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Traditional lipid fractions and renal function in a rural Bangladeshi cohort: Correlation findings

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Abstract

Background: Dyslipidemia is increasingly recognized as a contributor to renal dysfunction, yet community-level evidence from rural South Asia remains limited. This study examined the association between traditional lipid fractions and renal function among adults in a rural Bangladeshi cohort.

Methods: A cross-sectional study was conducted among 201 adults from Baidyerbazar Union, Sonargaon, Bangladesh. Sociodemographic, clinical, and biochemical data were collected through structured interviews and fasting blood tests. Renal function was assessed using serum creatinine, estimated glomerular filtration rate (eGFR, Modification of Diet in Renal Disease), and urine albumin-to-creatinine ratio (ACR). Traditional lipid markers (triglycerides [TG], total cholesterol [TC], low-density lipoprotein cholesterol [LDL-C], high-density lipoprotein cholesterol [HDL-C]) and non-traditional markers (apolipoprotein A1, apolipoprotein B, lipoprotein [a]) were measured. Correlation analyses and group comparisons were performed.

Results: Mean lipid levels indicated widespread dyslipidemia: TG 182.98 ± 104.94 mg/dL, TC 196.35 ± 47.44 mg/dL, LDL-C 121.04 ± 39.07 mg/dL, and HDL-C 38.97 ± 6.65 mg/dL. Albuminuria (ACR ≥30 mg/g) was present in 23.4% of participants. eGFR showed significant inverse correlations with TG ($r = -0.242$, $P = 0.001$), TC ($r = -0.342$, $P < 0.001$), and LDL-C ($r = -0.258$, $P < 0.001$). Participants with renal impairment had significantly higher TG, TC, and LDL-C (all $P < 0.01$). Non-traditional lipid markers showed limited association with renal indices.

Conclusion: Traditional lipid abnormalities are strongly linked to early renal dysfunction in this rural Bangladeshi population. Community-based lipid and renal screening may facilitate early identification of high-risk individuals.

Keywords: Dyslipidemia, estimated glomerular filtration rate, renal function, rural Bangladesh, triglycerides

Introduction

Chronic kidney disease (CKD) has emerged as a critical global health issue, afflicting more than 10% of the world's population and posing substantial burdens on individuals, families, and healthcare systems.[1] In low- and middle-income countries, particularly within South Asia, this burden is amplified by limited healthcare access, late-stage diagnosis, and the dual challenge of communicable and non-communicable diseases.[2] Bangladesh, despite being one of the most densely populated countries in the region, still lacks comprehensive community-level data

on early renal dysfunction and its association with common cardiovascular risk markers such as lipid abnormalities. Rapid urbanization, shifting dietary habits, and socioeconomic transition in rural areas have given rise to a growing prevalence of metabolic disorders, including dyslipidemia, even in previously low-risk populations.[3] The need to investigate CKD and its risk markers among these underrepresented populations, especially in rural Bangladeshi communities, has become increasingly urgent.

Dyslipidemia has been extensively linked to cardiovascular diseases, but mounting evidence suggests a strong association with renal dysfunction as well.[4,5] Elevated serum triglycerides (TG), total cholesterol (TC), and low-density lipoprotein cholesterol (LDL-C), along with decreased high-density lipoprotein cholesterol (HDL-C), have been identified not only as cardiovascular risk markers but also as potential contributors to the progression of CKD. In a large prospective cohort from Taiwan, Chen *et al*. demonstrated that higher quintiles of TC, non-HDL-C, and LDL-C significantly increased the risk of rapid estimated glomerular filtration rate (eGFR) decline and eventual progression to end-stage renal disease.[4] Similarly, findings from a longitudinal Japanese community-based study confirmed that dyslipidemia, particularly high TG and low HDL-C, was independently associated with incident CKD in non-diabetic adults over a 26-year follow-up.[5] Such associations suggest a direct pathophysiological link between lipid metabolism and renal impairment, possibly mediated through mechanisms such as lipid accumulation in renal tissue, oxidative stress, and endothelial dysfunction.[6]

Mechanistic insights into lipid-induced renal damage have also deepened our understanding of this relationship. Recent work by Han *et al*. reviewed the biological role of lipid dysregulation in diabetic kidney disease (DKD), showing that excessive free fatty acid uptake, intracellular lipid accumulation, and dysregulated sphingolipid metabolism contribute to podocyte injury, mesangial expansion, and tubulointerstitial fibrosis.[6] These processes, though initially observed in diabetic contexts, have relevance beyond hyperglycemia, highlighting how altered lipid profiles can independently initiate renal structural damage even in non-diabetic individuals. Such mechanisms reinforce the clinical relevance of lipid profile surveillance in identifying early renal decline in community populations.

Despite the increasing evidence of this lipid-renal linkage in global settings, Bangladesh-specific data remain scarce. Most CKD-related studies from the country have been confined to urban hospital-based samples or patients with known comorbidities such as diabetes or hypertension.[7] In a study of Bangladeshi type 2 diabetes patients, CKD prevalence was found to be as high as 21.3%, but the role of lipid fractions was not systematically explored, highlighting a critical data gap in national renal epidemiology. Moreover, population-level lipid studies in Bangladesh, such as the recent analysis by Ali *et al*., have revealed alarmingly high prevalence of hypertriglyceridemia (51.7%), hypercholesterolemia (41.6%), and low HDL-C (78.8%) in both rural and urban adults.[3] However, these studies do not link lipid profiles with renal markers such as eGFR or serum creatinine. This lack of correlation data is a missed opportunity for early detection and risk stratification, particularly in resource-constrained rural health systems.

Rural populations in South Asia, including Bangladesh, face a unique dual burden: While they still contend with undernutrition and infectious diseases, they are now also increasingly affected by chronic metabolic conditions due to lifestyle shifts, dietary westernization, and reduced physical activity.[8] Rural Bangladesh remains underserved in public health research, especially with respect to chronic diseases such as CKD. Studies such as the one by Sarker *et al*., which found a CKD prevalence of 22% in a peri-urban Bangladeshi sample, point to the importance of community-based surveillance. Yet, few investigations explore the interplay between lipid levels and renal function within this demographic. Given that traditional lipid testing is relatively accessible and low-cost, its potential

as a surveillance tool for identifying early renal dysfunction in rural populations is immense.

Therefore, this study aims to fill a crucial research gap by exploring the correlation between traditional lipid fractions, TC, TG, LDL-C, HDL-C, and renal function markers in a rural Bangladeshi cohort. By examining these associations in a community-based setting, our findings can help inform context-appropriate, affordable screening strategies for early detection of renal impairment, with implications for both prevention and health policy in low-resource settings. This work not only builds on global evidence of lipid–renal interconnections but also contributes novel data from an underrepresented and high-risk population.

Methods

This cross-sectional analytical study was conducted to evaluate the association between traditional lipid fractions and renal function among adults living in a rural Bangladeshi community. The research was carried out over a 12-month period at the National Institute of Kidney Diseases and Urology (NIKDU), Dhaka, in collaboration with the Kidney Care and Research Center, which operates a community-based screening program in Baidyerbazar Union of Sonargaon Upazila, Narayanganj District. The study population comprised adult residents (aged ≥18 years) of the selected rural union. Individuals who were pregnant, had known malignancies, acute kidney injury, CKD under treatment, or significant psychiatric illness were excluded. A total of 201 participants were selected purposively from the voter list of the community through consecutive sampling during weekly screening sessions. After obtaining informed written consent, participants underwent structured interviews and clinical assessments. Demographic data (age, sex, occupation, education, body mass index [BMI], and blood pressure) and clinical information (diabetes mellitus, hypertension, smoking status, and obesity) were recorded using a standardized questionnaire. Venous blood samples were collected after an overnight fast for laboratory analyses, including fasting blood sugar (FBS), glycosylated hemoglobin (HbA1C), TC, TG, LDL-C, and HDL-C. Additional biomarkers measured were serum creatinine, uric acid, total protein, albumin, hemoglobin, and non-traditional lipid parameters – apolipoprotein A1 (ApoA1), apolipoprotein B (ApoB), and lipoprotein (a) [Lp(a)]. A spot urine sample was collected for urine routine examination and albumin-to-creatinine ratio (ACR) determination. Serum creatinine was measured by the Jaffe kinetic method, and eGFR was calculated using the modification of diet in renal disease (MDRD) equation.

Renal function status was categorized based on eGFR and ACR values, with participants divided into normal and abnormal renal function groups for comparative analysis. Dyslipidemia was defined according to standard cutoffs: TC ≥200 mg/dL, TG ≥150 mg/dL, LDL-C ≥130 mg/dL, or HDL-C < 40 mg/dL. Data were checked for completeness, consistency, and normality before analysis. Descriptive statistics (means, standard deviations, and proportions) were used to summarize participant characteristics. Pearson's correlation coefficient was applied to assess the relationships between lipid parameters and renal function indices (serum creatinine, eGFR, ACR, uric acid, albumin, and total protein). Unpaired *t*-tests were conducted to evaluate differences between normal and abnormal renal function groups based on lipid abnormalities and other covariates. All analyses were performed using standard statistical software, and a $P < 0.05$ was considered statistically significant. Ethical approval was obtained from the institutional ethics committee of NIKDU, and all procedures followed the ethical principles outlined in the Declaration of Helsinki. Participants were provided with counseling on their test results, and those found with significant abnormalities were referred to specialized care at NIKDU for further management.

Results

Among the 201 participants included in the study, the mean age was 41.4 ± 13.8 years, with the majority (31.3%) aged between 31 and 40 years,

followed by 23.9% between 41 and 50 years. Slightly more than half of the participants were female (52.2%), indicating near gender balance in the study population. The average BMI was 25.0 ± 4.2 kg/m^2, with almost half of the participants having normal weight (46.8%), whereas 37.8% were overweight and 10.4% were obese, reflecting a substantial burden of excess body weight. Mean systolic and diastolic blood pressures were 129 ± 18 mmHg and 81 ± 11 mmHg, respectively, suggesting an overall trend toward pre-hypertensive levels. Regarding comorbid conditions, hypertension was the most prevalent (19.9%), followed by diabetes mellitus (14.4%) and nephropathy (12.9%), highlighting the coexistence of cardiometabolic and renal risk factors even within this rural cohort [Table 1].

The mean serum creatinine among participants was 0.82 ± 0.22 mg/dL, with a corresponding mean eGFR of 94.28 ± 23.41 mL/min/1.73 m^2, indicating that the majority had preserved renal function. However, albuminuria was noted in nearly one-quarter of participants, with 23.4% showing an ACR ≥ 30 mg/g, reflecting evidence of early renal involvement in a considerable subset of the population. FBS averaged 6.26 ± 2.36 mmol/L, and mean HbA1c was 6.17 ± 1.57%, suggesting the presence of mild hyperglycemia in part of the cohort. Uric acid levels were moderately elevated (5.57 ± 1.11 mg/dL), whereas serum total protein and albumin averaged 7.59 ± 0.73 g/dL and 4.84 ± 0.57 g/dL, respectively, remaining within physiological ranges. The mean hemoglobin concentration was 13.52 ± 1.68 g/dL, indicating an overall absence of anemia in most participants [Table 2].

The lipid profile of the study participants revealed considerable variability, with evidence of widespread dyslipidemia. Mean TG levels were 182.98 ± 104.94 mg/dL, and TC averaged 196.35 ± 47.44 mg/dL, both approaching or exceeding conventional upper normal limits for a healthy population. The mean LDL-C concentration was 121.04 ± 39.07 mg/dL, whereas HDL-C was notably low at 38.97 ± 6.65 mg/dL, suggesting a pattern of atherogenic dyslipidemia characterized by elevated TG and LDL-C with reduced HDL-C. Non-traditional lipid markers showed mean ApoA1 of 1.39 ± 0.97 g/L, ApoB of 1.07 ± 0.45 g/L, and Lp(a) levels averaging 20.02 ± 12.78 mg/dL [Table 3].

Correlation analysis demonstrated significant associations between traditional lipid fractions and several renal and biochemical markers. TC and LDL-C showed strong positive correlations with serum total protein (r = 0.405 and 0.326, respectively; $P < 0.001$), uric acid (r = 0.395 and 0.296; $P < 0.001$), and serum albumin (r = 0.403 and 0.310; $P < 0.001$), suggesting concurrent metabolic elevations in lipid and protein indices. Similarly, TG were positively correlated with serum total protein (r = 0.162; $P = 0.022$), uric acid (r = 0.298; $P < 0.001$), and albumin (r = 0.235; $P = 0.001$). With respect to renal function markers, serum creatinine correlated positively with TC (r = 0.164; $P = 0.020$) and LDL-C (r = 0.190; $P = 0.007$), whereas eGFR demonstrated significant inverse correlations with all three lipid parameters – TG (r = −0.242; $P = 0.001$), TC (r = −0.342; $P < 0.001$), and LDL-C (r = −0.258; $P < 0.001$). These findings indicate that elevated lipid levels, particularly TC and LDL-C, are associated with impaired renal function and metabolic burden within this rural cohort [Table 4].

Participants with combined renal impairment, defined by abnormal eGFR and/or ACR values, were older on average (47.81 ± 15.11 years) than those with normal renal function (38.86 ± 12.43 years; $P < 0.001$), suggesting age as a key determinant of renal decline. Although BMI was slightly higher among those with renal impairment, the difference did not reach statistical significance ($P = 0.071$). Serum creatinine levels were significantly elevated in the abnormal group (0.88 ± 0.29 vs. 0.79 ± 0.17 mg/dL; $P = 0.009$), whereas mean eGFR and ACR values showed marked deterioration ($P < 0.001$ for both). Lipid abnormalities were more pronounced among participants with renal impairment, with higher mean TG (212.74 ± 96.65 vs. 170.90 ± 106.07 mg/dL; $P = 0.010$), TC (217.94 ± 42.66 vs. 187.58 ± 46.59 mg/dL; $P < 0.001$), and LDL-C (137.43 ± 38.25 vs. 114.39 ± 37.51 mg/dL;

Table 1: Demographic and clinical characteristics of participants (*n*=201)

Category/summary	*n* (%) or Mean±SD
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 (kg/m^2)	
Underweight (<18.5)	10 (5.0)
Normal (18.5–24.9)	94 (46.8)
Overweight (25.0–29.9)	76 (37.8)
Obese (≥30.0)	21 (10.4)
Mean±SD	25.0±4.2
Blood pressure (mmHg)	
Systolic	129±18
Diastolic	81±11
Comorbidities	
Diabetes mellitus	29 (14.4)
Hypertension	40 (19.9)
Nephropathy	26 (12.9)

BMI: Body mass index, SD: Standard deviation

Table 2: Renal function and general laboratory indices

Analyte	Mean±SD	Min-Max
Serum creatinine (mg/dL)	0.82±0.22	0.50–1.80
eGFR (mL/min/1.73 m^2, MDRD)	94.28±23.41	6–148
ACR (mg/g)	37.57±89.54	1.60–713.50
Fasting blood sugar (mmol/L)	6.26±2.36	4.10–20.00
HbA1c (%)	6.17±1.57	4.60–14.60
Uric acid (mg/dL)	5.57±1.11	2.20–10.30
Serum total protein (g/dL)	7.59±0.73	5.40–8.90
Serum albumin (g/dL)	4.84±0.57	3.00–6.00
Hemoglobin (g/dL)	13.52±1.68	8.40–16.80

ACR: Albumin-to-creatinine ratio, MDRD: Modification of diet in renal disease, HbA1c: Glycosylated hemoglobin, SD: Standard deviation

Table 3: Lipid markers and dyslipidemia prevalence

Marker	Mean±SD	Min–Max
TG (mg/dL)	182.98±104.94	58–781
TC (mg/dL)	196.35±47.44	93–325
LDL-C (mg/dL)	121.04±39.07	33.8–222
HDL-C (mg/dL)	38.97±6.65	26–56
ApoA-I (g/L)	1.39±0.97	0.22–13.90
ApoB (g/L)	1.07±0.45	0.22–2.66
Lp (a) (mg/dL)	20.02±12.78	1.14–81.86

ApoA-I: Apolipoprotein A1, ApoB: Apolipoprotein B, Lp (a): Lipoprotein (a), LDL-C: Low-density lipoprotein cholesterol, HDL-C: High-density lipoprotein cholesterol, SD: Standard deviation, TC: Total cholesterol, TG: Triglycerides

Table 4: Pearson correlations of traditional lipids with renal and biochemical markers (*n*=201)

Outcome	TG	Total cholesterol	LDL-C
Serum total protein	0.162 (0.022)	0.405 (<0.001)	0.326 (<0.001)
Uric acid	0.298 (<0.001)	0.395 (<0.001)	0.296 (<0.001)
Serum albumin	0.235 (0.001)	0.403 (<0.001)	0.310 (<0.001)
Serum creatinine	0.020 (0.783)	0.164 (0.020)	0.190 (0.007)
eGFR	−0.242 (0.001)	−0.342 (<0.001)	−0.258 (<0.001)

TG: Triglycerides, LDL-C: Low-density lipoprotein cholesterol, eGFR: Estimated glomerular filtration rate

$P < 0.001$). HDL-C showed no significant variation between groups ($P = 0.225$). Systolic blood pressure was significantly higher in the abnormal renal function group (138.05 ± 20.72 vs. 128.09 ± 17.70 mmHg; $P = 0.001$) [Table 5].

Participants with traditional dyslipidemia – defined by abnormalities in any of the three parameters TG, TC, or LDL-C – showed clear biochemical differences compared with those having normal lipid profiles. The dyslipidemic group ($n = 162$) had significantly higher serum creatinine levels (0.83 ± 0.21 vs. 0.75 ± 0.21 mg/dL; $P = 0.036$) and notably lower mean eGFR (91.70 ± 23.68 vs. 105.00 ± 19.06 mL/min/1.73 m^2; $P = 0.001$), suggesting a

direct association between lipid derangement and renal function decline. Although mean ACR was higher in the abnormal lipid group (43.09 ± 97.62 vs. 14.64 ± 33.91 mg/g), this difference approached but did not reach statistical significance (P = 0.075). FBS levels were significantly elevated in the dyslipidemic group (P = 0.023), as were serum uric acid levels (P = 0.001), indicating broader metabolic disturbance. Expectedly, TG, TC, and LDL-C values were markedly higher in the abnormal group (all P < 0.001), whereas HDL-C remained comparable between groups (P = 0.129). Systolic blood pressure tended to be higher among those with dyslipidemia, though not significantly (P = 0.083), whereas diastolic pressure showed a modest but significant rise (P = 0.010). Among non-traditional lipids, both ApoB (P = 0.019) and Lp(a) (P = 0.017) were significantly elevated in the dyslipidemic group, suggesting their potential contributory role [Table 6].

Figure 1 shows that eGFR is inversely correlated with TG, TC, and LDL-C. As eGFR decreases, levels of these lipids increase, indicating that declining renal function is associated with worsening dyslipidemia in the study population.

Figure 2 shows a mild positive correlation between serum creatinine and TC as well as LDL-C. Higher creatinine values correspond to increased cholesterol and LDL levels, indicating that reduced renal function is associated with elevated atherogenic lipids.Figure 3 shows a clear positive correlation between serum uric acid and lipid levels. As uric acid increases,

TG, TC, and LDL-C also rise, indicating that higher uric acid is associated with greater lipid abnormalities.

Discussion

Our rural Bangladeshi cohort showed generally preserved kidney function on average; yet nearly

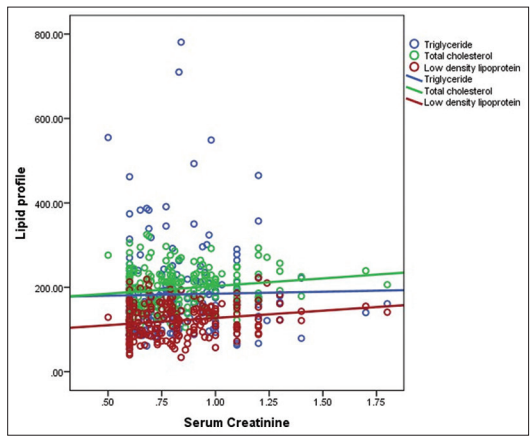

Figure 2: Scattered diagram showing the correlation of serum creatinine with lipid profiles

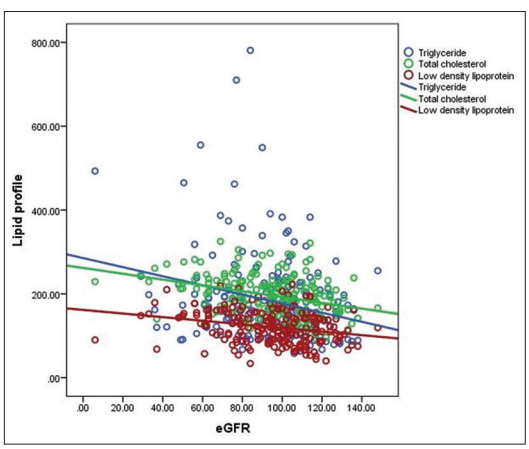

Figure 1: Scattered diagram showing correlation of estimated glomerular filtration rate with lipid profiles

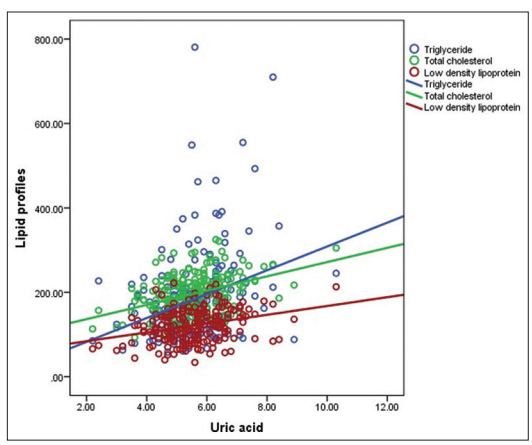

Figure 3: Scattered diagram showing the correlation of uric acid with lipid profiles

Table 5: Comparison by combined renal impairment status (eGFR+ACR)

Variable	Abnormal (*n*=58) (Mean±SD)	Normal (*n*=143) (Mean±SD)	*P*-value
Age (years)	47.81±15.11	38.86±12.43	<0.001
BMI (kg/m^2)	25.84±5.01	24.66±3.76	0.071
Serum creatinine (mg/dL)	0.88±0.29	0.79±0.17	0.009
eGFR (mL/min/1.73 m^2)	93.87±17.83	98.49±17.83	<0.001
ACR (mg/g)	108.62±144.34	8.75±5.44	<0.001
Triglycerides (mg/dL)	212.74±96.65	170.90±106.07	0.010
Total cholesterol (mg/dL)	217.94±42.66	187.58±46.59	<0.001
LDL-C (mg/dL)	137.43±38.25	114.39±37.51	<0.001
HDL-C (mg/dL)	39.86±7.01	38.60±6.49	0.225
SBP (mmHg)	138.05±20.72	128.09±17.70	0.001
ApoA (g/L)	1.40±0.42	1.38±1.12	0.895
ApoB (g/L)	1.11±0.41	1.04±0.46	0.379
Lp (a) (mg/dL)	18.31±12.16	18.89±13.78	0.779

eGFR: Estimated glomerular filtration rate, ACR: albumin-to-creatinine ratio, SD: Standard deviation, BMI: Body mass index, LDL-C: Low-density lipoprotein cholesterol, HDL-C: High-density lipoprotein cholesterol, SBP: Systolic blood pressure, Apo: Apolipoprotein, Lp (a): Lipoprotein (a)

Table 6: Comparison by traditional dyslipidemia composite (TG+TC+LDL)

Variable	Abnormal (*n*=162) (Mean±SD)	Normal (*n*=39) (Mean±SD)	*P*-value
Age (years)	42.12±13.13	38.64±16.31	0.159
Serum creatinine (mg/dL)	0.83±0.21	0.75±0.21	0.036
eGFR (mL/min/1.73 m^2)	91.70±23.68	105.00±19.06	0.001
ACR (mg/g)	43.09±97.62	14.64±33.91	0.075
Fasting blood sugar (mmol/L)	6.44±2.54	5.49±1.14	0.023
Uric acid (mg/dL)	5.69±1.11	5.03±0.93	0.001
TG (mg/dL)	201.53±108.47	105.90±23.43	<0.001
TC (mg/dL)	211.40±39.23	133.82±18.63	<0.001
LDL-C (mg/dL)	132.01±34.98	75.46±14.25	<0.001
HDL-C (mg/dL)	39.31±6.59	37.51±6.82	0.129
SBP (mmHg)	132.11±19.45	126.21±17.09	0.083
DBP (mmHg)	81.76±11.32	76.64±9.40	0.010
ApoA (g/L)	1.41±1.06	1.27±0.43	0.416
ApoB (g/L)	1.10±0.42	0.91±0.53	0.019
Lp (a) (mg/dL)	19.82±13.93	14.20±9.24	0.017
ApoB/ApoA	0.85±0.41	0.90±1.16	0.679

TG: Triglycerides, TC: Total cholesterol, eGFR: Estimated glomerular filtration rate, ACR: Albumin-to-creatinine ratio, SD: Standard deviation, LDL-C: Low-density lipoprotein cholesterol, HDL-C: High-density lipoprotein cholesterol, SBP: Systolic blood pressure, Apo: Apolipoprotein, Lp (a): Lipoprotein (a), DBP: Diastolic blood pressure

one in four had albuminuria, and lipid abnormalities were common. The 23.4% albuminuria aligns with community and peri-urban estimates from Bangladesh and the region, indicating a substantial

pool of early renal injury outside hospital settings.[9,10] Mean eGFR sat in the healthy range, but its gradients tracked cleanly with traditional lipids: eGFR correlated inversely with TG, TC, and LDL-C; serum creatinine rose with TC and LDL-C; HDL-C showed no meaningful between-group difference. These patterns echo large general-population analyses where higher TC and LDL-C, and atherogenic ratios, are associated with prevalent CKD or lower eGFR, and they are consistent with longitudinal signals that dyslipidemia, particularly higher TG and lower HDL-C, presage incident CKD in community cohorts.[5,11,12] In clinical CKD samples, higher TG relates to lower measured GFR and more advanced stages; our findings mirror that directionality despite a community design and a younger mean age (Wang *et al*., 2016). Taken together, the data situate traditional lipid fractions as practical correlates of subtle renal dysfunction in rural adults; this is important where affordable screening hinges on routine chemistry.

The renal-impaired subgroup in our data was older and had higher systolic blood pressure; both are canonical risk factors that travel with kidney decline and can amplify lipid–renal coupling via vascular and tubulointerstitial pathways.[1,2] Mechanistically, lipid accumulation in podocytes and tubular cells, altered fatty-acid handling, and ceramide signalling can drive mesangial expansion, podocyte injury, and fibrosis; such processes have been synthesized most clearly in DKD but are biologically relevant beyond hyperglycemia.[6] Our cohort also showed higher fasting glucose and uric acid among those with composite dyslipidemia; these clusters fit broader metabolic risk constellations and the observed positive correlations between uric acid and all three traditional lipids, while population data link higher uric acid to lower eGFR and cardiovascular risk.[13] At a country level, the lipid elevations we observed are plausible: Nationwide open-access analyses report high prevalence of hypertriglyceridemia, elevated LDL-C, and low HDL-C among Bangladeshi adults amid dietary and lifestyle transitions; our rural figures sit within that changing landscape.[3]

Non-traditional lipids told a subtler story. We did not see significant ApoA1, ApoB, or Lp(a) differences by renal-function status; this contrasts with community data where ApoB tracked independently with CKD and with studies linking higher Lp(a) to mildly reduced eGFR or early impairment, including in diabetes.[14-16] Several explanations are credible: Our sample was relatively young, community-based, and not enriched for diabetes; single-time measurements and modest subgroup sizes limit power to detect small Apo differences; and residual confounding by diet, statin use, or hormonal status cannot be excluded. Nevertheless, ApoB and Lp(a) were higher among those with composite dyslipidemia, hinting that non-traditional markers may still refine risk stratification when combined with standard lipids and renal indices, a point raised by studies showing stronger signals for lipid ratios and non-HDL constructs than for single fractions.[11]

This discussion should be read with our design in mind: cross-sectional data preclude causal inference; the single-union, purposive sampling constrains generalizability; MDRD-based eGFR and single-spot ACR can misclassify early disease; and we did not model confounding with multivariable regression. Even so, the convergence of findings across continuous correlations, binary group contrasts, and external literature strengthens the central message: In rural Bangladeshi adults, higher traditional lipid fractions, particularly TC and LDL-C, with supportive signals for TG, track with lower eGFR, higher creatinine, and albuminuria; this relationship persists alongside age and blood-pressure effects and within a broader metabolic milieu. In low-resource settings, these results support incorporating routine lipid panels into community renal surveillance and argue for integrated management of dyslipidemia and blood pressure to blunt early renal decline.[3,5,9]

Limitations of the study

This study was conducted in a single rural union, which may limit the generalizability of the findings to other regions of Bangladesh. The cross-sectional

design does not allow causal inference between lipid abnormalities and renal function impairment. In addition, non-traditional lipid markers were measured, but their interpretation may be limited by the relatively small subgroup variations.

Conclusion

This study demonstrates that traditional lipid fractions – particularly TG, TC, and LDL-C – are significantly associated with early markers of renal dysfunction among adults living in a rural Bangladeshi community. Participants with elevated lipid levels exhibited higher serum creatinine, lower eGFR, and greater albuminuria, reflecting a clear pattern of metabolic and renal interdependence. Non-traditional lipid markers showed limited discriminatory value across renal function groups, highlighting the continued importance of traditional lipid parameters for risk stratification in low-resource settings. The findings underscore the need for routine lipid and renal screening at the community level to identify high-risk individuals earlier, especially in rural populations undergoing rapid epidemiological transition.

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Conflict of Interest

None declared.

Ethical Approval

The study was approved by the Institutional Ethics Committee.

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