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Apolipoproteins and Lipoprotein(a) in Relation to eGFR and Albuminuria: Evidence from a Rural Population Study

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Abstract

Background: *The relationship between dyslipidaemia—including both traditional and non-traditional lipid markers—and early renal dysfunction remains underexplored in South Asian community settings. This study evaluated the associations between apolipoproteins, lipoprotein(a) [Lp(a)], and traditional lipid parameters with estimated glomerular filtration rate (eGFR) and albuminuria in a rural Bangladeshi population.*

Methods: *A cross-sectional study was conducted among 201 adults participating in a community-based renal screening program. Clinical assessments included serum creatinine, eGFR, urinary albumin-creatinine ratio (ACR), fasting glucose, uric acid, traditional lipid profile, apolipoprotein A1 (ApoA1), apolipoprotein B (ApoB), and Lp(a). Correlations between lipid markers and renal parameters were examined, and subgroup comparisons were performed based on renal status and combined traditional lipid abnormalities.*

Results: *Mean eGFR was 94.28 ± 23.41 mL/min/1.73 m² and elevated albuminuria (ACR ≥30 mg/g) was present in 23.4% of participants. Traditional lipid markers correlated significantly with renal outcomes: eGFR was inversely associated with triglycerides (r = –0.242), total cholesterol (r = –0.342), and LDL cholesterol (r = –0.258). Participants with abnormal renal status had significantly higher triglycerides, total cholesterol, LDL cholesterol, and systolic blood pressure. Non-traditional lipid markers showed no significant differences by renal category, though ApoB and Lp(a) were higher in participants with combined traditional dyslipidaemia.*

Conclusion: *Traditional lipid abnormalities were strongly associated with early renal impairment, whereas non-traditional lipid markers showed limited discriminatory value. These findings highlight the importance of integrating lipid and albuminuria screening into rural primary care.*

Keywords: *Dyslipidaemia; Apolipoproteins; Lipoprotein(A); Albuminuria; eGFR*

I. INTRODUCTION

Chronic kidney disease (CKD) has emerged as a major global public health concern, with prevalence steadily rising across all regions and socioeconomic strata. The *Global Burden of Disease Study 2021* estimated that more than 800 million individuals worldwide are affected by CKD, making it one of the leading causes of mortality and disability-adjusted life years (DALYs).[1] The prevalence and mortality associated with CKD have shown consistent growth over the past three decades, particularly in low- and middle-income countries where diagnostic coverage remains poor. Early renal impairment often progresses silently; most patients remain asymptomatic until substantial nephron loss occurs, underscoring the importance of laboratory-based surveillance systems for early detection. In this context, identifying modifiable metabolic and cardiovascular risk factors that contribute to renal decline has become an essential step in global prevention strategies.[2,3]

Among the modifiable determinants of CKD, dyslipidaemia is a well-recognised yet under-addressed contributor to renal dysfunction. Traditional dyslipidaemia, characterised by elevated triglycerides, total cholesterol, and low-density lipoprotein cholesterol (LDL-C) together with low high-density lipoprotein cholesterol (HDL-C), is highly prevalent in both diabetic and non-diabetic populations. Experimental and clinical data suggest that lipid accumulation within renal tissue promotes glomerular endothelial dysfunction, mesangial expansion, and tubulointerstitial fibrosis.[4,5] Oxidised LDL and triglyceride-rich lipoproteins can generate reactive oxygen species and initiate pro-inflammatory cascades that lead to podocyte injury and extracellular matrix deposition, thereby accelerating nephron loss. The mechanisms linking dyslipidaemia to renal pathology—collectively referred to as "lipotoxic nephropathy"—mirror those observed in atherosclerosis, positioning lipid abnormalities as a common metabolic driver of both cardiovascular and renal diseases.[6]

Recent attention has shifted from conventional lipid fractions toward non-traditional lipid markers, particularly apolipoproteins and lipoprotein(a) [Lp(a)], as potential predictors of renal risk. Apolipoproteins are structural proteins that regulate the metabolism and receptor recognition of lipoprotein particles. Apolipoprotein B (ApoB) is present on all atherogenic particles, very-low-density lipoprotein (VLDL), intermediate-density lipoprotein (IDL), and LDL, thus representing the total number of circulating atherogenic lipoproteins. In contrast, apolipoprotein A1 (ApoA1) is the principal component of HDL particles and is considered anti-atherogenic due to its role in reverse cholesterol transport. Consequently, the ApoB/ApoA1 ratio is increasingly used as an integrated index of atherogenic risk, and evidence suggests it may better reflect particle burden than cholesterol concentrations alone.[7] Parallel to this, Lp(a); a genetically determined LDL-like particle containing apolipoprotein(a), has emerged as a causal factor in cardiovascular disease and, more recently, as a candidate biomarker for renal injury.[8,9]

Several population-based studies have examined the relationship between apolipoproteins and renal outcomes, yet the evidence remains inconsistent. In large community cohorts, higher ApoB or an elevated ApoB/ApoA1 ratio has been associated with lower estimated glomerular filtration rate (eGFR) and a greater likelihood of CKD progression.[8,9] However, other investigations failed to demonstrate consistent longitudinal associations after adjustment for traditional lipids and confounders, suggesting that population differences, comorbidities, and measurement techniques may influence results. Evidence on Lp(a) and renal outcomes is similarly heterogeneous: while elevated Lp(a) has been linked to reduced eGFR and higher serum creatinine in Chinese and diabetic cohorts, its relation to albuminuria or subclinical renal injury is less clear.[4,9,11] Such variability indicates that the role of these non-traditional markers in renal dysfunction remains under-explored, and their potential clinical application for early CKD detection is still uncertain.

The scarcity of data is even more pronounced in South Asian settings, where dyslipidaemia and CKD often coexist at high prevalence but are poorly characterised outside hospital cohorts. Bangladesh exemplifies this gap. Nationwide estimates indicate that nearly nine in ten Bangladeshi adults exhibit at least one form of dyslipidaemia, with low HDL-C being most common.[12] Systematic reviews report a CKD prevalence approaching 17 %, much of it undiagnosed due to limited laboratory screening and health-care access in rural and semi-urban communities.[13] Despite this, lipid screening in Bangladeshi primary-care practice typically focuses on total cholesterol and triglycerides; measurements of apolipoproteins and Lp(a) remain rare. The regional dyslipidaemic phenotype, characterised by low HDL-C, elevated triglycerides, and high small dense LDL, is well-documented among South Asians and is thought to contribute to their disproportionate burden of cardiometabolic disease.[14] In such populations, introducing apolipoprotein and Lp(a) assessment could provide earlier insight into atherogenic and renal risk.

Furthermore, rural communities in Bangladesh face distinct barriers to prevention and early diagnosis. Late clinical presentation, scarcity of laboratory facilities, and limited awareness of metabolic risk contribute to delayed recognition of both dyslipidaemia and CKD. The combination of high cardiometabolic risk, low screening coverage, and evolving rural lifestyles makes these settings particularly vulnerable. Establishing population-based evidence on non-traditional lipid markers and their association with renal function could therefore help tailor early-detection strategies suitable for low-resource contexts.

Against this background, the present study aimed to examine the relationships between apolipoprotein A1, apolipoprotein B, and lipoprotein(a) with renal function parameters, estimated glomerular filtration rate (eGFR) and urinary albumin-creatinine ratio (ACR), in a rural Bangladeshi population. By evaluating both traditional and non-traditional lipid markers, this study seeks to clarify their interrelations and assess whether these markers can provide additional insights into early renal risk in community settings where CKD burden is growing rapidly yet remains under-recognized.

II. METHODS

This cross-sectional study was conducted over a 12-month period in the Baidyerbazar Union of Sonargaon Upazila, Narayanganj District, Bangladesh, an area where the Kidney Care and Research Center (KCRC) runs an ongoing community-based screening program for chronic kidney disease. The study population comprised adults aged 18 years or older residing in the selected rural community. Using the voter list as a sampling frame, eligible individuals were invited to attend designated weekly screening sessions, during which participants were recruited consecutively following verification of inclusion and exclusion criteria. Individuals were excluded if they were pregnant, had known malignancy, acute kidney injury, established chronic kidney disease under active treatment, or cognitive impairment that limited informed participation. After providing written informed consent, all participants underwent structured clinical and biochemical assessment. The total sample included 201 adult participants, consistent with the thesis dataset.

Sociodemographic variables collected included age, sex, education, occupation, height, weight, and calculated body mass index. Clinical variables included history of diabetes mellitus, hypertension, smoking status, and obesity. Blood pressure was measured using a standard calibrated sphygmomanometer after adequate rest. Venous blood samples were collected following an overnight fast to measure serum creatinine, fasting blood glucose, HbA1c, hemoglobin, uric acid, total protein, albumin, and a comprehensive fasting lipid panel comprising triglycerides, total cholesterol, HDL cholesterol, LDL cholesterol, apolipoprotein A1, apolipoprotein B, and lipoprotein(a). Urine samples were obtained for urinalysis (R/M/E) and for measurement of the spot urinary albumin-creatinine ratio (ACR). Estimated glomerular filtration rate (eGFR) was calculated using the Modification of Diet in Renal Disease (MDRD) equation. Dyslipidaemia was evaluated using both traditional lipid markers (triglycerides, total cholesterol, LDL-C, HDL-C) and non-traditional markers (ApoA1, ApoB, Lp(a)).

All biochemical analyses were performed at the designated local laboratory associated with the KCRC program, following standard operational procedures and internal quality-control protocols. To evaluate the association between lipid markers and renal function parameters, two primary analytical approaches were applied. Pearson's correlation coefficients were calculated to quantify the relationships between traditional lipid markers and continuous renal biomarkers, including serum creatinine, eGFR, ACR, serum total protein, serum albumin, and uric acid. Comparative analyses between renal-function subgroups were conducted using unpaired t-tests. These subgroup comparisons included: (i) participants with normal versus abnormal renal status based on combined ACR and eGFR categories, and (ii) participants with normal versus abnormal combined traditional lipid abnormalities (TG + total cholesterol + LDL-C). Statistical significance was interpreted at a two-sided p-value < 0.05. All analyses were performed using standard statistical software in accordance with the analytic framework documented in the thesis dataset. Ethical approval was obtained from the appropriate institutional review authority, and all procedures adhered to the principles of the Declaration of Helsinki.

III. RESULTS

Table 1. Demographic and Clinical Characteristics of the Study Population (n = 201)

Category / Mean ± SD	n (%)
Age (years)	
≤30	45 (22.4)
31–40	63 (31.3)
41–50	48 (23.9)
51–60	25 (12.4)
>60	20 (10.0)
Mean ± SD	41.4 ± 13.8
Sex	
Male	96 (47.8)
Female	105 (52.2)
BMI categories (kg/m²)	
Underweight (<18.5)	10 (5.0)
Normal (18.5–24.9)	94 (46.8)
Overweight (25–29.9)	76 (37.8)
Obese (≥30)	21 (10.4)
Mean ± SD	25.0 ± 4.2
Blood pressure (mmHg)	

Systolic BP (Mean ± SD)	129 ± 18
Diastolic BP (Mean ± SD)	81 ± 11
Primary diseases	
Diabetes mellitus	29 (14.4)
Hypertension	40 (19.9)
Nephropathy	26 (12.9)

The study population consisted of 201 adults with a mean age of 41.4 ± 13.8 years. Most participants were between 31–40 years (31.3 %), followed by those aged 41–50 years (23.9 %), while only 10 % were older than 60 years. Females comprised a slightly higher proportion of the cohort (52.2 %) compared with males (47.8 %). The mean BMI was 25.0 ± 4.2 kg/m², with nearly half of the participants having normal weight (46.8 %), while 37.8 % were overweight and 10.4 % were obese; only 5 % were underweight. Mean systolic and diastolic blood pressures were 129 ± 18 mmHg and 81 ± 11 mmHg, respectively. Regarding comorbid conditions, hypertension was present in 19.9 % of participants, diabetes mellitus in 14.4 %, and nephropathy in 12.9 %.

Table 2. Laboratory Parameters of Study Participants (n = 201)

Parameter	Mean ± SD	Range
Serum creatinine (mg/dL)	0.82 ± 0.22	0.50–1.80
eGFR (mL/min/1.73 m²)	94.28 ± 23.41	6–148
Urine ACR (mg/g)	37.57 ± 89.54	1.6–713.5
Fasting blood glucose (mmol/L)	6.26 ± 2.36	4.1–20.0
HbA1c (%)	6.17 ± 1.57	4.6–14.6
Uric acid (mg/dL)	5.57 ± 1.11	2.2–10.3
Serum total protein (g/dL)	7.59 ± 0.73	5.4–8.9
Serum albumin (g/dL)	4.84 ± 0.57	3.0–6.0
Hemoglobin (g/dL)	13.52 ± 1.68	8.4–16.8

Serum creatinine levels in the study population averaged 0.82 ± 0.22 mg/dL, with values ranging from 0.50 to 1.80 mg/dL, while the mean eGFR was 94.28 ± 23.41 mL/min/1.73 m² (range: 6–148). Urinary albumin-creatinine ratio showed wide variability, with a mean of 37.57 ± 89.54 mg/g and a range from 1.6 to 713.5 mg/g. The mean fasting blood glucose level was 6.26 ± 2.36 mmol/L, and HbA1c averaged 6.17 ± 1.57 %. Uric acid levels demonstrated a mean of 5.57 ± 1.11 mg/dL, whereas serum total protein and serum albumin averaged 7.59 ± 0.73 g/dL and 4.84 ± 0.57 g/dL, respectively. Hemoglobin values ranged from 8.4 to 16.8 g/dL with a mean of 13.52 ± 1.68 g/dL, reflecting overall adequate hematologic status in the cohort.

Table 3. Lipid and Apolipoprotein Profile of Participants (n = 201)

Marker	Mean ± SD	Range
Triglycerides (mg/dL)	182.98 ± 104.94	58–781
Total cholesterol (mg/dL)	196.35 ± 47.44	93–325
LDL cholesterol (mg/dL)	121.04 ± 39.07	33.8–222
HDL cholesterol (mg/dL)	38.97 ± 6.65	26–56
ApoA1 (g/L)	1.39 ± 0.97	0.22–13.90*
ApoB (g/L)	1.07 ± 0.45	0.22–2.66
Lipoprotein(a) [mg/dL]	20.02 ± 12.78	1.14–81.86

Mean triglyceride levels in the study population were 182.98 ± 104.94 mg/dL, with values ranging widely from 58 to 781 mg/dL. Total cholesterol averaged 196.35 ± 47.44 mg/dL, and LDL cholesterol showed a mean of 121.04 ± 39.07 mg/dL, spanning from 33.8 to 222 mg/dL. HDL cholesterol levels were comparatively lower, with a mean of 38.97 ± 6.65 mg/dL. Among the non-traditional lipid markers, ApoA1 demonstrated a mean concentration of 1.39 ± 0.97 g/L, though the range (0.22–13.90 g/L) indicated notable variability. ApoB levels averaged 1.07 ± 0.45 g/L, while lipoprotein(a) values showed a mean of 20.02 ± 12.78 mg/dL, ranging from 1.14 to 81.86 mg/dL.

Table 4. Correlation of Traditional Lipid Markers with Renal and Biochemical Variables (n = 201)

Outcome Variable	Lipid Marker	r	p-value
Total protein	TG	0.162	0.022
	Total cholesterol	0.405	<0.001
	LDL	0.326	<0.001
Uric acid	TG	0.298	<0.001
	Total cholesterol	0.395	<0.001
	LDL	0.296	<0.001
Albumin	TG	0.235	0.001
	Total cholesterol	0.403	<0.001
	LDL	0.310	<0.001
Serum creatinine	TG	0.020	0.783
	Total cholesterol	0.164	0.020
	LDL	0.190	0.007
eGFR	TG	–0.242	0.001
	Total cholesterol	–0.342	<0.001
	LDL	–0.258	<0.001

Traditional lipid markers showed several significant correlations with renal and biochemical parameters. Triglycerides demonstrated modest positive correlations with serum total protein ($r = 0.162$, $p = 0.022$), uric acid ($r = 0.298$, $p < 0.001$), and serum albumin ($r = 0.235$, $p = 0.001$), but were not significantly associated with serum creatinine. Total cholesterol and LDL cholesterol exhibited stronger positive correlations with total protein ($r = 0.405$ and 0.326, respectively; both $p < 0.001$), uric acid ($r = 0.395$ and 0.296; both $p < 0.001$), and albumin ($r = 0.403$ and 0.310; both $p < 0.001$). For renal function markers, total cholesterol ($r = 0.164$, $p = 0.020$) and LDL ($r = 0.190$, $p = 0.007$) showed significant positive correlations with serum creatinine, whereas triglycerides showed no meaningful association. In contrast, all three lipid markers demonstrated significant negative correlations with eGFR, with the strongest association observed for total cholesterol ($r = –0.342$, $p < 0.001$), followed by LDL ($r = –0.258$, $p < 0.001$) and triglycerides ($r = –0.242$, $p = 0.001$).

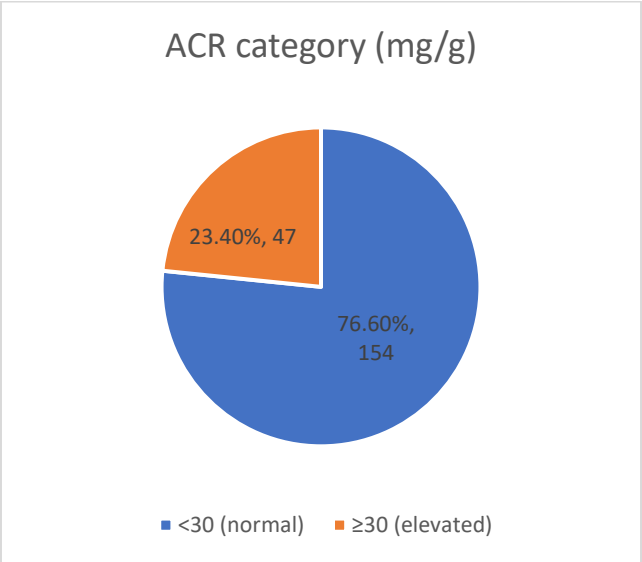

Figure 1. Distribution of Albuminuria Levels (ACR) Among Study Participants (n = 201)

76.6% (n = 154) had an ACR below 30 mg/g, while 23.4% (n = 47) demonstrated elevated albuminuria (≥30 mg/g). This indicates that nearly one-quarter of the rural cohort had evidence of increased urinary albumin excretion, reflecting early renal structural injury.

Table 5. Comparison of Demographic, Renal, and Lipid Variables by Renal Status (Normal vs Abnormal eGFR+ACR)

Variable	Normal eGFR+ACR (n=143) Mean ± SD	Abnormal eGFR+ACR (n=58) Mean ± SD	p-value
Age (years)	38.86 ± 12.43	47.81 ± 15.11	<0.001
BMI	24.66 ± 3.76	25.84 ± 5.01	0.071
Serum creatinine (mg/dL)	0.79 ± 0.17	0.88 ± 0.29	0.009
eGFR (mL/min/1.73 m²)	98.49 ± 17.83	93.87 ± 17.83	<0.001
ACR (mg/g)	8.75 ± 5.44	108.62 ± 144.34	<0.001
TG (mg/dL)	170.90 ± 106.07	212.74 ± 96.65	0.010
Total cholesterol (mg/dL)	187.58 ± 46.59	217.94 ± 42.66	<0.001
LDL (mg/dL)	114.39 ± 37.51	137.43 ± 38.25	<0.001
HDL (mg/dL)	38.60 ± 6.49	39.86 ± 7.01	0.225
SBP (mmHg)	128.09 ± 17.70	138.05 ± 20.72	0.001
ApoA1 (g/L)	1.38 ± 1.12	1.40 ± 0.42	0.895
ApoB (g/L)	1.04 ± 0.46	1.11 ± 0.41	0.379
Lp(a) (mg/dL)	18.89 ± 13.78	18.31 ± 12.16	0.779

Participants with abnormal renal status, defined by the combined eGFR and ACR criteria, demonstrated markedly different clinical and biochemical profiles compared with those with normal renal parameters. Individuals in the abnormal group were significantly older (47.81 ± 15.11 vs. 38.86 ± 12.43 years, $p < 0.001$) and had higher serum creatinine levels (0.88 ± 0.29 vs. 0.79 ± 0.17 mg/dL, $p = 0.009$), alongside lower mean eGFR values (93.87 ± 17.83 vs. 98.49 ± 17.83 mL/min/1.73 m², $p < 0.001$). Albuminuria showed a pronounced difference between groups, with the abnormal group displaying a markedly elevated mean ACR (108.62 ± 144.34 mg/g) compared with those with normal renal status (8.75 ± 5.44 mg/g; $p < 0.001$). Traditional lipid parameters were also significantly higher in the abnormal group, including triglycerides (212.74 ± 96.65 vs. 170.90 ± 106.07 mg/dL, $p = 0.010$), total cholesterol (217.94 ± 42.66 vs. 187.58 ± 46.59 mg/dL, $p < 0.001$), and LDL cholesterol (137.43 ± 38.25 vs. 114.39 ± 37.51 mg/dL, $p < 0.001$). Systolic blood pressure was similarly elevated among those with abnormal renal function (138.05 ± 20.72 vs. 128.09 ± 17.70 mmHg, $p = 0.001$). In contrast, no significant differences were observed for HDL cholesterol, ApoA1, ApoB, or Lp(a), suggesting that the divergence in renal status was primarily reflected through traditional lipid abnormalities rather than non-traditional lipid markers.

Table 6. Comparison of Laboratory Variables by Combined Traditional Lipid Abnormality Status (TG+TC+LDL)

Variable	Normal (n=39) Mean ± SD	Abnormal (n=162) Mean ± SD	p-value
Age (years)	38.64 ± 16.31	42.12 ± 13.13	0.159
Serum creatinine (mg/dL)	0.75 ± 0.21	0.83 ± 0.21	0.036
eGFR (mL/min/1.73 m²)	105.00 ± 19.06	91.70 ± 23.68	0.001
ACR (mg/g)	14.64 ± 33.91	43.09 ± 97.62	0.075
FBS (mmol/L)	5.49 ± 1.14	6.44 ± 2.54	0.023
Uric acid (mg/dL)	5.03 ± 0.93	5.69 ± 1.11	0.001
TG (mg/dL)	105.90 ± 23.43	201.53 ± 108.47	<0.001
Total cholesterol (mg/dL)	133.82 ± 18.63	211.40 ± 39.23	<0.001
LDL (mg/dL)	75.46 ± 14.25	132.01 ± 34.98	<0.001
HDL (mg/dL)	37.51 ± 6.82	39.31 ± 6.59	0.129
SBP (mmHg)	126.21 ± 17.09	132.11 ± 19.45	0.083
DBP (mmHg)	76.64 ± 9.40	81.76 ± 11.32	0.010
ApoA1 (g/L)	1.27 ± 0.43	1.41 ± 1.06	0.416
ApoB (g/L)	0.91 ± 0.53	1.10 ± 0.42	0.019
Lp(a) (mg/dL)	14.20 ± 9.24	19.82 ± 13.93	0.017
ApoB/ApoA1 ratio	0.90 ± 1.16	0.85 ± 0.41	0.679

Participants with combined traditional lipid abnormalities (elevated triglycerides, total cholesterol, and LDL cholesterol) demonstrated significantly different metabolic and renal profiles compared with those who had normal traditional lipid levels. Individuals in the abnormal lipid group showed higher serum creatinine

concentrations (0.83 ± 0.21 vs. 0.75 ± 0.21 mg/dL, p = 0.036) and lower mean eGFR values (91.70 ± 23.68 vs. 105.00 ± 19.06 mL/min/1.73 m², p = 0.001), indicating reduced renal function. Although ACR levels were higher in the abnormal group (43.09 ± 97.62 vs. 14.64 ± 33.91 mg/g), the difference did not reach statistical significance (p = 0.075). Markers of metabolic stress were more pronounced in the abnormal group, including higher fasting blood glucose (6.44 ± 2.54 vs. 5.49 ± 1.14 mmol/L, p = 0.023) and uric acid levels (5.69 ± 1.11 vs. 5.03 ± 0.93 mg/dL, p = 0.001).

As expected, traditional lipid values were markedly elevated among participants with abnormal profiles, including triglycerides (201.53 ± 108.47 vs. 105.90 ± 23.43 mg/dL, p < 0.001), total cholesterol (211.40 ± 39.23 vs. 133.82 ± 18.63 mg/dL, p < 0.001), and LDL cholesterol (132.01 ± 34.98 vs. 75.46 ± 14.25 mg/dL, p < 0.001). Blood pressure values were modestly higher in the abnormal group, with diastolic blood pressure showing a significant difference (81.76 ± 11.32 vs. 76.64 ± 9.40 mmHg, p = 0.010). Regarding non-traditional lipid markers, ApoB (1.10 ± 0.42 vs. 0.91 ± 0.53 g/L, p = 0.019) and Lp(a) levels (19.82 ± 13.93 vs. 14.20 ± 9.24 mg/dL, p = 0.017) were significantly higher among participants with abnormal traditional lipid levels, while ApoA1 values and the ApoB/ApoA1 ratio did not differ significantly between groups.

IV. DISCUSSION

The present study examined traditional and non-traditional lipid markers in relation to renal function indicators within a rural Bangladeshi population, revealing several patterns that merit comparison with existing evidence. Participants demonstrated a relatively young mean age of 41 years, a balanced sex distribution, and a high prevalence of overweight and obesity, reflecting the ongoing epidemiological transition observed across South Asian populations. Rising cardiometabolic risk in Bangladesh, including increasing rates of hypertension and dyslipidaemia, has been demonstrated in national surveys and population-level analyses, supporting the demographic profile observed in this cohort.[15] These characteristics are consistent with community-based samples undergoing lifestyle transitions and underscore the need for early metabolic and renal risk assessment in rural populations.

Renal and biochemical parameters in this study indicated generally preserved kidney function at the population level, with a mean eGFR of 94 mL/min/1.73 m², though albuminuria was present in nearly one-quarter of participants. Comparable community-based studies have demonstrated that albuminuria prevalence ranging from 20% to 30% is strongly associated with adverse renal and cardiovascular outcomes, even in individuals with normal or near-normal eGFR values.[16] Elevated uric acid levels observed in the sample also align with growing evidence that uric acid contributes to renal functional decline and interacts with metabolic disturbances.[17] These findings reinforce the clinical relevance of albuminuria and uric acid as early indicators of renal stress in asymptomatic community populations.

The lipid profile of participants demonstrated substantial abnormalities, with high triglycerides, elevated LDL cholesterol, and relatively low HDL cholesterol. These patterns are consistent with the atherogenic dyslipidaemia commonly reported in Asian and South Asian populations. Importantly, the study revealed that traditional lipids correlated significantly with renal biomarkers: triglycerides, total cholesterol, and LDL cholesterol all showed inverse relationships with eGFR and positive associations with serum creatinine and albuminuria. Similar associations have been reported in large community-based studies, where mixed dyslipidaemia patterns, particularly elevated triglycerides and LDL cholesterol, were linked to lower eGFR and higher proteinuria.[18,19] These parallels suggest that traditional lipid abnormalities remain important markers of renal vulnerability in populations with rising metabolic risk.

Comparisons of renal subgroups further strengthened these observations. Participants with abnormal renal status (elevated ACR and/or reduced eGFR) were older and exhibited significantly higher triglycerides, total cholesterol, and LDL cholesterol than those with preserved renal function. Similar gradients of lipid-renal impairment have been documented in both diabetic and non-diabetic populations, where lipid abnormalities contribute to microvascular dysfunction and albuminuria.[20] Elevated systolic blood pressure in the abnormal renal group adds further support to the clustering of metabolic stressors in early renal impairment.

In contrast to the strong associations observed for traditional lipids, non-traditional lipid markers in this study—ApoA1, ApoB, and Lp(a)—did not differ between renal function groups. This contrasts with some published evidence, including work demonstrating positive associations between ApoB and CKD risk and between ApoB and uric acid levels.[21,22] However, the absence of significant between-group differences in the present cohort may reflect population variability, small effect sizes, or early-stage renal changes not yet sensitive to apolipoprotein alterations. Notably, individuals with combined traditional lipid abnormalities did exhibit higher ApoB and Lp(a) levels, a pattern consistent with previous studies reporting that non-traditional markers rise in parallel with atherogenic lipid loads and endothelial dysfunction.[23]

Overall, the findings from this rural Bangladeshi population align closely with global and regional evidence linking dyslipidaemia, hyperuricaemia, elevated blood pressure, and albuminuria with early renal impairment. The lack of significant associations for apolipoproteins and Lp(a) across renal function groups highlights the need for further research into non-traditional lipid markers in South Asian community settings, where cardiometabolic risk is rapidly evolving.

Limitations of The Study

This study was cross-sectional and therefore could not establish causal relationships between lipid markers and renal outcomes. The sample was drawn from a single rural community, which may limit generalizability to broader populations. Additionally, non-traditional lipid markers such as ApoA1, ApoB, and Lp(a) were assessed at a single time point, preventing evaluation of temporal changes or long-term renal implications.

V. CONCLUSION

In this rural Bangladeshi population, traditional lipid abnormalities were consistently associated with early indicators of renal impairment, including higher serum creatinine, lower eGFR, and elevated albuminuria. Non-traditional lipid markers showed limited discriminatory value across renal status categories, although ApoB and Lp(a) were elevated among those with combined traditional dyslipidaemia. These findings underscore the strong renal relevance of traditional atherogenic lipids in low-resource settings undergoing rapid epidemiological transition. Routine assessment of conventional lipid markers may offer practical value for early detection of renal vulnerability, while the role of apolipoproteins and Lp(a) warrants further investigation in longitudinal studies.

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