient had rested for at least ten minutes. The reading at the first appearance of the korotkoff sound (phase I) was taken as the systolic BP and that at its disappearance (phase V) was taken as the diastolic blood pressure. Hypertension was defined as values 140/90 mmHg and above on two or more different occasions (Jordan *et al.*, 2018). Height was measured by a stadiometer. Weight was measured using an electronic patient weighing scale. Body mass index (BMI) was calculated from the weight (kg) divided by the height (meter) squared. Obesity was defined as BMI equal to or greater than 30 kg/m². Waist circumference (WC) was measured with a measuring tape at the approximate midpoint between the lower margin of the palpable rib and the top of the iliac crest.

Blood sample: About five to ten milliliters (5-10 ml) of blood was collected from each participant by venipuncture into a plain bottle and allowed to clot. The specimen was centrifuged at 3000 rpm. The serum was separated from cells using a Pasteur pipette and kept at -20°C until analysis. Morning and evening temperature recordings were taken to monitor temperature of the freezer. Serum creatinine and urine creatinine were assayed on a spectrophotometer using the kinetic modification of the Jaffe procedure. Estimated glomerular filtration rate (eGFR) was predicted 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 was analyzed with SPSS version 23. Continuous variables including age, blood pressure measurements and anthropometric variables, and biochemical parameters were tested for normality. Normally distributed continuous variables were summarized as mean, standard deviation, and ranges. Categorical variables 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 Lipoprotein (a) with uric acid levels in hypertensives and controls. Statistical significance was set at <0.05.

Ethical Clearance: This study was approved by the ethics and research committee of the Delta State Central Hospital, Warri.

RESULTS AND DISCUSSION

A total of 300 participants were recruited into the study including 200 hypertensives and 100 controls. The hypertensives had an age range of 19.0 – 92.0 years with a mean age and standard deviation of 58.2 ± 13.1 years and the controls had an age range of 22.0 – 66.0 years with a mean age and standard deviation of 43.8 ± 10.7 years. The hypertensives were significantly older in years than the controls ($p = 0.001$). The hypertensives include 147 (73.5%) females and 53 (26.5%) males while the controls included 53 (53.0%) females and 47 (47.0%) males. The difference in the sex distribution was also statistically significant ($p = 0.007$) (Table 1).

Anthropometrics: The body weight of the hypertensives and the controls did not differ significantly as shown in table 1 (76.0 ± 18.0 vs. 76.2 ± 9.2 kg respectively, $p = 0.946$). The body height also did not differ significantly between the hypertensives and controls (1.62 ± 0.07 vs. 1.64 ± 0.09 , respectively, $p = 0.208$). In like manner, the BMI did not differ significantly between them (28.9 ± 6.5 vs. 28.8 ± 5.1 kg/m², $p = 0.891$) (Table 2). Seventy-two (36.0%) of the hypertensives and 35 (35.0%) of the controls were obese (BMI ≥ 30 kg/m²). The difference in proportion was not statistically significant ($p = 0.635$).

Social history: Only one study participant reported smoking and the subject is in the control group. There was no significant difference in the distribution of smoking in the study groups ($p = 0.723$). Thirty-three (16.5%) of the hypertensives drink alcohol compared to 7 (7.0%) of the controls and the difference in proportion was not statistically significant ($p = 0.104$).

Renal function: Serum creatinine was higher in the hypertensives than in the controls but the difference in mean did not reach statistical significance (1.2 ± 1.1 vs. 1.0 ± 0.2 mg/dl, $p = 0.073$). The mean eGFR was significantly lower in the hypertensive population than in the controls (80.6 ± 36.9 vs. 91.8 ± 24.2 ml/m²/min, $p = 0.035$). Seventy-two (36.0%) of the hypertensive subjects had renal impairment (eGFR < 60ml/m²/min) compared to 32 (32.0%) of the controls. The difference in proportion was not statistically significant $p = 0.579$).

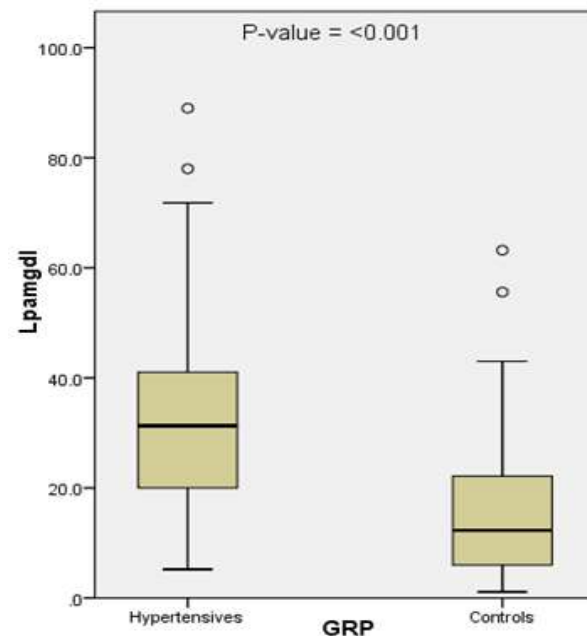


Fig 1: Boxplot showing the distribution of lipoprotein (a) in the study population

Lipoprotein-A: Only one hundred and fifteen (57.5%) hypertensives and fifteen (15%) normotensive controls had plasma concentration of Lp (a) above 30 mg/dl. The lipoprotein (a) levels in the hypertensives ranged from 5.2 – 89.0 mg/dl with a mean of 32.8 ± 16.6 mg/dl. The controls had a range of 1.1 – 63.2 mg/dl with a mean of 16.9 ± 13.9 mg/dl. The difference in mean Lp (a) levels was statistically significant ($p < 0.001$) (Figure I and Table 2)

Uric acid levels: Uric acid level ranged from 1.4 – 9.8mg/dL in the hypertensives with a mean of 4.1 ± 1.8 mg/dL. In the controls, the mean value was 2.7 ± 1.2 mg/dL. The difference in mean was statistically significant ($p < 0.001$) (Figure II and Table 2). Forty-two (21.0%) of the hypertensives compared to 5 (5.0%) of the controls had hyperuricaemia ($p = 0.005$). (Figure 2).

Correlation of Lp (a) levels with Uric acid in hypertensives and controls: In the hypertensives, Lp (a) correlated positively with uric acid ($r = 0.238, p = 0.009$). However, in the controls, there was no significant correlations between Lp (a) and uric acid ($r = 0.006, p = 0.966$) (Table 3, Figure III). In the controls, there were no significant correlations between Lp (a), age, blood pressure indices anthropometric variables and renal parameters (serum creatinine and eGFR). We conducted this study to evaluate the serum levels of Lp (a) and uric acid and their correlation in hypertensive patients. We found that the Lp (a) and uric acid levels in hypertensive patients were significantly higher than in the controls. Also, among the hypertensive population, Lp (a) was positively associated with uric acid. In this present study, Lp (a) was significantly higher in the hypertensive patients, suggesting a higher risk of atherosclerotic cardiovascular disease in hypertension. Many other studies have reported similar findings. Significantly elevated Lp (a) was reported in hypertensives in a cross-sectional study carried out among 100 newly diagnosed cases with hypertension and 50 normotensive controls (Mahto *et al.*, 2022). In a study by Bhavani *et al.*, on plasma Lp (a) levels in patients with untreated essential hypertension, a significantly higher Lp (a) level in hypertensive patients was recorded (Bhavani *et al.*, 2003). Also, a significantly higher Lp (a) level in hypertensives compared to controls was reported in a study done among hypertensive patients in a tertiary care hospital (Sur *et al.*, 2015). Furthermore, Lp (a) is also an independent risk factor of cardiovascular disease. It has been documented as an established risk factor for atherosclerosis, coronary artery disease, stroke, thrombosis and aortic stenosis (Farzam *et al.*, 2022). Thus, the higher level of Lp (a) in hypertensive patients will compound the risk of cardiovascular disease. In a large multicenter study, it was reported that elevated Lp (a) in patients with essential hypertension was associated with a significant

increase in cardiovascular disease (Rikhi *et al.*, 2023). As a result of these findings, the European Atherosclerosis Society (EAS) has recommended routine measurement of Lp (a) among patients with moderate to high risk of atherosclerosis and cardiovascular disease (EAS, 2010). The significantly higher level of uric acid in hypertensive patients found in this study imply a higher risk of cardiovascular disease in hypertensive patients. Several other studies have reported similar findings. In a cross-sectional study conducted on 270 essential hypertensive patients in southern Ethiopia, a prevalence of hyperuricemia of 27.4% was reported (Timerga *et al.*, 2021).

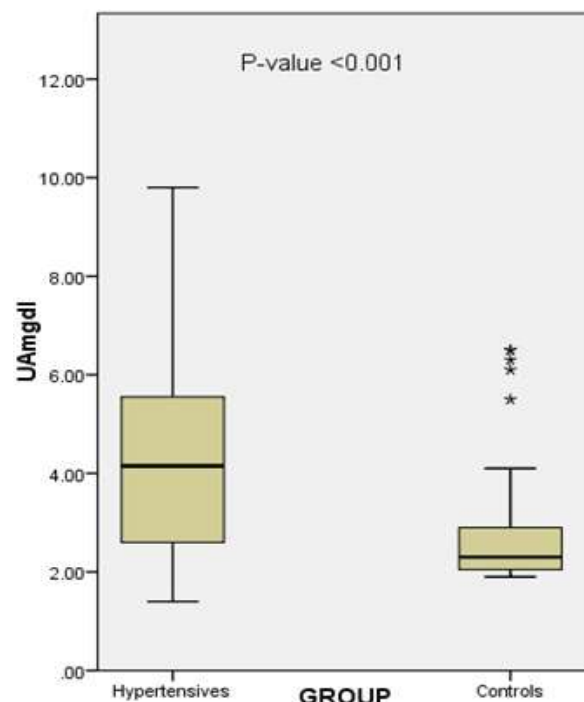


Fig 2: Boxplot showing the distribution of uric acid in the study population

Table 1: Sociodemographic characteristics of the study population

	Hypertensive n = 200	Controls n = 100	Statistical test	P-value
Age group				
<40	8 (4.0)	30 (30.0)		
40 – 59	97 (48.5)	63 (63.0)	$\chi^2 = 38.830$	<0.001
≥ 60	95 (47.5)	7 (7.0)		
Sex				
Male	53 (26.5)	47 (47.0)	$\chi^2 = 7.200$	0.007
Female	147 (73.5)	53 (53.0)		
BMI				
Underweight	3 (1.5)	2 (2.0)		
Normal	55 (27.5)	20 (20.0)	$\chi^2 = 0.675^*$	0.635
Overweight	70 (35.0)	43 (43.0)		
Obese	72 (36.0)	35 (35.0)		
Alcohol intake	33 (16.5)	7 (7.0)	Fishers Exact	0.067
Smoking	0 (0.0)	1 (1.0)	Fishers Exact	0.333

*Adjusted Chi square test. Abbreviation: BMI, body mass index

Table 2: Age, blood pressure measurements, anthropometrics and biochemical parameters of the study population.

	Hypertensives n= 200	Controls n = 100	t test	P-value
Age	58.2 ± 13.0 19.0 – 92.0	43.8 ± 10.7 22.0 – 66.0	7.410	<0.001
SBP	169.2 ± 15.6 140.0 – 220.0	118.3 ± 11.9 60.0 – 90.0	22.252	<0.001
DBP	106.5 ± 9.7 90.0 – 140.0	76.6 ± 7.4 60.0 – 90.0	20.948	<0.001
Weight	76.0 ± 18.0 44.0 – 135.0	76.2 ± 9.2 59.0 – 106.0	-0.067	0.946
Height	1.62 ± 0.07 1.50 – 1.80	1.64 ± 0.09 1.45 – 1.80	-1.265	0.208
BMI	28.9 ± 6.5 16.2 – 51.4	28.8 ± 5.1 18.4 – 41.4	0.137	0.891
Weight circumference	98.8 ± 13.2 70.0 – 149.0	91.3 ± 8.1 80.0 – 129.0	4.056	<0.001
Creatinine	1.2 ± 1.1 0.4 – 9.4	1.0 ± 0.2 0.3 – 1.3	1.802	0.073
eGFR	80.6 ± 36.9 5.0 – 277.0	91.8 ± 24.2 55.0 – 171.0	-2.126	0.035
Lp (a)	32.8 ± 16.6 5.2 – 89.0	16.9 ± 13.9 1.1 – 63.2	6.380	<0.001
Uric acid	4.1 ± 1.8 1.4 – 9.8	2.7 ± 1.2 1.9 – 6.5	4.082	<0.001

Abbreviation: SBP, systolic blood pressure; DBP, diastolic blood pressure; BMI, body mass index; eGFR, estimated glomerular filtration rate

Table 3. Correlation of Lp (a) levels with Uric acid in hypertensives and controls

	Hypertensives	Controls
R	0.238	0.006
Uric acid P-value	0.009	0.966

Dependent variable: Lp (a)

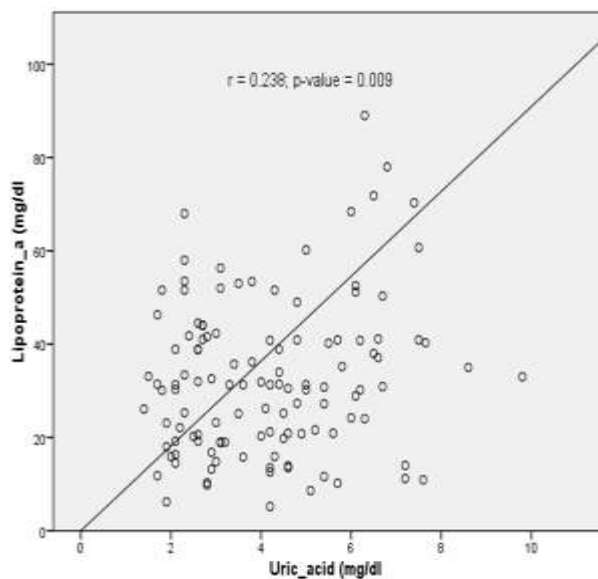


Fig 3: Scatterplot showing correlation between lipoprotein (a) and uric acid in hypertensives

The prevalence of hyperuricemia was 18.2% among hypertensive adult patients aged 50-79 years in southern China (Zhang *et al.*, 2022). Mehta *et al* reported a prevalence of hyperuricemia of 25% among 168 hypertensive patients in a hospital based cross-sectional study in Nepal (Mehta *et al.*, 2021). In a research undertaken to investigate the prevalence of

hyperuricemia in newly diagnosed essential hypertensive patients, a prevalence of 26% was reported (Mishra *et al.*, 2017). Studies done in other parts of Nigeria reported a higher prevalence of hyperuricemia in hypertensive patients. In a study in western Nigeria, the prevalence of hyperuricemia in hypertensive patients was 36.7% (Fasae *et al.*, 2018). In another study carried out in 130 newly diagnosed hypertensive patients in Nigeria, the prevalence of hyperuricemia was 46.9% (Ofori *et al.*, 2015). The difference in prevalence between this present study and other studies done in Nigeria may be due to several factors that influence serum uric acid levels including diet, drugs, alcohol, obesity, renal impairment, and hypertension.

The probable mechanism that links uric acid to hypertension include stimulating the growth of vascular muscle cells, eliciting vascular inflammation, impairing endothelial cell function, inducing insulin resistance and activating renin-angiotensin system (Sheng *et al.*, 2024).

In this study, Lp (a) was positively associated with uric acid in the hypertensive patients. We could not find similar studies on the association of Lp (a) with uric acid in hypertensive patients. However, since both Lp (a) and uric acid are both risk factors of cardiovascular disease, the following study suggests a synergistic effect of both analytes in causing cardiovascular events. Nishino *et al* reported that uric acid and Lp (a) are correlated with vasospastic angina (Nishino *et al.*, 2014). There are a few limitations. The study design is a cross-sectional study and cannot establish a causal

relationship between Lp (a) and uric acid with hypertension. Also, being a hospital-based study, it may not truly represent the population.

Conclusion: Lp (a) and uric acid levels in hypertensive patients were significantly higher than in the controls. These findings imply a higher risk of cardiovascular disease in hypertension. Also, in the hypertensive population, Lp (a) correlated positively with uric acid. Thus, routine screening of hypertensive patients for Lp (a) can be used to demonstrate the presence of cardiovascular risk. Further studies are needed to investigate the mechanisms linking Lp (a) with hypertension and cardiovascular disease.

Conflicts of Interest: The authors declare that they have no conflict of interest concerning this article.

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REFERENCES

- Adeloye, D; Basquill, C; Aderemi, AV; Thompson, JY; Obi, FA (2015). An estimate of the prevalence of hypertension in Nigeria: a systemic review and meta-analysis. *Hypertens.* 33(2):230-42.
- Atoe, K; Eguvbe, AO (2021). Assessment of serum uric acid levels in patients with cervical intraepithelial neoplasia in Benin City. *Annals of Clinical and Biomedical Research.* 2:99. doi:10.4081/acbr.2021.99
- Bhavani, BA; Padma, T; Sastry, BKS; Reddy, NK (2003). Plasma lipoprotein (a) levels in patients with untreated essential hypertension. *Indian J. Hum. Genet.* 9(2):65-68.
- Brosolo, G; Porto, AD; Bulfone, L; Vacca, A; Bortin, N; Colussi, G; Cavarape, A; Sechi, LA; Catena, C (2021). Plasma lipoprotein (a) levels as determinants of arterial stiffening in hypertension. *Biomedicines.* 9(11):1510.
- European Atherosclerosis Society (EAS), new recommendations from the European Atherosclerosis Society consensus panel resolve controversy about lipoprotein (a) and cardiovascular risk 2010. Available from: [http://www.diabetesincontrol.com/european-atherosclerosis-society-recommends-screening-for-lpa/\[Ref list\]](http://www.diabetesincontrol.com/european-atherosclerosis-society-recommends-screening-for-lpa/[Ref list]).
- Farzam, K; Senthikumar, S (2022). Lipoprotein A. [updated 2022 Sep 2]. In: StatPearls [Internet]. Treasure Island (FL): *StatPearls Publishing.*
- 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
- Grayson, PC; Kim, SY; Lavalley, M; Choi, HK (2011). Hyperuricaemia and incident hypertension: a systemic review and meta-analysis. *Arthritis Care Res.* 63:102-110.
- Jordan, J; Kurschat, C; Reuter, H (2018). Arterial hypertension. *Dtsch. Arztebl. Int.* 115(33-34):557-568.
- Kuwabara, M; Hisatome, I; Niwa, K; Hara, S; Roncal-Jimenez, CA; Bjornstad, P; Nakagawa, T; Andres-Hernando, A; Sato, Y; Jensen, T; Garcia, G; Rodriguez-Iturbe, B; Ohno, M; Lanasa, MA; Johnson, RJ (2018). Uric acid is a strong risk marker for developing hypertension from prehypertension: a 5-year Japanese cohort study. *Hypertension.* 71:78-86.
- 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.
- Mahto, SK; Sheoran, A; Gadpayle, AK; Gupta, K; Gupta, PK; Chitkarn, A; Agarwal, N (2022). Evaluation of lipoprotein (a) and lipid abnormalities in patients with newly detected hypertension and its association with severity of hypertension. *J. Family Med. Prim. Care.* 11(4):1508-13.
- Maranhao, RC; Carvalho, PO; Strunz, CC; Pileggi, F (2014). Lipoprotein (a): Structure, pathophysiology and clinical implications. *Arq. Bras. Cardiol.* 103(1):76-84.
- Mehta, RK; 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):404-406.
- Nagahama, K; Inoue, T; Iseki, K; Touma, T; Kinjo, K; Ohya, YL; Takishita, S (2004). Hyperuricemia as a predictor of hypertension in a screened cohort in Okinawa Japan. *Hypertens Res*. 27:11:835-841.
- Nishino, M; Mori, N; Yoshimura, T; Nakamura, D; Lee, Y; Taniike, M; Makino, N; Kato, H; Egami, Y; Shutta, R; Tanouchi, J; Yamada, Y (2014). Higher serum uric acid and lipoprotein (a) are correlated with coronary spasm. *Heart Vessels*. 29:186-190.
- Ofori, SN; Akpa, M (2015). Relationship between uric acid and left ventricular mass and geometry in Nigerian patients with untreated essential hypertension. *Nig. J. Cardiol*. 12(2):129-35.
- Oguanobi, NI (2021). Management of hypertension in Nigeria: The barriers and challenges. *J. Cardiol. Cardiovasc. Med*. 6, 23-25.
- Papadakis, JA; Ganotakis, ES; Jagroop, IA; Mikhailidis, DP; Winder, AF (1999). Effect of hypertension and its treatment on lipid, lipoprotein (a), fibrinogen, and bilirubin levels in patients referred for dyslipidemia. *Am. J. Hypertens*. 12(7):673-81.
- Remedious, C; Shali, M; Bhaster, AG; Lakdawala, M (2012). Hyperuricaemia: a reality in the Indian obese. *OBES. SURG*. 22:945-48.
- Rikhi, R; Bhatra, HS; Schaich, CL; Ashburn, N; Tsai, MY; Michos, ED; Chevli, PA; Herrington, DM; Tsimikas, S; Shapiro, MD (2023). Association of lipoprotein (a) and hypertension in primary prevention of cardiovascular disease: The MESA. *Hypertension*. 80:352-60.
- Sheng, S; Chen, J; Zhang, Y; Li, W; Li, F; Gou, R (2024). A cohort study on the correlation between serum uric acid trajectory and new onset hypertension based on Longitudinal Health Examination Data in Henan Area. *J. Inflamm. Res*. 17:1365-1375.
- 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. *Hyperten*. 45:28-33.
- Sur, A; Tirkey, BN; Mishra, PK (2015). Evaluation of role of serum lipoprotein and lipid profile in essential hypertension patients in a tertiary care hospital. *J. Hypertens*. 4:204. Doi: 10.4172/21667-1095.1000204
- Timerga, A; Haile, K (2021). Evaluation of uric acid disorders and associated factors in essential hypertensive patients at Wolkite University Specialized Hospital, Southern Ethiopia. *PLoS One*. 16(9):e0256557.
- Tsimikas, S; Karwadowska-Prokopezuk, E; Gouni-Berthold, L; Tardif, JC; Baum, SJ; Steinhagem-Thiessen, E; Shapiro, MD; Stroes, ES; Moriarty, PM; Nordestgaard, BG; Xia, S; Guerriero, J; Viney, NJ; O’Dea, L; Witztum JL (2020). Lipoprotein (a) reduction in persons with cardiovascular disease. *N. Engl. J. Med*. 382:244-55.
- Ward, NC; Nolde, JM; Chan, J; Carnagarin, R; Watts, GF; Schlaich, MP (2021). Lipoprotein (a) and hypertension. *Curr. Hypertens. Rep*. 23:44. Doi: 10.1007/s11906-021-01161-6
- Zhang, Y; Nie, F; Huang, X; Tang, W; Hu, R; Zhang, WQ; Liu, JX; Xu, RH; Liu, Y; Wei, D; Wang, TD; 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). Doi: 10.1186/s12872-021-02427-2