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Assessment of Albuminuria and Serum Levels of Lipoprotein (A) and Their Correlation in Hypertensive Patients in a Government Hospital, Warri, Delta State, Nigeria

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ABSTRACT: Hypertension is the most important risk factor for cardiovascular disease and the main cause of death worldwide. Some studies have reported a positive association between microalbuminuria and lipoprotein (a) with cardiovascular disease in hypertensive patients. Hence, the objective of this paper was to assess albuminuria and serum levels of Lipoprotein (a) (Lp [a]) and their correlation in hypertensive patients in a Government hospital in Warri, Delta State, Nigeria using appropriate standard techniques. Data collected showed that Thirty (15.0%) vs. 0 (0.0%) had microalbuminuria in the hypertensive and control groups respectively. The difference in the two groups was statistically significant (P-value =0.046). The mean urine albumin-creatinine ratio (UACR) were 1.02 ± 1.42 vs. 0.28 ± 0.16 mg/mmol in the hypertensive and control groups respectively. The difference in the two groups was statistically significant (<0.001). One hundred and fifteen (57.5%) vs. 18 (18.0%) had elevated Lp (a) in hypertensive and control groups respectively. The difference in the two groups was statistically significant (P-value <0.001). The mean Lp (a) were 32.77 ± 16.61 vs. 16.88 ± 13.85 mg/dl in the hypertensive and control groups respectively. The difference in the two groups was statistically significant (<0.001). There was a significant weak negative correlation between UACR and Lp (a) in the hypertensive group (Pearson's Correlation = -0.214, P-value = 0.019). The serum levels of Lp (a) and UACR were significantly higher in the hypertensives than in the controls. There was a significant weak negative correlation between UACR and Lp (a) in the hypertensive group. Thus, routine screening for serum Lp (a) will enhance the assessment of hypertensive patients for risk of cardiovascular disease.

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Hypertension is the most important risk factor for cardiovascular disease and the main cause of death worldwide (Okubadejo *et al.*, 2019, Buelt *et al*., 2021). Some studies have reported a positive association between microalbuminuria and lipoprotein (a) (Lp [a]) with cardiovascular disease in hypertensive patients. The presence of microalbuminuria in hypertensive patients has been seen as a sensitive marker of kidney

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damage and a risk factor for cardiovascular disease and end stage renal disease (Poundel *et al*., 2012). Microalbuminuria was previously defined as the presence of very little quantities of albumin in the urine that could not be detected by standard ‘’dip stick’’ methods (Eguvbe *et al*., 2019). Presently, it is defined as “urinary albumin excretion between 30 and 300 mg/day, if measured in a 24 hour urine collection, 20–200 ug/min, if measured in a timed urine collection or 30–300 mg/g, if measured with the use of urinary albumin to creatinine ratio (UACR) in a spot urine collection” (Teimoury *et al*., 2014). Concentrations of urine albumin can be lowered and worsening of proteinuria prevented by aggressive control of blood pressure especially with drugs that act against the renin-angiotensin-aldosterone system, and treatment of diabetes (Chugh *et al*., 2007). High serum concentration of Lp (a) has been identified as an independent risk factor of cardiovascular disease (Brosolo *et al*., 2021). It has been suggested to contribute to an increased risk for atherosclerosis in hypertensive patients (Tangvarasittichai *et al*., 2016). Lp (a) has a structure that is similar to low density lipoprotein. Apolipoprotein (a) is covalently bound to apolipoprotein B by a disulfide bridge in a 1:1 molar ratio. Lp (a) enhances the formation of foam cell and deposition of cholesterol in atherosclerotic plaques by binding to macrophages (Saeedi *et al*., 2016). Significantly elevated Lp (a) has been reported in hypertensive patients and it increases the risk of cardiovascular disease by mechanisms that involve the pro-atherogenic effects of its LDL-like portion, pro-inflammatory effects of its phospholipid content, and pro-thrombotic effects through its plasminogen-like protease domain on apolipoprotein (a) (Eguvbe *et al*., 2024, Tsimikas *et al*., 2020). Studies correlating UACR with Lp (a) in hypertensive patients have not been done in this environment. Hence, the objective of this paper was to assess albuminuria and Serum Levels of Lipoprotein (A) and their Correlation in Hypertensive Patients in a Government Hospital in Warri, Delta State, Nigeria

MATERIALS AND METHODS

Study design and population: It was a cross-sectional study among three hundred participants that consisted of two hundred hypertensive and one hundred normotensive individuals, conducted in the Delta State Central Hospital, Warri, between October 2022 and December 2023. The hypertensive and normotensive cases were patients seen at the General outpatient Department and the Cardiology Unit of the Delta State Central Hospital, Warri. The inclusion criteria were participants who were aged eighteen years and above, diagnosed with hypertension (blood pressure ≥ 140/90 mmHg) and normotensive individuals with blood pressure below 140/90 mmHg. Participants with the following medical history were excluded from the study: diabetes mellitus, chronic alcoholism or smoking, renal disease, liver disease, pregnancy, menstruating or breast feeding women, febrile disease, urinary tract infection, vigorous physical activity in the previous 24 hours, hypothyroidism/hyperthyroidism, hypertriglyceridemia (> 400 mg/dl), hereditary familiar hypercholesterolemia/dyslipoproteinemia, women on hormone replacement therapy, Patients on phenytoin, carbamazepine, metformin, pentoxyphylline, methotrexate, vitamin D supplements and lipid lowering agents. Central Hospital Warri is one of the biggest secondary health facilities in Delta State with 254 beds. They provide general and specialized medical and surgical services to the inhabitants of Warri and most parts of Delta State.

Sample size and sampling procedure: The formula for a cross-sectional study is provided in equation 1.

$$n = (Z^2Pq)/d^2 \qquad (1)$$

Where n = sample size, Z = standard deviation, P = prevalence of hypertension, q = 1-P and d = degree of precision to be used (0.05), was used to estimate the sample size. For the recruitment of participants, consecutive sampling was utilized. Three hundred participants made up of two hundred hypertensive and one hundred normotensive individuals were involved in the study. Written informed consent was obtained from each study participant. The study used a structured questionnaire that was administered by interviews. The questionnaire included identification number, age, gender, weight, height, waist circumference, medical history and laboratory results.

Data collection: Demographic and baseline characteristics: The study used a structured questionnaire that was administered by interviews. The questionnaire included identification number, age, gender, weight, height, waist circumference, medical history and laboratory results. A medical history of past or current co-morbidities was taken. BP measurement was done with a mercury sphygmomanometer after allowing the patient to rest for at least ten minutes. The first and second readings which represented the systolic and diastolic BP respectively, were taken at the first appearance of the korotkoff sound (phase I) and that at its disappearance (phase V). Hypertension was defined as BP measurement equal to 140/90 mmHg and above on two or more different occasions (Jordan et al., 2018). Height measurement was done with a stadiometer.

Weight measurement was done with an electronic patient weighing scale. Body mass index (BMI) was derived from the weight in kilogram divided by the height in meter squared. Obesity was defined as BMI that is 30 kg/m^2 or greater. Waist circumference (WC) measurement was done 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: Five milliliters (5 ml) of venous blood was obtained from the brachial vein of each participant into a plain bottle and allowed to clot. The samples were separated and serum kept at -20℃ and analysed weekly. Temperature of the freezer was monitored by temperature recordings in the morning and evening. Serum level of Lp (a) was analysed by immunoturbidimetry according to the manufacturer's protocol, while serum creatinine was assayed on a spectrophotometer using the kinetic modification of the Jaffe procedure. Estimated glomerular filtration rate (eGFR) was derived from serum creatinine with the aid of the Modification of Diet for Renal Disease formula based on age, sex, race and serum creatinine (Levey *et al.,* 2000).

Urine sample: About 10 milliliters of random urine was provided by each study participant in plain bottles and stored at 2 to 8 ℃ until analysis for urine albumin and creatinine. Urine albumin was assayed on a spectrophotometer using the immunoturbidimetric assay. Urine albumin creatinine ratio was derived by dividing urine albumin concentration in milligrams by the urine creatinine concentration in grams.

Statistical analysis: SPSS version 23 was used for data analysis. Test for normality was done for continuous variables including age, blood pressure measurements and anthropometric variables, and biochemical parameters. Normally distributed continuous variables were presented as mean, standard deviation, and ranges.

Categorical variables were presented as frequencies and percentages. Student T test was used to compare the differences in means of continuous variables between the hypertensive group and controls. Chi square test was used for univariate analysis. The Pearson's correlation coefficient was used to correlate UACR with Lipoprotein (a) levels in hypertensives and controls. Statistical significance was set at <0.05.

Ethical Clearance: The ethics and research committee of the Delta State Central Hospital, Warri, gave approval for this study. Written informed consent was gotten from each study participant and participant's strict confidentiality was maintained with patient's data.

RESULTS AND DIISCUSSION

The study recruited 200 hypertensive and 100 controls. The Demographic and Social Parameters of participants are shown in Table 1. The mean age and standard deviation were 58.23 ± 13.07 years and 43.78 ± 10.68 years in the hypertensive and control groups respectively. The difference was statistically significant (P-value <0.001). In the hypertensive arm, the modal age, median age and range were 67 years, 58 years and 73 years respectively. These varied in the control group with the modal age, median age and range being 42 years, 45 years and 44 years respectively. The majority of the participants, who were in their middle ages, where 53 (26.5%) and 38 (38.0%) and were within 50-59 years and 40-49 years in the hypertensive and control groups respectively. In the hypertensive group, 53 (26.5%) were also within 60-69 years. This was followed by 43 (21.5%) that were within 40-49 years, 42 (21.0%) were ≥70 years while only 4 (2.0%) were < 30 years. On the other hand, 25 (25%) were within the age of 50-59 years, followed by 18 (18.0%) who were within 30-39 years. None were ≥70 years while only 12 (12.0%) were < 30 years. The differences in age distribution were statistically significant (P-value <0.001). The sex distribution in both groups also varied significantly (P-value = 0.007). In the hypertensive group, 53 (26.5%) were male while 147 (73.5%) were female. In the control group, 53 (53.0%) were male while 47 (47.0%) were female. The mean weights of study participants were 76.03 ± 17.98 vs. 76.20 ± 9.22 kg. The mean heights were 1.62 ± 0.07 vs. 1.64 ± 0.08 m, while the mean BMI were 28.88 ± 6.47 vs. 28.56 ± 4.67 kg/m^2 in the hypertensive and control respectively. There were no statistically significant differences in the weight, height and BMI in the two groups (P-values >0.05). Most of the participants were overweight and obese in both groups. Alcohol intake was reported in 33 (16.5%) participants in the hypertensive group while it was 7 (7.0%) in the control arm. None of the hypertensive group indulged in smoking but only 1 (1.0%) smokes cigarette. There were no statistically significant differences in alcohol and smoking habits in both groups (P-values 0.063 and 0.246 respectively). The mean systolic and diastolic blood pressures in both groups are illustrated in Figure 1 below. The mean systolic blood pressures are 169.18 ± 15.58 vs. 118.26 ± 11.90 mmHg, while the diastolic blood pressures are 106.53 ± 9.71 vs. 76.20 ± 9.23 mmHg in the hypertensive and control groups respectively. The differences in both groups' blood pressures were statistically significant (P-values< 0.001).

Table 1: Demographic and Social Parameters

Variable	Hypertensive N=200 n (%)	Control N=100 n (%)	P – values
Age			<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	<0.001*
Modal age	67 years	42 years	
Median age	58yrs	45yrs	
Range	92-19= 73yrs	66-22= 44yrs	
Sex			0.007*
Male	53 (26.5)	53 (53.0)	
Female	147 (73.5)	47 (47.0)	
Weight (kg) (Mean ± SD)	76.03 ± 17.98	76.20 ± 9.22	0.946
Height (m) (Mean ± SD)	1.62 ± 0.07	1.64 ± 0.08	0.208
BMI (Kg/m^2)			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	0.736
Waist circumference (cm) (Mean ± SD)	98.78 ± 13.17	91.25 ± 8.14	<0.001*
Alcohol	33 (16.5)	7 (7.0)	0.063
Smoking	0 (0.0)	1 (1.0)	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)

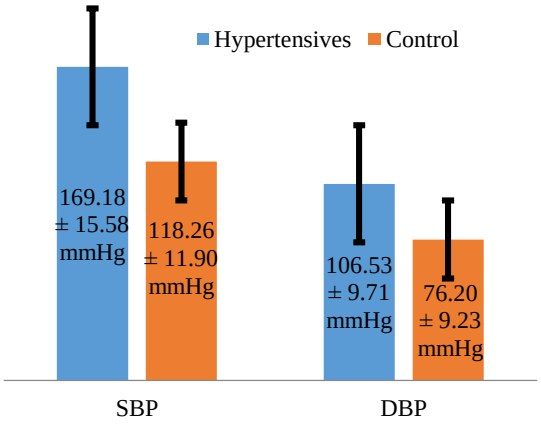


Fig 1: Systolic and diastolic Blood pressure between the two groups
SBP= Systolic blood pressure, DBP = Diastolic blood pressure; SBP (Student T-test= 495.139, P< 0.001), DBP (Student T-test= 438.814, P< 0.001)

The Biochemical variables of study participants are shown in Table 2 below. The mean serum creatitinine were 106.1 ± 94.6 vs. 84.9 ± 15.0 µmol/l in the hypertensive and control group respectively. The lowest value in the hypertensive group was 35.4 while the highest was 831.0, versus 53.0 as lowest and 114.9 as highest in the control group. There were no statistically significant differences in the two groups (0.095). The mean eGFR were 0.74 ± 0.27 vs. 0.87 ± 0.20 mL/s/m^2 in the hypertensive and control groups respectively. The lowest value in the hypertensive group was 0.05 while the highest was 1.23, versus 0.53 as lowest and 1.20 as highest in the control group. The differences in the two groups were statistically significant (0.001). The mean UACR were 1.02 ± 1.42 vs. 0.28 ± 0.16 mg/mmol in the hypertensive and control group respectively. The lowest value in the hypertensive group was 0.02 while the highest was

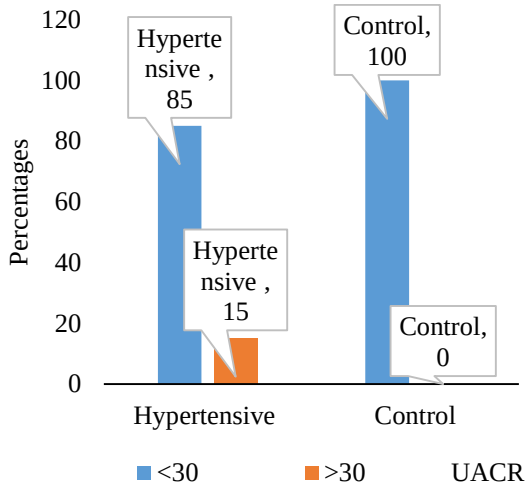
4.99, versus 0.01 as lowest and 0.76 as highest in the control group. The differences in the two groups were statistically significant (<0.001). The mean Lp (a) were 32.77 ± 16.61 vs. 16.88 ± 13.85 mg/dl in the hypertensive and control group respectively. The lowest value in the hypertensive group was 5.20 while the highest was 89.00, versus 1.10 as lowest and 63.20 as highest in the control group. The differences in the two groups were statistically significant (<0.001).

Table 2: Biochemical variables [Range; (Mean ± SD)] of study participants

Variable	Hypertensive N=200 n (%)	Control N=100 n (%)	P –values
Serum Creatinine (µmol/l)	35.4-831.0 (106.1 ± 94.6)	53.0-114.9 (84.9 ± 15.0)	0.095
eGFR (mL/s/m^2)	0.05-1.23 (0.74 ± 0.27)	0.53-1.20 (0.87 ± 0.20)	0.001*
UACR (mg/mmol)	0.02-4.99 (1.02 ± 1.42)	0.01-0.76 (0.28 ± 0.16)	<0.001
Lp (a) **(mg/dl)**	5.20-89.00 (32.77 ± 16.61)	1.10-63.20(16.88 ± 13.85)	<0.001*

*eGFR= Glomerular filtration rate, ACR =Albumin-creatinine ratio, LP(a)= Lipoprotein (a), t= Student's T-test, SD= standard deviation, *=statistically significant (p<0.05)*

The Proportion of hypertensive and controls with elevated UACR is shown in Figure 2 below. It was observed that 170 (85.0%) vs. 100 (100.0%) had normal UACR in hypertensive and control groups respectively. Thirty (15.0%) of the hypertensives had elevated UACR (≥30) compared to none (0%) of the controls. There was a statistically significant difference in the two groups (P-value =0.046).



X^2= 4.000, P= 0.046, UACR= Urine Albumin-creatinine ratio

Fig 2: Proportion of hypertensive and controls with elevated UACR

The Proportion of hypertensive and controls with normal and elevated Lp (a) is shown in Figure 3 below. It was observed that 85 (42.5%) vs. 82 (82.0%) had normal Lp (a) in hypertensive and control groups respectively, while 115 (57.5%) of the hypertensives had elevated Lp (a) compared to 18 (18.0%) of the controls. There was a statistically significant difference in the two groups (P-value <0.001). The Table 3 below shows findings of Correlation between UACR with lipoprotein (a) in hypertensive patients and controls. There was a significant weak negative correlation between UACR and Lp (a) in the hypertensive group (Pearson's Correlation = -0.214, P-value = 0.019). In the control group, there was no significant correlation between UACR and Lp (a) (Pearson's Correlation = -0.005, P-value = 0.971). The association between UACR with lipoprotein (a) in hypertensive patients and controls is shown in Table 4

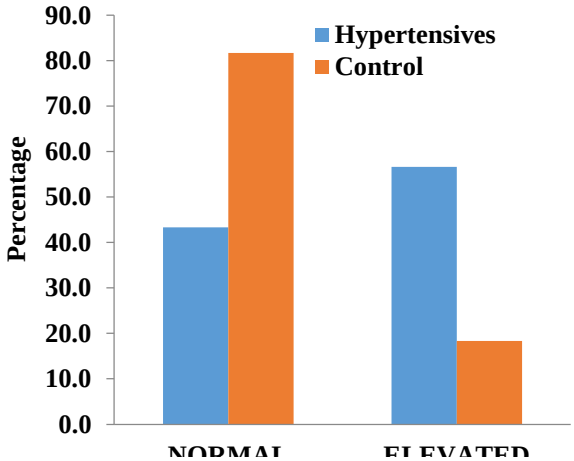


Fig 3: Proportion of hypertensives and controls with normal and elevated Lipoprotein (a) X^2= 23.868, P< 0.001
Elevated lipoprotein (a) corresponds to > 30 mg/dl

Table 3: Correlation between ACR with lipoprotein (a) in hypertensive patients and controls.

		ACR	Lp (a)
Hypertensive group			
UACR	Pearson Correlation	1	- 0.214
	Sig. (2-tailed)		0.019*
	N	200	200
Lp (a)	Pearson Correlation	- 0.214	1
	Sig. (2-tailed)	0.019*	
	N	200	200
Control group			
UACR	Pearson Correlation	1	-0.005
	Sig. (2-tailed)		0.971
	N	100	100
Lp (a)	Pearson Correlation	-0.005	1
	Sig. (2-tailed)	0.971	
	N	100	100

UACR = Urine Albumin-creatinine ratio, Correlation is significant at < 0.05 (*)

In the hypertensive group, 70 (41.2%) with normal UACR had normal Lp (a) while a higher proportion of

100 (58.8%) had elevated Lp (a). In hypertensive with elevated UACR, 17 (56.7%) had normal Lp (a) while a lower proportion of 13 (43.3%) had elevated Lp (a). These findings were not statistically significant (P-values =0.256). On the other hand, in the control group, 82 (82.0%) with normal UACR had normal Lp (a) while 18 (18.0) had elevated Lp (a). None of the controls had elevated UACR. This present study was undertaken to evaluate the levels of albuminuria and serum Lp (a) and their correlation in hypertensive patients. We found that serum levels of Lp (a) and UACR were significantly higher in the hypertensives than in the controls, implying that both can serve as biomarkers of cardiovascular risk. Also, the prevalence of microalbuminuria and elevated Lp (a) in the hypertensive patients were 15% and 57.5% respectively. There was a significant weak negative correlation between UACR and Lp (a) in the hypertensive group.

Table 4: Association between ACR with lipoprotein (a) in hypertensive patients and controls.

Variable	LP (a)		Total	Statistics	P - values
	Normal n (%)	Elevated n (%)			
ACR					
Hypertensive (N= 200) (ACR)					
Normal	70 (41.2)	100 (58.8)	170 (100.00)	1.288[c]	0.256
Elevated	17 (56.7)	13 (43.3)	30 (100.0)		
Control (N= 100)					
ACR					
Normal	82 (82.0)	18 (18.0)	100 (100.00)	-	-
Elevated	0(0.0)	0 (0.0)	0 (0.0)		

ACR = Albumin creatinine ratio, Lp (a) = Lipoprotein (a), c= Chi-square, P< 0.05 is statistically significant

The mechanisms linking UACR with hypertension are largely unknown (Ren *et al*., 2021). However, the suggested mechanisms linking microalbuminuria to cardiovascular disease include endothelial dysfunction, chronic low-grade inflammation, or increased transvascular leakage of macromolecules (Stehouwer *et al.,* 2006). Increase in UACR within the reference limits has been associated with a higher risk of hypertension in the general population (Ren *et al*., 2021). As seen in this study, significant microalbuminuria in hypertensive patients has been reported in several other studies. Lower prevalence of microalbuminuria in hypertensive patients of 6.6%, 7.1%, 10.1% and 11% were reported in some studies while others documented higher prevalence of 44% and 62.5% (Kim et al., 2013, Ardeleanu *et al.,* 2015, Choi *et al*., 2006, Visaria *et al*., 2021, Maggon *et al*., 2018, Alvaro *et al*., 2005). Studies done in other parts of Nigeria by Ogbu *et al* and Odili *et al*, recorded prevalence of 22% and 41% respectively (Ogbu *et al*., 2013, Odili *et al*., 2008). The differences in the prevalence of microalbuminuria among the different studies may be due to differences in the sample size, severity of hypertension, average age of the study population, racial/ethnic differences, associated comorbid diseases such as diabetes, renal insufficiency, and study methodologies (Kim *et al*., 2013).

Elevated Lp (a) in hypertensive patients, as reported in this present study and in several other studies, is an indicator of the presence and severity of hypertensive vascular damage (Brosolo *et al*., 2021). Lp (a) has been suggested to promote the pathogenesis and progression of a number of cardiovascular diseases independent of other cardiovascular risk factors, due to its pro-inflammatory, pro-atherosclerotic and pro-thrombotic effects (Di Fusco *et al.,* 2023). Bhavani *et al*. (2003) in a study to investigate plasma Lp (a) in hypertensive patients, documented a positive link between elevated Lp (a) concentrations and hypertension. They also reported that elevated plasma concentrations of Lp (a) that is within the reference reference interval, could be an independent risk factor for atherosclerosis (Bhavani *et al*., 2003).

This present study also reported a significant weak negative correlation between UACR and Lp (a) in the hypertensive group. We could not find any study investigating the relationship of UACR with serum Lp (a) levels in hypertensive patients. However, several studies have reported microalbuminuria and serum Lp (a) as independent risk factors of cardiovascular disease. Microalbuminuria has been shown to be an independent risk factor for coronary heart disease and cardiovascular disease in the population (Xia *et al*., 2015). Albuminuria has been linked to a higher risk of coronary artery disease, stroke, heart failure, arrhythmias, and microvascular disease (Barzilay *et al*., 2024). Similarly, in addition to being an independent risk factor of cardiovascular disease, elevated Lp (a) levels also predict cardiovascular events (Le Bras *et al.,* 2018). Elevated Lp (a) has been associated with a higher risk of ischaemic cardiovascular disease, aortic valve stenosis and heart failure (Vinci *et al*., 2023).

The limitation of this present study include the single measurement of UACR because the rate of albumin loss is influenced by a number of physiological factors such as exercise, posture and time of day. Hence, repeated measurement is more reliable. Also, it may not truly represent the general population because it is a hospital-based study.

*Conclusion***:** The serum levels of Lp (a) and UACR were significantly higher in the hypertensives than in the controls, implying that both can serve as biomarkers of cardiovascular risk. There was a significant weak negative correlation between UACR and Lp (a) in the hypertensive group. Further research with a larger sample size will provide more information on the relationship between UACR and Lp (a) in hypertensive patients. However, routine screening for serum Lp (a) will enhance the assessment of hypertensive patients for risk of cardiovascular disease.

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

Data Availability Statement: Data are available upon request from the first author.

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