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Serum Lipoprotein(a) and Lipid Profile as Predictors of Cardiovascular Risk in Non-Dialysis-Dependent Chronic Kidney Disease: A Case-Control Study

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ABSTRACT

Background: Chronic kidney disease (CKD) is associated with a markedly elevated risk of cardiovascular morbidity and mortality. Dysregulated lipoprotein metabolism, particularly elevated serum lipoprotein(a) [Lp(a)], may be a critical mediator of this risk.

Objectives: To compare serum Lp(a) levels and lipid profile between non-dialysis-dependent CKD patients and healthy controls, and to assess correlations between Lp(a) and other biochemical markers.

Methods: 40 non-dialysis-dependent CKD patients (CKD stages 1–4) attending Gauhati Medical College & Hospital and 40 age- and sex-matched healthy controls were enrolled for the study. Fasting serum Lp(a) was measured by sandwich ELISA; lipid profile, renal function tests, liver enzymes, and fasting plasma glucose were measured enzymatically. Unpaired Student's t-test and Pearson's correlation coefficient were used for statistical analysis.

Results: Serum Lp(a) was significantly higher in CKD cases (47.54 ± 10.40 mg/dL) compared to controls (18.56 ± 5.54 mg/dL; $p<0.0001$). Total cholesterol, triglycerides, LDL, and VLDL were all significantly elevated in CKD patients ($p<0.05$). Lp(a) showed significant positive correlations with serum urea ($r=0.51$), creatinine ($r=0.45$), total cholesterol ($r=0.40$), triglycerides ($r=0.77$), VLDL ($r=0.77$), and LDL ($r=0.45$); and a significant negative correlation with HDL ($r=-0.43$).

Conclusion: Serum Lp(a) is markedly elevated in non-dialysis-dependent CKD patients and correlates significantly with markers of renal dysfunction and atherogenic dyslipidemia. Routine estimation of Lp(a) may facilitate early identification and prevention of cardiovascular complications in CKD patients.

Keywords: *Chronic kidney disease; Lipoprotein(a); Lipid profile; Cardiovascular risk; Dyslipidemia; Atherosclerosis*

INTRODUCTION

Chronic kidney disease (CKD) is characterized by persistent abnormalities in kidney structure or function lasting for at

least three months, including evidence of kidney damage or a reduced glomerular filtration rate (GFR) of <60 mL/min/1.73 m². According to the National Kidney Foundation Kidney Disease Outcomes Quality Initiative (K/DOQI) guidelines, CKD is classified into five stages based on the severity of renal impairment, ranging from Stage 1, which represents the mildest form of disease with preserved kidney function, to Stage 5, which denotes kidney failure with a GFR of <15 mL/min/1.73 m².[1] In India, approximately 17 in every 100 individuals are affected by CKD, making it a major public health burden.[2]

Cardiovascular disease (CVD) is the leading cause of death in CKD patients, occurring at a rate seven to ten times higher than in age- and sex-matched individuals without kidney disease.[3] By the time patients require renal replacement therapy, their risk of cardiovascular death or non-fatal myocardial infarction is approximately 17 times that of the general population.[4] This striking excess risk is only partly explained by traditional risk factors, underscoring the importance of novel, CKD-specific risk markers.

Lipoprotein(a) [Lp(a)] is a unique lipoprotein consisting of an LDL-like particle containing apolipoprotein B-100 (apoB-100) covalently linked via a disulfide bond to apolipoprotein(a) [apo(a)]. [5] Apo(a) shares strong structural homology with plasminogen but lacks protease activity, enabling Lp(a) to competitively inhibit fibrinolysis and promote a prothrombotic, pro-atherogenic state. [6] Evidence consistently shows that Lp(a) rises early in renal failure [7] and increases with declining GFR, making it a potentially valuable early cardiovascular risk marker in CKD. [8]

Dysregulation of lipid metabolism in CKD is well-established and typically manifests as hypertriglyceridemia, elevated VLDL, reduced HDL-cholesterol, and variable changes in total and LDL-cholesterol. [9] However, the precise interrelationship between Lp(a) and these lipid parameters in non-dialysis-dependent CKD patients, particularly in the Indian population, remains incompletely characterized. The present study was therefore undertaken to compare serum Lp(a) and complete lipid profile between non-dialysis-dependent CKD patients and healthy controls, and to determine whether Lp(a) correlates with conventional lipid parameters and markers of renal dysfunction.

Aims and Objectives

The study aimed to: (1) measure serum Lp(a) levels in non-dialysis-dependent CKD patients and healthy controls; (2) assess the complete fasting lipid profile including total cholesterol, HDL, LDL, VLDL, and triglycerides in both groups; (3) evaluate correlations between serum Lp(a) and lipid parameters; (4) examine correlations between Lp(a) and markers of CKD severity (serum urea and creatinine); and (5) generate evidence to support introduction of routine Lp(a) estimation in CKD for early cardiovascular risk stratification.

MATERIALS & METHODS

Study Design and Setting

This hospital-based cross-sectional case-control study was conducted in the Department of Biochemistry in collaboration with the Department of Nephrology at Gauhati Medical College & Hospital (GMCH), Guwahati, Assam, India. The study protocol was reviewed and approved by the Institutional Ethics Committee of Gauhati Medical College. Written informed consent was obtained from all participants prior to their enrolment in the study.

Study Population

The case group comprised 40 patients with a confirmed diagnosis of non-dialysis-dependent CKD (Stages 1–4) admitted to the Department of Nephrology at GMCH. The control group comprised 40 apparently healthy, age- and sex-matched volunteers from the general community. Controls were recruited regardless of socioeconomic status

and had no history of renal, hepatic, cardiovascular, or metabolic disease.

Inclusion and Exclusion Criteria

Cases were included if they were aged >18 years, of either sex, with clinically diagnosed CKD (Stages 1–4) due to chronic glomerulonephritis, hypertension, nephrotic syndrome, or tubulointerstitial disease, and had not yet commenced dialysis. Patients were excluded if they had diabetes mellitus, cerebral stroke, recent myocardial infarction, hepatobiliary disorder, history of allergy, ischemic heart disease, smoking, or were on lipid-lowering medications. These criteria were applied to minimize confounding of Lp(a) and lipid measurements.

Clinical and Anthropometric Assessment

A standardized proforma was used to document demographic details, medical history, and physical examination findings. Body mass index (BMI) was calculated. Blood pressure was measured per American Heart Association guidelines using a mercury sphygmomanometer after five minutes of seated rest, and hypertension was defined as systolic BP ≥140 mmHg or diastolic BP ≥90 mmHg.

Sample Collection and Processing

A 5 mL fasting venous blood sample was collected. Plasma for glucose estimation was collected in sodium fluoride (NaF) vials. For all other estimations, blood was collected in clot vials. Separated serum samples were either processed immediately or stored at −80°C for a maximum of 6 months until analysis.

Laboratory Methods

Fasting plasma glucose was estimated by the GOD-POD enzymatic method at 505 nm. Serum urea was estimated by the Modified Berthelot (urease-GLDH) method at 570 nm. Serum creatinine was measured using the Modified Jaffé kinetic alkaline picrate method at 520 nm. Total cholesterol and triglycerides were measured by the CHOD-PAP and GPO-PAP enzymatic colorimetric methods, respectively, at 505 nm. HDL-cholesterol was quantified after phosphotungstate-magnesium chloride precipitation using the enzymatic CHOD-PAP method at 500 nm. LDL- and VLDL-cholesterol were calculated using the Friedewald formula: LDL = Total cholesterol − (Triglyceride/5) − HDL. All these analyses were performed on a MERCK Microlab 300 semi-autoanalyzer.

Serum Lp(a) was quantified using the Elabscience Human Lp(a) ELISA kit (sandwich ELISA format) on a BIO-RAD 680 ELISA microplate reader (version 1.7) at 450 nm. Briefly, standards and samples were incubated with anti-Lp(a) antibody-coated wells, followed by biotinylated detection antibody and streptavidin-HRP conjugate. After addition of TMB substrate, the reaction was stopped with H_2SO_4 and optical density was read. Lp(a) concentrations were derived from a standard curve constructed using Curve Expert 1.3 and expressed in mg/dL (conversion: 1 mg/dL = 10,000 ng/mL × dilution factor). Serum AST and ALT were measured by Reitman and Frankel's colorimetric method to exclude hepatic dysfunction.

Statistical Analysis

All data are expressed as mean ± standard deviation (SD). Differences between groups were assessed using the unpaired Student's t-test. Correlations between Lp(a) and other continuous variables within the case group were assessed using Pearson's correlation coefficient (r). A two-tailed p-value <0.05 was considered statistically significant. All statistical analyses were performed using GraphPad InStat (GraphPad Software, San Diego, CA, USA).

RESULTS

A total of 80 participants were enrolled: 40 non-dialysis-dependent CKD patients and 40 healthy controls. The mean age of the case group was 51.55 ± 8.81 years (range 31–70 years), compared to 51.07 ± 9.11 years in controls (p>0.05). The two groups

were also well-matched for sex, with 24 males (60%) and 16 females (40%) in each group. The majority of participants in both groups fell in the 51–60-year age bracket (45% each). Mean BMI was comparable between cases (24.31 ± 3.69 kg/m²) and controls (23.62 ± 1.45 kg/m²; p=0.2754), confirming adequacy of matching.

Table 1. Demographic and Anthropometric Characteristics of Study Groups

Parameter	Controls (n=40)	Cases (n=40)	p-value
Age (years), mean ± SD	51.07 ± 9.11	51.55 ± 8.81	>0.05
Male / Female, n (%)	24(60%) / 16(40%)	24(60%) / 16(40%)	1.000
BMI (kg/m²), mean ± SD	23.62 ± 1.45	24.31 ± 3.69	0.2754
Systolic BP (mmHg), mean ± SD	115.12 ± 7.73	147.00 ± 18.00	<0.0001
Diastolic BP (mmHg), mean ± SD	76.05 ± 4.90	88.17 ± 7.85	<0.0001

Blood Pressure

As shown in Table1, both systolic and diastolic blood pressure were significantly higher in the case group than controls (systolic: 147.00 ± 18.00 vs. 115.12 ± 7.73 mmHg; diastolic: 88.17 ± 7.85 vs. 76.05 ± 4.90 mmHg; both p<0.0001). This reflects the well-known high prevalence of hypertension in CKD and confirms that patients had clinically significant renal disease.

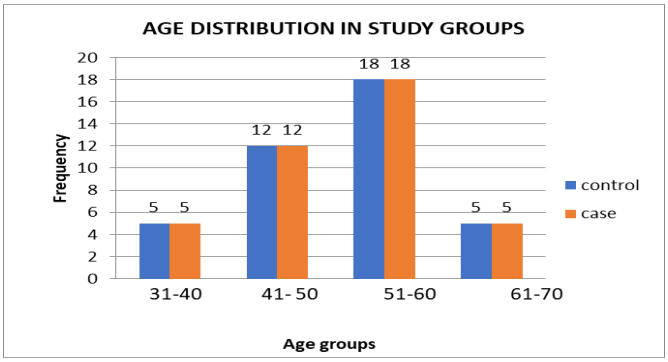

Figure 1: Showing frequency distribution of controls and cases in different age groups

Subjects in the two groups were further divided into four different age groups with age ranging from 31 to 70 as depicted in Figure 1. The maximum number of subjects in each of the two groups was in the age class of 51-60 years, with a relative frequency of 0.45 in that age group.

Table 2. Comparison of Biochemical Parameters between Controls and CKD Cases

Parameter	Controls (mean±SD)	Cases (mean±SD)	p-value
FPG (mg/dL)	88.00 ± 11.49	89.47 ± 15.10	0.3628
Serum Urea (mg/dL)	31.86 ± 8.31	56.28 ± 13.57	<0.0001
Serum Creatinine (mg/dL)	0.81 ± 0.21	2.77 ± 1.00	<0.0001
Total Cholesterol (mg/dL)	140.30 ± 31.97	162.32 ± 13.11	0.0031
Triglycerides (mg/dL)	100.52 ± 23.89	149.82 ± 13.13	<0.0001
HDL-C (mg/dL)	46.52 ± 9.68	44.47 ± 7.36	0.1878
LDL-C (mg/dL)	69.97 ± 30.89	87.80 ± 14.37	0.0307
VLDL-C (mg/dL)	20.11 ± 4.73	30.02 ± 2.64	<0.0001

Serum Lp(a) (mg/dL)	18.56 ± 5.54	47.54 ± 10.40	<0.0001
AST (U/mL)	22.97 ± 7.38	21.72 ± 5.73	0.4375
ALT (U/mL)	25.60 ± 6.46	24.92 ± 5.25	0.5183

FPG = Fasting Plasma Glucose; HDL-C = HDL-Cholesterol; LDL-C = LDL-Cholesterol; VLDL-C = VLDL-Cholesterol; Lp(a) = Lipoprotein(a); AST = Aspartate Aminotransferase; ALT = Alanine Aminotransferase. Values expressed as mean ± SD; unpaired Student's t-test used.

Renal Function, Blood glucose and Liver Enzymes

As depicted in Table 2, Serum urea and creatinine were markedly elevated in cases compared to controls (urea: 56.28 ± 13.57 vs. 31.86 ± 8.31 mg/dL; creatinine: 2.77 ± 1.00 vs. 0.81 ± 0.21 mg/dL; both p<0.0001), confirming impaired renal function in the case group. Fasting plasma glucose was within normal limits in both groups and did not differ significantly (cases: 89.47 ± 15.10 vs. controls: 88.00 ± 11.49 mg/dL; p=0.3628), confirming effective exclusion of diabetes. Serum AST and ALT did not differ significantly between groups (p>0.05), excluding hepatic dysfunction as a confound.

Serum Lipoprotein(a)

Serum Lp(a) was significantly elevated in the CKD case group (47.54 ± 10.40 mg/dL) compared to controls (18.56 ± 5.54 mg/dL, p<0.0001). Notably, the mean Lp(a) in the case group exceeded the established cardiovascular risk threshold of 30 mg/dL, while controls remained well below this threshold. This is consistent with prior literature demonstrating early and progressive elevation of Lp(a) in CKD.

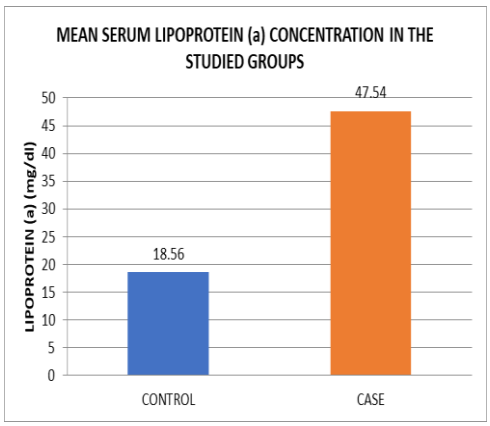

Figure 2: Comparison of mean lipoprotein (a) in controls and cases

Lipid Profile

As summarized in Table 2. The mean Serum Cholesterol concentration was 140.30 mg/dl and 162.32 mg/dl in the control and the case groups respectively. In the Unpaired t test between control and case groups, the two-tailed p value is 0.0031. This difference is considered very significant. The mean Serum Triglyceride concentration was 100.52 mg/dl and 149.82 mg/dl in the control and the case groups respectively. The two-tailed p value is <0.0001. This difference is extremely significant.

The mean Serum VLDL-Cholesterol concentration was 20.11 mg/dl and 30.02 mg/dl in the control and the case groups respectively. In the Unpaired t test between control and case groups, the two-tailed p value is <0.0001. This difference is considered extremely significant. The mean Serum HDL Cholesterol concentration was 46.52 mg/dl and 44.47 mg/dl in the control and the case groups respectively. The two-tailed p value is 0.1878. This difference is not significant. The mean Serum LDL Cholesterol concentration was 69.97mg/dl and 87.8 mg/dl in the control and the case groups respectively. In the Unpaired t test between control and case groups, the two-tailed p value is <0.0001. This difference between the two groups is extremely significant.

Correlation of Lp(a) with Other Parameters in CKD Cases

Within the CKD case group, Pearson's correlation analysis revealed significant positive correlations between serum Lp(a) and serum urea (r=0.51, p=0.0007), serum creatinine (r=0.45, p=0.0037), total

cholesterol (r=0.40, p=0.0101), triglycerides (r=0.77, p<0.0001), VLDL-cholesterol (r=0.77, p<0.0001), and LDL-cholesterol (r=0.45, p=0.0040). Serum Lp(a) showed a significant negative correlation with HDL-cholesterol (r=−0.43, p=0.0056). No significant correlations were found between Lp(a) and age, BMI, fasting plasma glucose, AST, or ALT.

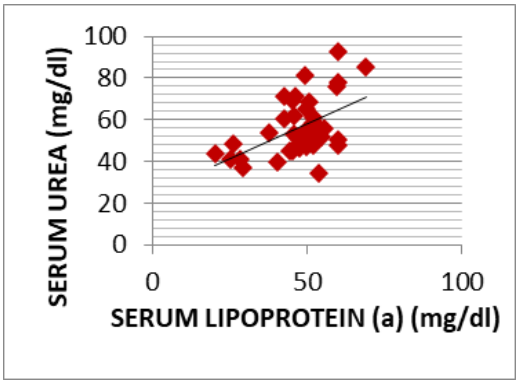

Figure 3: Showing correlation of lipoprotein(a) and serum urea in case group

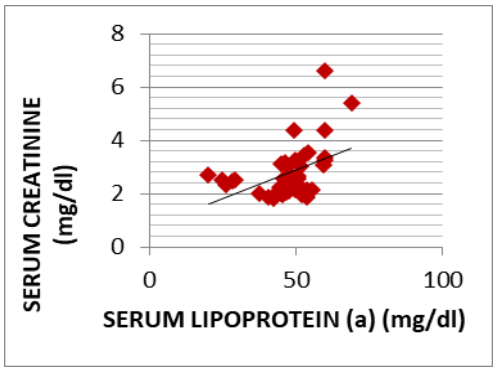

Figure 4: Showing correlation of lipoprotein(a) and serum creatinine in case group

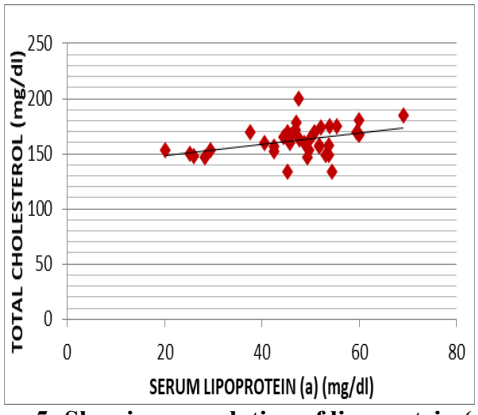

Figure 5: Showing correlation of lipoprotein (a) and total cholesterol in case group

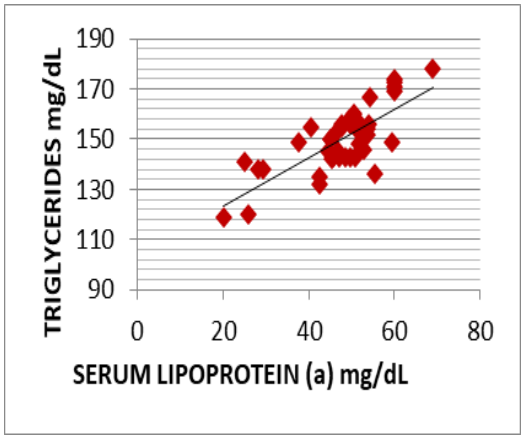

Figure 6: Showing correlation of lipoprotein (a) and triglyceride in case group

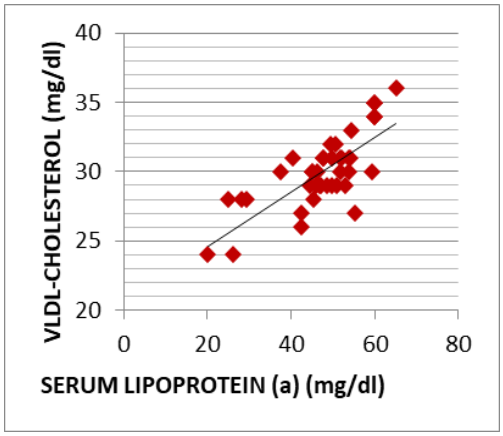

Figure 7: Showing correlation of lipoprotein (a) and VLDL cholesterol in case group

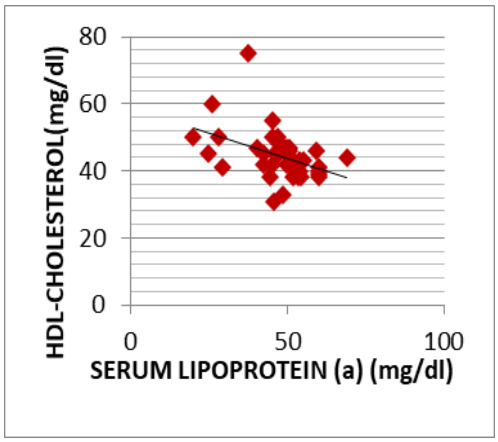

Figure 8: Showing correlation of lipoprotein (a) and HDL in case group

DISCUSSION

CKD has assumed the proportions of a significant public health problem. The present case-control study demonstrates that serum Lp(a) is markedly elevated in non-dialysis-dependent CKD patients compared

to healthy controls, and that this elevation correlates significantly with conventional markers of atherogenic dyslipidemia and renal dysfunction. These findings have important implications for cardiovascular risk stratification in CKD. There is strong evidence that lipoprotein (a) is a risk factor for CVD in the general population.[10] Due to its structural homology with plasminogen, lipoprotein (a) competes with this protein for binding to plasminogen receptors, fibrinogen and fibrin .[11] In kidney disease, GFR influences plasma Lp(a) levels.

The mean age of the case group was 51.55 ± 8.81 years in the present study which were in corroboration with a study in 2013 by *Singh* et al [2] (age distribution was 18 – 98 years and mean age of cases was 52.27 ± 14.78 years) and another study by *Mannangi* et al. [12]

In the present study, the majority of the patients were males, who constituted 60% of the total patients and 40 % were females which corroborated with study by *Singh* et al. [2]

In our study, mean BMI of the case group was 24.31 ± 3.69 kg/m^2 and for control was 23.62 ± 1.45 kg/m^2 which was not significantly different in the two groups. *Mannangi* et al [12] also did not find significant difference in BMI between the case and control groups.

The mean Systolic BP in cases was 147 ± 18.00 mm of Hg and in controls was 115.12 ± 7.73 mm of Hg, which was extremely significant as compared with controls p< 0.0001. The mean Diastolic BP in cases was 88.17mm ± 7.85 of Hg and 76.05 mm ± 4.90 of Hg in controls, the increase in Diastolic BP was also extremely significant (p< 0.0001). This corroborated with studies by *Singh* et al [2] who reported an extremely significant (p< 0.0001) elevation of BP in cases with 64.5% of cases were hypertensive. This signifies that the frequency of hypertension is higher in CKD patients than in controls.

In the present study, the average Serum lipoprotein (a) level in patients was significantly high as compared to controls. This suggests that lipoprotein (a) level is increased in non-dialysis–dependent chronic kidney disease. *Rao* et al. [5] in their study in 2010 also found that levels of lipoprotein (a) were significantly higher in patients. *Mannanagi* et al. [12] also reported a significant increase in lipoprotein(a) [Lp(a)] levels among cases compared with controls (p < 0.001). Elevated Lp(a) concentrations in chronic kidney disease (CKD) are recognized as an independent risk factor for premature atherosclerotic coronary heart disease. Although the precise mechanism underlying the cardiovascular risk associated with Lp(a) remains unclear, its proatherogenic and prothrombotic properties have been proposed as major contributing factors. Atherosclerotic renal disease accounts for more than one-third of all cases of end-stage renal disease. [13,14] Furthermore, CKD itself may contribute to increased Lp(a) levels through enhanced hepatic synthesis secondary to an acute-phase response or through protein losses associated with proteinuria. [14] *Koch* et al [15] in 1994 also observed Lp(a) levels were increased in patients of chronic kidney disease.

In patients undergoing hemodialysis, in vivo turnover studies using stable isotope techniques conducted by Frischmann KE et al. [16] demonstrated that the fractional catabolic rate of apolipoprotein(a) was significantly reduced, resulting in a prolonged plasma residence time of approximately 9 days compared with 4.4 days in controls. This impaired catabolism may contribute to the elevated circulating lipoprotein(a) [Lp(a)] levels observed in chronic kidney disease. Similarly, Mohanalakshmi P et al. [17] also reported significantly higher Lp(a) concentrations in cases compared with controls. *Haffner* et al [18] observed increase in Lipoprotein (a) in subjects with renal failure.

The average serum total cholesterol in the present study was 162.32 ± 13.11 mg/dL in cases and 140.30± 31.97 mg/dL in controls and these values were significantly high as compared to that of the normal age and sex-

matched controls ($p<0.05$) . *Rao* et al[5] also observed total cholesterol level raised in cases as compared to controls but not significantly. *Mohanalakshmi P* et al [17] also observed increase in total cholesterol level in cases. *Koch* et al [15], *Mannangi* et al [12] observed no significant difference in cases and control.

The average Serum LDL-Cholesterol was 87.80 ± 14.37mg/dL in the case group and significantly high compared to control group, 69.97 ± 30.89 mg/dL, $p<0.05$. *Mannangi* et al [12] observed significant increase in LDL-cholesterol, in cases as compared to controls. *Rao* et al [5] also observed rise in LDL-cholesterol level. *Bhagwa*t et al [19] also observed rise in LDL-cholesterol level but not significantly high. *Koch* et al [15] reported no change in LDL-cholesterol level.

The average serum triglyceride was 149.82 ± 13.13 mg/dL in the case group and this increase is extremely significant when compared to serum triglyceride level of 100.52 ± 23.89 mg/dL in the control group, $p<0.0001$. *Rao* et al [5] demonstrated a significant rise in Serum Triglyceride levels in cases as compared to controls ($p<0.05$). *Mannangi* et al [12] also showed a significant increase in Serum Triglyceride level. Increase in triglyceride was also observed by *Mohanalakshmi P* et al .[17] However, *Koch* et al [15] and demonstrated no change in Triglyceride level.

In the case group, the serum VLDL-Cholesterol level was 30.02 ±2.64 mg/dL, which is an extremely significant elevation ($p<0.0001$) as compared to controls. *Mohanalakshmi P* et al [17] also observed an increase in VLDL-Cholesterol level. *Mannangi* et al [12] observed no significant difference in VLDL-Cholesterol level.

Significant hypertriglyceridemia in chronic kidney disease may result from both increased production and reduced catabolism of triglyceride-rich lipoproteins.[20] Renal insufficiency is frequently associated with insulin resistance, which promotes hepatic very-low-density lipoprotein (VLDL) synthesis and contributes to elevated serum triglyceride levels. However, in predialysis patients, the predominant mechanism appears to be delayed catabolism with impaired clearance of these lipoproteins. This reduced catabolic capacity is largely attributed to decreased activity of endothelium-associated lipoprotein lipase enzymes, including hepatic lipase and lipoprotein lipase (LPL). Furthermore, downregulation of hepatic lipase and LPL gene expression, partly mediated by secondary hyperparathyroidism, may further aggravate dyslipidemia in these patients.[21]

On the other hand, the average Serum HDL-Cholesterol was 44.47 ±7.36 mg/dL, and there was no significant decrease when compared to controls. *Rao* et al [5] showed no significant change as well. *Mannangi* et al [12] also observed no changes in serum HDL-level.

Pearson's correlation was derived between Serum lipoprotein (a) and other biochemical parameters in the cases.

The significant positive correlations of Lp(a) with serum urea ($r=0.51$) and creatinine ($r=0.45$) in our study reinforce the mechanistic link between renal impairment and Lp(a) accumulation. As renal function deteriorates, reflected by rising urea and creatinine, Lp(a) clearance is progressively impaired, leading to its accumulation. This suggests that Lp(a) could serve as an early, sensitive biomarker of cardiovascular risk that tracks the severity of renal dysfunction.

The strong positive correlation of Lp(a) with total cholesterol ($r=0.40$), triglycerides ($r=0.77$) and VLDL ($r=0.77$) observed in our study reflects the broader dysregulation of lipoprotein metabolism in CKD. Reduced activity of lipoprotein lipase and hepatic lipase in uremia impairs VLDL clearance, leading to hypertriglyceridemia. The co-elevation of Lp(a) with these atherogenic particles creates a compounded cardiovascular risk. Lp(a)'s pro-thrombotic effect through competitive inhibition of plasminogen binding, combined with elevated triglyceride-rich lipoproteins, may

act synergistically to accelerate atherosclerosis in CKD.

The significant negative correlation of Lp(a) with HDL-cholesterol (r=−0.43) is also clinically relevant. HDL exerts anti-atherogenic effects through reverse cholesterol transport and antioxidant properties. The concurrent elevation of Lp(a) and reduction of HDL in CKD creates a particularly unfavorable lipoprotein profile that substantially heightens cardiovascular risk.

Strengths and Limitations

Strengths of this study include strict inclusion and exclusion criteria that minimized important confounders (diabetes, liver disease, smoking, and lipid-lowering medications), careful matching of controls for age and sex, and validated laboratory methods for all biochemical measurements. Limitations include the relatively small sample size (40 per group), the cross-sectional design that precludes causal inferences, the absence of CKD staging-specific sub-group analyses, and the lack of baseline pre-CKD Lp(a) measurements.

CONCLUSION

Serum lipoprotein(a) is significantly elevated in non-dialysis-dependent CKD patients and correlates positively with markers of renal dysfunction (serum urea and creatinine) and atherogenic dyslipidemia (triglycerides, VLDL, LDL, total cholesterol), and negatively with cardioprotective HDL-cholesterol. The mean Lp(a) in CKD patients exceeded the established cardiovascular risk threshold of 30 mg/dL. These findings suggest that routine estimation of serum Lp(a) in addition to standard lipid profiling may provide important incremental information for early identification of patients at high cardiovascular risk. Targeting elevated Lp(a) through emerging lipid-lowering therapies may represent a novel strategy to reduce the heavy cardiovascular burden in CKD.

Declaration by Authors

Ethical Approval: Approved by the Institutional Ethics Committee, Gauhati Medical College, vide letter no.MC/02/2015/225

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REFERENCES

1. Levey AS, Coresh J, Balk E, Kausz AT, Levin A, Steffes MW, Hogg RJ, Perrone RD, Lau J, Eknoyan G; National Kidney Foundation. National Kidney Foundation practice guidelines for chronic kidney disease: evaluation, classification, and stratification. Ann Intern Med. 2003 Jul 15;139(2):137-47. doi: 10.7326/0003-4819-139-2-200307150-00013. Erratum in: Ann Intern Med. 2003 Oct 7;139(7):605.
2. Singh AK, Farag YM, Mittal BV, Subramanian KK, Reddy SR, Acharya VN, Almeida AF, Channakeshavamurthy A, Ballal HS, P G, Issacs R, Jasuja S, Kirpalani AL, Kher V, Modi GK, Nainan G, Prakash J, Rana DS, Sreedhara R, Sinha DK, V SB, Sunder S, Sharma RK, Seetharam S, Raju TR, Rajapurkar MM. Epidemiology and risk factors of chronic kidney disease in India - results from the SEEK (Screening and Early Evaluation of Kidney Disease) study. BMC Nephrol. 2013 May 28;14:114. doi: 10.1186/1471-2369-14-114.
3. Sarnak MJ, Coronado BE, Greene T, Wang SR, Kusek JW, Beck GJ, Levey AS. Cardiovascular disease risk factors in chronic renal insufficiency. Clin Nephrol. 2002 May;57(5):327-35. doi: 10.5414/cnp57327.
4. Baigent C, Burbury K, Wheeler D. Premature cardiovascular disease in chronic renal failure. Lancet. 2000 Jul 8;356(9224):147-52. doi: 10.1016/S0140-6736(00)02456-9.
5. Rao AM, Bitla AR, Reddy EP, Sivakumar V, Srinivasa Rao PV. Lipid abnormalities, lipoprotein (a) and apoprotein pattern in non-dialyzed patients with chronic kidney disease. Indian J Clin Biochem. 2010 Jan;25(1):47-50. doi: 10.1007/s12291-010-0010-5. Epub 2010 Feb 10.

6. Jacobson TA. Lipoprotein(a), cardiovascular disease, and contemporary management. Mayo Clin Proc. 2013 Nov;88(11):1294-311. doi: 10.1016/j.mayocp.2013.09.003.
7. Kronenberg F, Kuen E, Ritz E, et al. Lipoprotein(a) serum concentrations and apolipoprotein(a) phenotypes in mild and moderate renal failure. Journal of the American Society of Nephrology: JASN. 2000 Jan;11(1):105-115. DOI: 10.1681/asn.v111105.
8. Sechi LA, Zingaro L, De Carli S, Sechi G, Catena C, Falleti E, Dell'Anna E, Bartoli E. Increased serum lipoprotein(a) levels in patients with early renal failure. Ann Intern Med. 1998 Sep 15;129(6):457-61. doi: 10.7326/0003-4819-129-6-199809150-00006.
9. Crook ED, Thallapureddy A, Migdal S, Flack JM, Greene EL, Salahudeen A, Tucker JK, Taylor HA Jr. Lipid abnormalities and renal disease: is dyslipidemia a predictor of progression of renal disease? Am J Med Sci. 2003 Jun;325(6):340-8. doi: 10.1097/00000441-200306000-00005.
10. Craig WY, Neveux LM, Palomaki GE, Cleveland MM, Haddow JE. Lipoprotein(a) as a risk factor for ischemic heart disease: meta-analysis of prospective studies. Clin Chem. 1998 Nov;44(11):2301-6.
11. Harpel PC, Gordon BR, Parker TS. Plasmin catalyzes binding of lipoprotein (a) to immobilized fibrinogen and fibrin. Proc Natl Acad Sci U S A. 1989 May;86(10):3847-51. doi: 10.1073/pnas.86.10.3847.
12. Mannangi NB, Jayaram S, Viruprakash HS, Kashinakunti S, Manjula R. Novel lipid indices in chronic kidney disease. Int J Med Sci Public Health. 2015;4(5):1-10.
13. Raitakari OT, Adams MR, Celermajer DS. Effect of Lp(a) on the early functional and structural changes of atherosclerosis. Arterioscler Thromb Vasc Biol. 1999 Apr;19(4):990-5. doi: 10.1161/01.atv.19.4.990.
14. Dieplinger H, Lackner C, Kronenberg F, Sandholzer C, Lhotta K, Hoppichler F, Graf H, König P. Elevated plasma concentrations of lipoprotein(a) in patients with end-stage renal disease are not related to the size polymorphism of apolipoprotein(a). J Clin Invest. 1993 Feb;91(2):397-401. doi: 10.1172/JCI116213.
15. Shah B, Nair S, Sirsat RA, Ashavaid TF, Nair KG. Dyslipidemia in patients with chronic renal failure and in renal transplant patients. J Postgrad Med. 1994 Apr-Jun;40(2):57-60.
16. Frischmann ME, Kronenberg F, Trenkwalder E, Schaefer JR, Schweer H, Dieplinger B, Koenig P, Ikewaki K, Dieplinger H. In vivo turnover study demonstrates diminished clearance of lipoprotein(a) in hemodialysis patients. Kidney Int. 2007 May;71(10):1036-43. doi: 10.1038/sj.ki.5002131.
17. Mohanalakshmi P, Silambanan S, Jothimalar R. Correlation of Lipoprotein(a) in normal individuals and in chronic kidney disease patients with Diabetes Mellitus. International J of current Microbiology and Applied Sciences. 2014; 3(3): 1074-1080.
18. Haffner SM, Gruber KK, Aldrete B, Morales PA, Stem MP, Katherine R. Increased Lipoprotein (a) concentrations in chronic kidney disease. J Am Soc Nephrol. 1992; 3:1156-1162.
19. Bhagwat R, Joshi SP, Salgia P, Sepaha A. Lipid abnormalities in chronic renal failure. Indian J Clin Biochem. 1997 Dec;12(1):81-5. doi: 10.1007/BF02867962.
20. Batista MC, Welty FK, Diffenderfer MR, Sarnak MJ, Schaefer EJ, Lamon-Fava S, et al. Apolipoprotein A-I, B-100, and B-48 metabolism in subjects with chronic kidney disease, obesity, and the metabolic syndrome. Metabolism. 2004 Oct;53(10):1255-61. doi: 10.1016/j.metabol.2004.05.001.
21. Klin M, Smogorzewski M, Ni Z, Zhang G, Massry SG. Abnormalities in hepatic lipase in chronic renal failure: role of excess parathyroid hormone. J Clin Invest. 1996 May 15;97(10):2167-73. doi: 10.1172/JCI118657.

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