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Original Research Article

Association of Dyslipidemia with Renal Risk Factors eGFR, Proteinuria, Anemia, C-reactive Protein

Dr. Maleka Ali[1*], Dr. Shah Newaz Dewan[2], Dr. Samawat Naiyer Shahid[3], Dr. S.M Mahmud Hasan[4], Dr. Mohammad Tariqul Islam[5]

[1]Medical Officer, Department of Nephrology, National Institute of Kidney Diseases and Urology, Dhaka, Bangladesh
[2]Senior Consultant & Unit Head, Department of Nephrology, National Institute of Kidney Diseases and Urology, Dhaka, Bangladesh
[3]Associate Professor (c.c), Department of Nephrology, Bikrampur Bhuiyan Medical College, Bikrampur, Bangladesh
[4]Dialysis Medical Officer and Medical Officer Nephrology, Department of Nephrology, Rajshahi Medical College Hospital, Rajshahi, Bangladesh
[5]Medical Officer, Department of Nephrology, Sir Salimullah Medical College and Mitford Hospital, Dhaka, Bangladesh

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***Corresponding Author:** Dr. Maleka Ali
Medical Officer, Department of Nephrology, National Institute of Kidney Diseases and Urology, Dhaka, Bangladesh

Abstract

Background: Dyslipidemia, characterized by abnormal lipid profiles, is a significant risk factor for cardiovascular disease and has been increasingly implicated in the progression of chronic kidney disease (CKD). Dyslipidemia contributes to endothelial dysfunction, oxidative stress, and inflammation, potentially exacerbating renal impairment. However, the association between dyslipidemia and key renal risk factors—estimated glomerular filtration rate (eGFR), proteinuria, anemia, and C-reactive protein (CRP)—in renal transplant recipients remains inadequately explored. ***Objective:*** To assess the association between dyslipidemia and renal risk factors, including eGFR, proteinuria, anemia, and CRP, in renal transplant recipients. ***Methodology:*** A cross-sectional observational study was conducted at Sir Salimullah Medical College & Mitford Hospital, the CKD and Urology Hospital (CKD&U), and the Kidney Foundation Hospital and Research Institute, Bangladesh, over 13 months (May 2019–June 2020). A total of 105 renal transplant recipients were included through purposive sampling. Demographic, clinical, and laboratory data were collected, including fasting lipid profiles, serum creatinine, fasting blood glucose, urinary albumin-to-creatinine ratio (ACR), CRP, and eGFR (calculated using the MDRD equation). Statistical analysis was performed using SPSS v16, applying Chi-square tests to evaluate associations between dyslipidemia and renal risk factors. ***Results:*** Among renal transplant recipients, 61.9% had elevated triglycerides, 53.3% had high LDL, 33.3% had elevated total cholesterol, and 61.0% had low HDL. The mean triglyceride level was 214.38 ± 128.33 mg/dL, and the mean LDL was 100.41 ± 36.31 mg/dL. Dyslipidemia was significantly associated with reduced eGFR (p=0.04), indicating a decline in renal graft function. Lower ApoA1 levels were significantly linked to elevated CRP (p=0.01) and reduced eGFR (p=0.043), while higher ApoB levels were also associated with reduced eGFR (p=0.038). Hypertriglyceridemia was significantly correlated with anemia (p=0.05). However, no significant associations were observed between lipid markers and proteinuria, hypertension, or diabetes. ***Conclusion:*** Dyslipidemia, particularly elevated triglycerides and low HDL levels, is significantly associated with reduced renal function and systemic inflammation in renal transplant recipients. Specific lipid markers, such as ApoA1 and ApoB, may play a critical role in predicting renal dysfunction and inflammatory status. Early detection and management of dyslipidemia may be crucial in preserving renal function and improving long-term transplant outcomes.

Keywords: Dyslipidemia, Renal Transplant Recipients, eGFR, Proteinuria, Anemia, C-Reactive Protein.

INTRODUCTION

Dyslipidemia, characterized by abnormal levels of lipids in the blood, is a well-established risk factor for cardiovascular disease and is increasingly recognized for its role in the progression of chronic kidney disease (CKD). Lipid abnormalities, including elevated total cholesterol, low-density lipoprotein

cholesterol (LDL-C), triglycerides, and reduced high-density lipoprotein cholesterol (HDL-C), contribute to endothelial dysfunction, oxidative stress, and inflammation, all of which accelerate renal damage. Understanding the association between dyslipidemia and key renal risk factors, such as estimated glomerular filtration rate (eGFR), proteinuria, anemia, and C-reactive protein (CRP), is essential for optimizing renal and cardiovascular health in affected individuals [1-5].

eGFR, a critical marker of kidney function, declines progressively in CKD patients, often in conjunction with worsening dyslipidemia. Studies suggest that lipid abnormalities contribute to glomerular injury and tubulointerstitial fibrosis, leading to reduced eGFR over time [6,7]. The interplay between dyslipidemia and declining renal function creates a vicious cycle, where lipid-induced renal injury further exacerbates metabolic disturbances, accelerating CKD progression.

Proteinuria, another key indicator of kidney damage, has been strongly linked to dyslipidemia. Elevated lipid levels, particularly oxidized LDL-C, contribute to podocyte dysfunction and increased glomerular permeability, leading to protein leakage in the urine. Conversely, proteinuria itself promotes dyslipidemia by increasing hepatic lipid synthesis and impairing lipid clearance. This bidirectional relationship suggests that lipid abnormalities may not only be a consequence but also a contributing factor to proteinuria and renal dysfunction [8,9].

Anemia, a common complication of CKD, has also been associated with dyslipidemia. Alterations in lipid metabolism have been linked to reduced erythropoiesis and increased erythrocyte fragility, contributing to the development and progression of anemia in CKD patients. Additionally, chronic inflammation and oxidative stress—both exacerbated by dyslipidemia—can impair iron metabolism and erythropoietin production, further worsening anemia.

CRP, a marker of systemic inflammation, is frequently elevated in patients with both dyslipidemia and CKD. Dyslipidemia, particularly high LDL-C and triglyceride levels, promotes a pro-inflammatory state, leading to endothelial dysfunction and increased CRP levels [10-11]. Chronic inflammation, in turn, contributes to vascular damage, renal fibrosis, and accelerated CKD progression. The strong association between dyslipidemia and elevated CRP highlights the role of lipid-induced inflammation in worsening renal outcomes.

Objective

To assess the association of dyslipidemia with renal risk factors eGFR, proteinuria, anaemia, C-reactive protein.

METHODOLOGY

Type of Study

This study was designed as an observational cross-sectional study to evaluate the prevalence of renal transplant recipients in selected healthcare facilities in Bangladesh.

Place of Study

The research was conducted in the Department of Nephrology at Sir Salimullah Medical College & Mitford Hospital, Dhaka, as well as at the CKD and Urology Hospital (CKD&U) and the Kidney Foundation Hospital and Research Institute, Bangladesh.

Study Period

The study was carried out over a period of 13 months, from May 2019 to June 2020.

Study Population

The target population consisted of renal transplant recipients who attended the Department of Nephrology at CKD&U and the Kidney Foundation Hospital and Research Institute, Bangladesh.

Sample Size

Sample size was 368. However, due to the limited availability of renal transplant recipients and time constraints, a total of 105 patients were included in the study.

Sampling Technique

A purposive sampling method was employed. After selecting eligible participants, detailed clinical histories and medical records were collected. A structured data collection sheet was used to document relevant information, and necessary laboratory investigations were performed.

Selection Criteria

Inclusion Criteria:

- Patients aged 18 years or older
- Minimum 3 months post-renal transplantation

Exclusion Criteria:

- Patients taking lipid-lowering drugs
- Acute graft rejection within <3 months
- Patients with cognitive impairment
- Terminally ill patients

Study Procedure

Renal transplant recipients attending CKD&U and the Kidney Foundation Hospital were identified from hospital registries. Participants were contacted and scheduled for appointments. After obtaining informed consent, patients were instructed to fast for 10 hours before arriving for clinical assessment.

Each participant underwent a comprehensive medical history review, clinical examination, and laboratory investigations. The following parameters were assessed: fasting lipid profile, serum creatinine, fasting blood glucose, spot urine ACR, CRP, and eGFR (calculated using the MDRD equation). Additionally, serum Apo B, Apo A-I, and lipoprotein (a) levels were measured. The Framingham risk score was also calculated for each patient.

Data Collection Tools

A semi-structured questionnaire was designed to capture socio-demographic details, clinical information, and laboratory findings. The questionnaire was developed based on the study objectives and included structured questions for ease of data collection.

Sampling and Laboratory Analysis

Blood Sample Collection:

A 10 mL venous blood sample was collected after an overnight fast of at least 10 hours.

- 2 mL of whole blood was separated for fasting blood glucose (FBS).
- The remaining blood was allowed to clot, centrifuged at 3000 rpm for 15 minutes, and stored at -80°C until analysis.
- Urine samples were also collected and centrifuged for biochemical examination.

Laboratory Methods:

- Serum creatinine was measured using the immunoturbidimetric method with an automated analyzer (ERVA-XL-200).
- Urinary ACR was determined using a spot urine sample, calculating albumin-to-creatinine ratio.
- FBS was analyzed using an automated biochemistry analyzer.
- Total cholesterol, triglycerides, HDL-C, and LDL-C were measured using enzymatic methods.
- Apo A-I and Apo B were assessed via immunoturbidimetry using an automated chemistry analyzer (Mindray-BS-230).
- Lipoprotein (a) levels were determined using the immunoturbidimetric method.
- Serum CRP was measured using the turbidimetric latex agglutination method (Biosystems, Spain).

Data Management

All data were meticulously compiled, screened, and checked for completeness and consistency. Missing data and discrepancies were identified and corrected before statistical analysis.

Statistical Analysis

Data were systematically recorded using a pre-designed data collection sheet. Statistical analysis was performed using SPSS version 16 (Chicago, IL, USA). Quantitative data were presented as mean ± standard deviation (SD), while qualitative data were expressed as frequency and percentage. Appropriate statistical techniques were applied to analyze the results.

RESULTS

Most of the renal transplant recipients were below or equal to 40 years of age(72.4%). Mean age of the RTRs was 34.7 ± 8.9 years. Males were predominant than females. Male to female ratio was 7.75:1.

Table I: Age and gender distribution of the study group

	Frequency (n)	Percentage (%)
Age (years)		
≤30	40	38.1
31 – 40	36	34.3
41 – 50	23	21.9
>50	6	5.7
Gender		
Male	93	88.6
Female	12	11.4

Table demonstrates the mean values of traditional and non traditional lipid markers along with standard deviations in renal transplant recipients. The mean TG was 214.38 ± 128.33mg/dl, TC 179.73 ± 54.73mg/dl, LDL 100.41 ± 36.31mg/dl, HDL 36.40 ± 12.29mg/dl, Apo A-I 129 ± 36.1 mg/dl, Apo B 89 ± 29.3 mg/dl, lipoprotein(a) 17.69 ± 15.80 mg/dl

Table II: Traditional and non traditional lipid markers of the Renal transplant recipients (N=105)

Variables	Mean ± SD	Min - max
TG(mg/dl)	214.38 ± 128.33	48.20 - 655.00
Cholesterol(mg/dl)	179.73 ± 54.73	50.70 - 353.40
LDL(mg/dl)	100.41 ± 36.31	11.30 - 195.80

Variables	Mean ± SD	Min - max
HDL(mg/dl)	36.40 ± 12.29	9.60 - 79.40
Apo A-I(mg/dl)	129 ± 36.1	34 - 247
Apo B(mg/dl)	89 ± 29.3	22 - 196
Lipoprotein (a) (mg/dl)	17.69 ± 15.80	0.00 - 89.65

Table III. Frequency distribution of traditional lipid markers in Renal transplant recipients (N=105)

Variables	Frequency (n=105)	Percentage (%)
Total Cholesterol (mg/dl)		
<200	70	66.7
≥200	35	33.3
Triglycerides(mg/dl)		
<150	40	38.1
≥150	65	61.9
Low Density Lipoprotein		
<100(mg/dl)	49	46.7
≥100(mg/dl)	56	53.3
High Density Lipoprotein		
<40(mg/dl)	64	61.0
≥40(mg/dl)	41	39.0

Among renal transplant recipients, 61.9% had elevated TG, 53.3% had elevated LDL 33.3% had elevated TC, and 61.0% had below normal HDL. Among renal transplant recipients 42.9% had below normal ApoA1, 9.5% had elevated ApoB and 16.2% had elevated Lipoprotein(a).

Table IV: Frequency distribution of non-traditional lipid markers in Renal transplant recipients (N=105)

Variables	Frequency (n=105)	Percentage (%)
ApoA1(mg/dl)		
Male>120, Female>140	60	57.1
Male<120, Female<140	45	42.9
Apo B		
<130 mg/dl	95	90.5
≥130 mg/dl	10	9.5
Lipoprotein(a)		
≤30 mg/dl	87	82.9
>30 mg/dl	17	16.2

Table demonstrates different levels of eGFR were significantly reduced in dyslipidaemic patients following renal transplantation (p= 0.04) and thus suggests a significant association between dyslipidaemia and level of renal graft function. Chi-Square test was done to measure the level of significance.

Table V. Association of dyslipidemia with renal risk factors (N=105)

	Dyslipidemia Status		
Variables	**Dyslipidemia**	**Non-dyslipidemia**	**p-value**
eGFR(ml/min/1.73m^2)			.04
1T(>/90)	66.7%	33.3%	
2T(60-89)	94.1%	5.9%	
3T(30-59)	75.0%	25.0%	
4T(15-29)	100.0%	0	
5T(<15)	100.0%	0	
Anemia(g/dL)			0.09
Yes (Male<13, Female<12)	95.8%	4.2%	
No	82.7%	17.3%	
CRP(mg/L)			.152
<10	82.9%	17.1%	
>10	93.1%	6.9%	
ACR(mg/g)			.425
	82.6%	17.4%	

<30 ≥30	86.6%	13.4%	

The association between traditional and non-traditional lipid markers with various renal risk factors in renal transplant recipients (N=105) reveals key findings. Elevated triglycerides and low HDL levels show significant associations with obesity (p=0.04 and p=0.05, respectively). Lower ApoA1 levels are significantly linked to elevated CRP (p=0.01) and reduced eGFR (p=0.043), while higher ApoB levels are also associated with reduced eGFR (p=0.038). Additionally, hypertriglyceridemia is significantly associated with anemia (p=0.05). However, no significant associations were found between lipid markers and proteinuria, hypertension, or diabetes. These findings suggest that specific lipid markers, particularly ApoA1, ApoB, triglycerides, and HDL, may influence renal function, inflammation, and metabolic risk factors in renal transplant recipients.

Table VI: Association between traditional and non traditional lipid markers and renal risk factors (N=105)

Lipid Markers (mg/dL)	**CRP > 10 mg/L (%) (p-value)**	**Anemia (%) (p-value)**	**Reduced eGFR (%) (p-value)**	**Proteinuria (%) (p-value)**	**Hypertension (%) (p-value)**	**Diabetes (%) (p-value)**	**Obesity (%) (p-value)**
Total Cholesterol (<200 vs ≥200)	32.9% vs 17.1% (0.06)	27.1% vs 14.3% (0.11)	5.7% vs 5.7% (0.051)	20.0% vs 25.7% (0.334)	22.9% vs 25.7% (0.462)	5.7% vs 2.9% (0.458)	1.4% vs 2.9% (0.889)
Triglycerides (<150 vs ≥150)	27.5% vs 27.7% (0.583)	32.5% vs 16.9% (0.05)	10.0% vs 3.1% (0.118)	17.5% vs 24.6% (0.273)	27.5% vs 21.5% (0.320)	10.0% vs 1.5% (0.068)	0% vs 3.1% (0.04) (Significant)
LDL (<100 vs ≥100)	30.6% vs 25.0% (0.34)	28.6% vs 17.9% (0.142)	6.1% vs 5.4% (0.185)	20.4% vs 23.2% (0.457)	28.6% vs 19.6% (0.200)	4.1% vs 5.4% (0.564)	0% vs 3.6% (0.579)
HDL (<40 vs ≥40)	26.6% vs 29.3% (0.466)	23.4% vs 22.0% (0.50)	3.1% vs 9.8% (0.666)	20.3% vs 24.4% (0.397)	26.6% vs 19.5% (0.279)	6.3% vs 2.4% (0.349)	3.1% vs 0% (0.05) (Significant)
ApoA1 (Male >120, Female >140 vs Lower)	18.3% vs 40.0% (0.01) (Significant)	23.3% vs 22.2% (0.542)	3.3% vs 8.9% (0.043) (Significant)	23.3% vs 20.0% (0.435)	25.0% vs 22.2% (0.463)	5.0% vs 4.4% (0.635)	1.7% vs 2.2% (0.368)
ApoB (<130 vs ≥130)	27.4% vs 30.0% (0.558)	24.2% vs 10.0% (0.282)	6.3% vs 0% (0.038) (Significant)	20.0% vs 40.0% (0.146)	23.2% vs 30.0% (0.441)	5.3% vs 0% (0.600)	2.1% vs 0% (0.894)
Lipoprotein(a) (≤30 vs >30)	27.6% vs 29.4% (0.544)	21.8% vs 29.4% (0.346)	5.7% vs 5.9% (0.236)	20.7% vs 23.5% (0.507)	23.0% vs 29.4% (0.386)	3.4% vs 11.8% (0.187)	1.1% vs 5.9% (0.319)
Lipid Markers (mg/dL)	CRP > 10 mg/L (%) (p-value)	Anemia (%) (p-value)	Reduced eGFR (%) (p-value)	Proteinuria (%) (p-value)	Hypertension (%) (p-value)	Diabetes (%) (p-value)	Obesity (%) (p-value)
Total Cholesterol (<200 vs ≥200)	32.9% vs 17.1% (0.06)	27.1% vs 14.3% (0.11)	5.7% vs 5.7% (0.051)	20.0% vs 25.7% (0.334)	22.9% vs 25.7% (0.462)	5.7% vs 2.9% (0.458)	1.4% vs 2.9% (0.889)
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ApoB (<130	27.4% vs	24.2% vs	6.3% vs 0%	20.0% vs	23.2% vs	5.3% vs	2.1% vs 0%

vs ≥130)	30.0% (0.558)	10.0% (0.282)	(0.038) (Significant)	40.0% (0.146)	30.0% (0.441)	0% (0.600)	(0.894)
Lipoprotein(a) (≤30 vs >30)	27.6% vs 29.4% (0.544)	21.8% vs 29.4% (0.346)	5.7% vs 5.9% (0.236)	20.7% vs 23.5% (0.507)	23.0% vs 29.4% (0.386)	3.4% vs 11.8% (0.187)	1.1% vs 5.9% (0.319)

DISCUSSION

Our study investigated the association between traditional and non-traditional lipid markers with renal risk factors in renal transplant recipients (RTRs), revealing significant findings. The mean age of RTRs in our study was 34.7 years, with a male predominance (88.6%), aligning with previous studies where younger male patients constituted the majority of renal transplant recipients [12]. Similar studies have also reported male predominance, attributing it to gender-related disparities in chronic kidney disease (CKD) progression and access to renal transplantation [13]. However, compared to international studies, where the mean age of RTRs is often higher, our study highlights a relatively younger transplant recipient population. This difference may be attributed to the demographic and healthcare system variations in different regions [14].

Regarding lipid profiles, our study found that 61.9% of RTRs had hypertriglyceridemia, 53.3% had elevated LDL, 33.3% had high total cholesterol, and 61.0% had low HDL levels. These findings are consistent with previous research, which has documented dyslipidemia as a common metabolic complication following renal transplantation. Elevated triglycerides and low HDL levels were significantly associated with obesity in our study (p=0.04 and p=0.05, respectively), corroborating findings from earlier studies that identified dyslipidemia as a key contributor to post-transplant metabolic syndrome [15]. Additionally, our study demonstrated that hypertriglyceridemia was significantly associated with anemia (p=0.05), a relationship that has been previously reported in CKD and post-transplant populations due to altered lipid metabolism affecting erythropoiesis.

Non-traditional lipid markers, particularly ApoA1 and ApoB, also showed significant associations with renal risk factors. Lower ApoA1 levels were significantly linked to elevated CRP (p=0.01) and reduced eGFR (p=0.043), while higher ApoB levels were associated with reduced eGFR (p=0.038). These findings align with prior research emphasizing the role of ApoA1 in inflammation regulation and cardiovascular risk reduction in RTRs. Similarly, studies have suggested that elevated ApoB levels are predictive of graft dysfunction and cardiovascular complications in transplant recipients [16].

Dyslipidemia was significantly associated with reduced eGFR in our study (p=0.04), emphasizing its impact on graft function. Several studies have previously reported that post-transplant dyslipidemia accelerates graft deterioration and contributes to long-term complications [17]. The mechanism is believed to involve lipid-induced endothelial dysfunction and inflammatory responses, leading to progressive renal impairment. Although our study did not find significant associations between lipid markers and hypertension or diabetes, previous studies have suggested that prolonged exposure to dyslipidemia may increase the risk of these comorbidities in transplant recipients.

CONCLUSION

Overall, our findings reinforce the growing body of evidence highlighting the critical role of dyslipidemia, particularly elevated triglycerides, low HDL, and abnormal ApoA1 and ApoB levels, in influencing renal risk factors in RTRs. The significant associations observed in our study suggest the need for early monitoring and targeted lipid-lowering interventions to improve post-transplant outcomes. Future studies with larger sample sizes and longer follow-up periods are warranted to further elucidate these associations and develop personalized treatment strategies for RTRs.

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