THE PLANET

Original Article

Association of Serum Apolipoprotein B-Apolipoprotein A-I Ratio with Renal Function Decline in Patients with Chronic Kidney Disease

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ARTICLE INFO

Received: 21 Feb 2026
Accepted: 27 Feb 2026
Published Online: 1 Mar 2026

DOI: 10.5281/zenodo.18816515

Volume: 9, Number: 1, Page: 79-82

e-ISSN: 2789-5912
ISSN: 2617-0817

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ABSTRACT

Background: Dyslipidemia is common in chronic kidney disease (CKD) and contributes to accelerated atherosclerosis and disease progression. The serum apolipoprotein B/apolipoprotein A-I (ApoB/ApoA-I) ratio is an established marker of atherogenic risk; however, its role in predicting renal function decline in CKD patients remains inadequately explored, particularly in South Asian populations. **Objective:** To evaluate the association between serum ApoB/ApoA-I ratio and renal function decline in patients with chronic kidney disease. **Methods & Materials:** This prospective cohort study was conducted at IOJH Hospital, Dhaka, Bangladesh, from January 2023 to December 2024. A total of 80 CKD patients were enrolled using purposive sampling. Renal function decline was defined as a ≥25% reduction in eGFR or progression to a higher CKD stage during follow-up. Data were analyzed using SPSS version 23.0. **Results:** The mean age of participants was 52.6 ± 11.4 years, with a male predominance (62.5%). The mean ApoB/ApoA-I ratio was significantly higher in patients who experienced renal function decline compared to those who did not ($p<0.001$). The ApoB/ApoA-I ratio showed a significant negative correlation with eGFR ($p<0.001$). After adjustment for age, sex, diabetes, hypertension, and baseline eGFR, a higher ApoB/ApoA-I ratio remained an independent predictor of renal function decline ($p=0.002$). **Conclusion:** An elevated serum ApoB/ApoA-I ratio is independently associated with accelerated renal function decline in CKD patients. This ratio may serve as a simple and useful biomarker for risk stratification and early intervention in chronic kidney disease.

Keywords: *Apolipoprotein A-I, Apolipoprotein B, Chronic kidney disease, Dyslipidemia, eGFR, Renal function decline.*

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INTRODUCTION

Chronic kidney disease (CKD) is a growing global public health problem, affecting approximately 10–13% of the adult population worldwide and contributing substantially to morbidity, mortality, and healthcare costs [1,2]. The burden of CKD is particularly pronounced in low- and middle-income countries, including Bangladesh, due to the rising prevalence of diabetes mellitus, hypertension, population aging, and limited access to early diagnostic and preventive care [3]. Progressive decline in renal function ultimately leads to end-stage kidney disease (ESKD), which is associated with poor quality of life and increased cardiovascular mortality [4]. Cardiovascular disease (CVD) remains the leading cause of death among patients with CKD, exceeding the risk of progression to ESKD in many cases [5]. Traditional cardiovascular risk factors alone cannot fully explain the markedly increased cardiovascular burden observed in CKD patients. Consequently, non-traditional risk factors, such as chronic inflammation, oxidative stress, endothelial dysfunction, and abnormalities in lipid metabolism, have gained increasing attention [6]. Dyslipidemia in CKD is characterized by qualitative and quantitative alterations in lipoproteins rather than simple elevations in total cholesterol levels. Typical lipid abnormalities include elevated triglycerides, increased levels of small dense low-density lipoprotein (LDL) particles, and reduced high-density lipoprotein (HDL) functionality [7]. Apolipoproteins, the protein components of lipoproteins, provide a more accurate representation of atherogenic and anti-atherogenic lipid particles. Apolipoprotein B (ApoB) reflects the total number of atherogenic lipoproteins, including LDL and very-low-density lipoprotein, whereas apolipoprotein A-I (ApoA-I) is the main structural protein of HDL and plays a protective role through reverse cholesterol transport [8]. The ApoB/ApoA-I ratio has emerged as a robust marker of cardiovascular risk, outperforming traditional lipid parameters in predicting atherosclerotic events in both the general population and high-risk groups [9]. This ratio reflects the balance between atherogenic and anti-atherogenic forces and has been shown to correlate with endothelial dysfunction, inflammation, and plaque burden [10]. In recent years, interest has grown in exploring the role of the ApoB/ApoA-I ratio beyond cardiovascular outcomes, particularly its association with renal disease progression. Emerging evidence suggests that dyslipidemia may directly contribute to renal injury through mechanisms such as lipid accumulation in glomerular and tubular cells, promotion of oxidative stress, and activation of pro-inflammatory pathways [11]. Several observational studies have reported associations between adverse lipid profiles and accelerated decline in estimated glomerular filtration rate (eGFR) in CKD patients [12]. However, data focusing specifically on apolipoprotein-based markers, especially the ApoB/ApoA-I ratio, remain limited and inconsistent, with most studies conducted in Western or East Asian populations. In Bangladesh, research on apolipoprotein profiles in CKD patients is scarce, and the prognostic significance of the ApoB/ApoA-I ratio for renal function decline has not been well established. Given ethnic differences in lipid metabolism, cardiovascular risk, and CKD progression, locally generated evidence is essential [13]. Identifying simple, cost-effective biomarkers that can predict renal function deterioration may facilitate early risk stratification and targeted interventions. Therefore, this prospective cohort study was

designed to evaluate the association between the serum ApoB/ApoA-I ratio and renal function decline in patients with chronic kidney disease attending a tertiary care hospital in Bangladesh.

METHODS & MATERIALS

This prospective cohort study was conducted at IOJH Hospital, Dhaka, Bangladesh, from January 2023 to December 2024. A total of 80 patients diagnosed with chronic kidney disease (CKD) were enrolled using purposive sampling. CKD was defined according to the kidney disease: Improving Global Outcomes (KDIGO) guidelines as evidence of kidney damage or reduced estimated glomerular filtration rate (eGFR <60 mL/min/1.73 m²) persisting for at least three months. Patients were recruited from both outpatient and inpatient nephrology services.

Inclusion criteria: Adult patients aged ≥18 years with established CKD stages 2–5, who provided informed written consent, were included in the study. Participants were required to have a stable clinical status at enrollment and available baseline laboratory investigations, including serum apolipoprotein B and apolipoprotein A-I levels.

Exclusion criteria: Patients with acute kidney injury, nephrotic syndrome, active infection, chronic liver disease, malignancy, pregnancy, or a history of renal transplantation were excluded. Individuals receiving lipid-lowering therapy within the preceding three months were also excluded to avoid confounding effects on apolipoprotein levels.

Study procedure: Baseline demographic data, clinical history, and comorbid conditions were recorded using a structured questionnaire. Blood samples were collected after an overnight fast for biochemical analysis. Serum ApoB and ApoA-I levels were measured using standardized immunoassay methods, and the ApoB/ApoA-I ratio was calculated. Renal function was assessed by serum creatinine and eGFR at baseline and during follow-up.

Data analysis: Data were analyzed using SPSS version 23.0. Continuous variables were expressed as mean ± standard deviation, and categorical variables as frequencies and percentages. Associations between the ApoB/ApoA-I ratio and renal function decline were assessed using correlation analysis and multivariate regression, with statistical significance set at $p < 0.05$.

RESULT

A total of 80 patients with chronic kidney disease were included in the final analysis. The mean age of the study population was 52.6 ± 11.4 years, with most patients belonging to the 41–60-year age group (45.0%). Male participants constituted 62.5% of the cohort. Hypertension (70.0%) and diabetes mellitus (55.0%) were the most common comorbidities. At baseline, a majority of patients were in CKD stage 3 (42.5%), followed by stage 4 (31.2%), stage 2 (15.0%), and stage 5 (11.3%) *Table I.*

Table I
Baseline demographic and clinical characteristics of the study population (*n*=80)

Variables	n/Mean	%/SD
Age (years)	52.6	±11.4
Male sex	50	62.5%
Hypertension	56	70.0%
Diabetes mellitus	44	55.0%
CKD stage 2	12	15.0%
CKD stage 3	34	42.5%
CKD stage 4	25	31.2%
CKD stage 5	9	11.3%

The baseline biochemical profile showed a mean serum creatinine level of 2.18 ± 0.86 mg/dL and a mean eGFR of 41.3 ± 16.7 mL/min/1.73 m². The mean serum ApoB and ApoA-I levels were 98.4 ± 21.6 mg/dL and 112.7 ± 24.1 mg/dL, respectively, resulting in a mean ApoB/ApoA-I ratio of 0.87 ± 0.19. Higher ApoB/ApoA-I ratios were observed in patients with advanced CKD stages (*Table II*).

Table II
Baseline biochemical parameters of the study population.

Parameters	Mean	SD
Serum creatinine (mg/dL)	2.18	0.86
eGFR (mL/min/1.73 m²)	41.3	16.7
ApoB (mg/dL)	98.4	21.6
ApoA-I (mg/dL)	112.7	24.1
ApoB/ApoA-I ratio	0.87	0.19

During the follow-up period, renal function decline was documented in 38 (47.5%) patients, defined as a ≥25% reduction in eGFR or progression to a higher CKD stage. Patients with renal function decline had a significantly higher mean ApoB/ApoA-I ratio compared to those without decline (0.96 ± 0.18 vs. 0.78 ± 0.16; $p < 0.001$) (*Table III*).

Table III
Comparison of the ApoB/ApoA-I ratio between patients with and without renal function decline.

Renal function status	ApoB/ApoA-I ratio (Mean ± SD)	p-value
Decline present (n=38)	0.96 ± 0.18	<0.001
No decline (n=42)	0.78 ± 0.16	

Data analysis test: Independent samples t-test

A greater proportion of patients with elevated ApoB/ApoA-I ratios (≥0.90) experienced renal deterioration (68.4%) compared to those with lower ratios (28.6%) *Table IV*.

Table IV
Distribution of renal function decline according to ApoB/ApoA-I ratio category.

ApoB/ApoA-I ratio	Renal decline	
	Present	**Absent**
<0.90 (n = 42)	12 (28.6%)	30 (71.4%)
≥0.90 (n = 38)	26 (68.4%)	12 (31.6%)

p-value: 0.001, Data analysis test: Chi-square test.

Correlation analysis demonstrated a significant negative relationship between the ApoB/ApoA-I ratio and baseline eGFR ($r = -0.42$, $p < 0.001$), indicating that a higher atherogenic burden was associated with poorer renal function. The ratio also showed a modest but significant association with annual eGFR decline ($r = -0.36$, $p = 0.002$) *Table V*.

Table V
Correlation of ApoB/ApoA-I ratio with renal function parameters.

Variables	Correlation coefficient (r)	p-value
Baseline eGFR	−0.42	<0.001
Annual eGFR decline	−0.36	0.002

Data analysis test: Pearson correlation.

Multivariate logistic regression analysis revealed that an elevated ApoB/ApoA-I ratio was an independent predictor of renal function decline after adjustment for age, sex, diabetes mellitus, hypertension, and baseline eGFR (adjusted OR = 2.74; 95% CI: 1.32–5.68; p = 0.007). Diabetes mellitus and lower baseline eGFR were also significantly associated with renal deterioration, whereas age and sex did not show independent significance (*Table VI*).

Table VI
Multivariate logistic regression analysis for predictors of renal function decline.

Variables	Adjusted OR (95% CI)	p-value
ApoB/ApoA-I ratio (high)	2.74 (1.32–5.68)	0.007
Diabetes mellitus	2.11 (1.01–4.42)	0.047
Hypertension	1.38 (0.64–2.98)	0.409
Baseline eGFR (low)	3.26 (1.54–6.91)	0.002

Data analysis test: Multivariate logistic regression.

DISCUSSION

This prospective cohort study demonstrated a significant association between an elevated serum apolipoprotein B/apolipoprotein A-I (ApoB/ApoA-I) ratio and accelerated renal function decline in patients with chronic kidney disease (CKD). Nearly half of the study participants experienced deterioration of renal function during follow-up, and those patients had significantly higher ApoB/ApoA-I ratios compared to individuals with stable kidney function. These findings support the growing evidence that atherogenic dyslipidemia plays an important role not only in cardiovascular morbidity but also in the progression of CKD. The observed negative correlation between the ApoB/ApoA-I ratio and baseline eGFR indicates that patients with poorer renal function tend to have a more atherogenic lipid profile. This observation is consistent with earlier studies reporting progressive lipid abnormalities with advancing CKD stages [14,15]. Reduced activity of lipoprotein lipase, impaired HDL maturation, and increased oxidative modification of lipoproteins in CKD may explain the elevation of ApoB-containing particles and reduction in ApoA-I levels [16]. Our findings align with previous studies that highlighted the superiority of apolipoprotein-based indices over traditional lipid parameters in predicting adverse outcomes. The ApoB/ApoA-I ratio reflects the balance between atherogenic and anti-atherogenic lipoproteins and is a strong predictor of cardiovascular events and mortality [17,18]. Emerging data suggest that similar mechanisms may contribute to renal damage. Lipid accumulation in mesangial and tubular epithelial cells promotes inflammation, fibrosis, and glomerulosclerosis, thereby accelerating renal function decline [19]. The significantly higher proportion of renal deterioration among patients with an ApoB/ApoA-I ratio ≥0.90 in the present study underscores the potential clinical utility of this marker for risk stratification. Similar associations have been reported in cohort studies from Japan and Europe, where higher ApoB/ApoA-I ratios were linked to faster eGFR decline and increased incidence of end-stage kidney disease [20,21]. However, evidence from South Asian populations has been limited. Given ethnic differences in lipid metabolism and cardiovascular risk profiles, our findings provide important region-specific data. Multivariate analysis confirmed that a high ApoB/ApoA-I ratio was an independent predictor of renal function decline even after adjustment for age, sex, diabetes mellitus, hypertension, and baseline eGFR. Diabetes mellitus and lower baseline eGFR were also significant predictors, which is consistent with established literature identifying these factors as key determinants of CKD progression [22]. The lack of independent association of age and sex in the adjusted model suggests that metabolic factors may have a stronger influence on short-term renal outcomes in this cohort. The pathophysiological link between dyslipidemia and CKD progression is multifactorial. Atherogenic lipoproteins can induce endothelial dysfunction, reduce

nitric oxide bioavailability, and activate pro-inflammatory cytokines, leading to microvascular injury within the kidney [23]. In contrast, ApoA-I and functional HDL exert anti-inflammatory and antioxidant effects that may protect against renal injury. Therefore, an elevated ApoB/ApoA-I ratio may reflect an imbalance that favors renal damage. From a clinical perspective, measuring the ApoB/ApoA-I ratio is relatively simple and may offer incremental prognostic value over conventional lipid profiles, particularly in CKD patients whose standard lipid levels may appear deceptively normal [24]. Early identification of high-risk individuals could enable intensified lifestyle modifications and pharmacological interventions aimed at optimizing lipids and protecting the kidneys. Nevertheless, this study has some limitations. The sample size was modest, and the follow-up duration was relatively short, which may limit the generalizability of the findings. Additionally, inflammatory markers and direct measures of HDL functionality were not assessed. Despite these limitations, the prospective design and adjustment for major confounders strengthen the validity of the results. In conclusion, the present study provides evidence that an elevated ApoB/ApoA-I ratio is independently associated with renal function decline in CKD patients. Incorporating apolipoprotein-based markers into routine assessments may improve risk stratification and guide preventive strategies in chronic kidney disease, particularly in resource-limited settings such as Bangladesh [25].

LIMITATIONS

This study was limited by its relatively small sample size and single-center design, which may restrict generalizability. The follow-up duration was short, and inflammatory biomarkers and HDL functionality were not assessed, which may have limited the mechanistic interpretation of the findings.

CONCLUSION

This prospective cohort study demonstrates that an elevated serum apolipoprotein B/apolipoprotein A-I ratio is independently associated with accelerated renal function decline in patients with chronic kidney disease. The ApoB/ApoA-I ratio may serve as a simple and effective biomarker for early risk stratification. Incorporating apolipoprotein-based assessment into routine clinical evaluation could support timely preventive and therapeutic strategies in CKD management.

RECOMMENDATION

Routine assessment of the ApoB/ApoA-I ratio should be considered in patients with chronic kidney disease for early risk stratification. Larger, multicenter studies with longer follow-up and interventional evaluation of lipid-modifying strategies are recommended to confirm clinical utility.

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