DYSFUNCTIONAL HDL- A DEVIL IN DISGUISE AS A PREDICTOR OF PROGRESSION OF CHRONIC KIDNEY DISEASE TO PROVIDE AN ALTERNATIVE TO URINARY ALBUMIN CREATININE RATIO [UACR] MEASUREMENT

Dr. Md Rashid Ahsan Lodhi,[1] Dr.Sayantaann Saha,[2] Dr. C. R. Mallikarjuna,[3]

Dr. Asif Ayaz[4*]

[1]MBBS, MD, Associate Professor, Department Of Biochemistry, Gouri Devi Institute of Medical Sciences and Hospital, Durgapur, West Bengal, Email id: nufayl.lodhi@gmail.com

[2]MBBS, MD, Assistant Professor, Department Of Biochemistry, Gouri Devi Institute of Medical Sciences and Hospital, Durgapur, West Bengal, Email id: sayantaann@gmail.com

[3]MBBS, MD, Professor, Department Of Biochemistry, S.S. Institute of Medical Sciences and Research Centre, Davangere, Karnataka, Email id: crmsaiila@gmail.com

Address for Correspondence

[4*]MBBS, MS,Assistant Professor, Department Of Surgery, Nil Ratan Sircar Medical College And Hospital, Kolkata, West Bengal, Email id: drasifauzia@gmail.com

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ABSTRACT

Background: In subjects with chronic kidney disease, the levels of High-density lipoprotein cholesterol (HDL-C) are usually decreased. However, enough data assessing this relationship is scarce in different literature commenting on kidney disease progression in patients having chronic kidney disease

Aim: The present study was conducted to assess the correlation between the progression of chronic kidney disease and serum high-density lipoprotein cholesterol levels to provide an alternative to urinary albumin-creatinine ratio (UACR) measurement.

Methods: The present study assessed the relationship between the progression of chronic kidney disease and serum high-density lipoprotein cholesterol levels in 542 subjects with diagnosed chronic kidney disease. The primary and secondary outcomes assessed were a 50% reduction in the estimated GFR (Glomerular Filtration Rate) from the baseline values or the onset of end-stage renal disease (ESRD). The secondary outcomes assessed were the development of end-stage renal disease.

Results: On assessing the clinical outcomes in the study subjects based on eGFR categories and etiology of chronic kidney diseases, overall outcomes were renal in 15.31% (n=83) subjects, was >50% reduction in eGFR in 8.30% (n=45) subjects, and end-stage renal disease in 12.54% (n=68) study subjects. Highest renal outcome was seen in eGFR category of <30-≥15 mL/min per 1.73 m^2,highest >50% reduction in eGFR was seen in category of <30-≥15 mL/min per 1.73 m^2 35.5% (n=16) subjects and least in ≥90 mL/min per 1.73 m2 group with 4.44% (n=2) subjects, and end-stage renal disease was also highest in eGFR category of <30-≥15 mL/min per 1.73 m^2 with 41.17% (n=28). For the association of HDL-C and renal outcome in the study subjects, it was seen that for HDL-C serum levels of <30, the event was seen in 9.63% (n=8) subjects which were statistically significant with p=0.003. In ≥60 serum

HDL-C levels, there were 21% (n=114) subjects where the event was seen in 14.45% (n=12) study subjects. This was statistically significant with p=0.001

Conclusion: The present study found an established correlation between the levels of serum HDL-C and adverse outcomes in kidney damage in subjects with chronic kidney disease. The present study concludes that both high, as well as low serum HDL-C levels, are harmful in subjects with non-dialysis chronic kidney disease and thus HDL-C may be considered as an alternative to urinary albumin-creatinine ratio (UACR) measurement

Keywords: Chronic kidney disease, eGFR, ESRD, high-density lipoprotein [HDL],kidney disease,urinary albumin-creatinine ratio (UACR) measurement.

INTRODUCTION

Serum HDL (High-density lipoprotein) is generally considered and suggested to be a protective factor in subjects with cardiovascular diseases owing to its antiatherogenic effect which is a process involving multiple organs and removes excess cholesterol from peripheral tissues and lipid-laden macrophages by the process known as reverse cholesterol transport. Additively, various mechanisms are associated with the protective effects of HDL against atherosclerosis including antithrombotic effects, antioxidative effects, and anti-inflammatory effects. Various previous literature data shows a possible role of apolipoproteinJ, apolipoprotein A-IV, and apolipoprotein E in these effects. However, the exact mechanism behind this is largely unknown. Previous literature data established a clear and inverse relationship between cardiovascular disease and HDL-C levels. However, recent data doubts the relevance of HDL-C as a surrogate marker for cardiovascular disease risk.[1]

Various studies showed that no clinical benefit was on increasing HDL-C for reduction of risk for cardiovascular diseases. Additively, no casual relation was seen between raised HDL-C levels genetically and myocardial infarction risk. Also, significant data shows that HDL composition assesses the functional properties of the circulating HDL-C levels. Collectively, these results question the reliability of high levels of HDL-C as a reliable biomarker for risk assessment and protective role for the adverse disease outcomes.[2]

Recently, chronic kidney disease (CKD) is an increasing health concern globally posing a high burden on the healthcare sector worldwide with increased cases of cardiovascular diseases and death due to these events. In subjects with chronic kidney disease, dyslipidemia is a common finding, which is commonly associated with high triglyceride levels and low HDL-C levels, and high levels of oxidized low-density lipoprotein cholesterol. Considerable evidence in the literature suggests that an increased risk of renal dysfunction is associated with low levels of HDL-C. However, Contradictory results are shown by previous literature data concerning the relationship between disease progression in subjects with chronic kidney disease, not undergoing dialysis, and levels of serum HDL-C.[3]

Also, these studies only assessed the levels of HDL-C at baseline, and not analyzed the changes in these values over time. Hence, there is uncertainty concerning the prognostic value of HDL-C in assessing the disease progression in subjects with CKD and not undergoing dialysis. Recently, a study with a large sample size reported that there exists a U shape relationship in incidence and progression of chronic kidney disease in non-dialysis subjects and serum levels of HDL-C, where the risk of adverse kidney outcomes was increased. HDL-C function and composition were also changed significantly in subjects with

chronic kidney disease leading to its transformation into toxic particles and loss of its protective properties.[4]

All these findings collectively led to a hypothesis formulation that both high and low levels of HDL-C negatively affect the renal function of subjects with CKD and non-dialysis. Demonstration of microalbuminuria usually assessed by urinary albumin-creatinine ratio (UACR) is currently the gold standard for detection and prediction of kidney damage However, its predictive power is limited whereby a decline in the renal function of patients is not always accompanied by micro albuminuria and in other cases micro albuminuria might be noted in absence of renal impairment. Hence, the present study was conducted to assess the correlation between the progression of chronic kidney disease and serum high-density lipoprotein cholesterol levels to provide an alternative to urinary albumin-creatinine ratio (UACR) measurement.

MATERIALS AND METHODS

The present study was conducted to assess the correlation between the progression of chronic kidney disease and serum high-density lipoprotein cholesterol levels. The study was conducted on the subjects visiting atDepartment of Medicine and performed in the Department of Biochemistry, Gouri Devi Institute of Medical Sciences and Hospital, Durgapur, West Bengalafter obtaining the clearance from the concerned Ethical committee. The study population was comprised of the subjects visiting the Department of Medicine of the said Institute. The study included a total of 542 subjects from both genders within the age range of 19 years to 76 years with a confirmed diagnosis of chronic kidney disease in stages 1-5 and not undergoing dialysis. After explaining the detailed study design, informed consent was taken from all the study subjects in both written and verbal form.

After the finalinclusion of the study subjects, the detailed history was recorded followed by demographics including age, gender, comorbid conditions, medical history, physical activity, alcohol intake, and smoking status. Alcohol intake was assessed as high, moderate, and none, whereas, smoking status was current, former, and never.Physical activity was assessed as moderate or vigorous. BMI was also calculated as kg/m^2. Fasting blood samples were collected under strict aseptic and sterile conditions for assessing creatinine levels and lipid profile which were assessed every 6 months for 1 year and then annually. Other parameters assessed were cholesterol (low-density lipoprotein cholesterol, HDL-C, and triglycerides), iron profiles (including total iron-binding capacity and serum ferritin), C-reactive protein, calcium, phosphorus, albumin, creatinine, blood urea nitrogen, fasting glucose, and complete blood count. CKD epidemiologic collaboration equation was used to assess eGFR levels. Urine samples were assessed for proteinuria and urinary albumin-creatinine ratio (mg/g).

HDL-C levels were used as variables and were divided into 5 categories of <30, 30 to <40, 40 to <50, 50 to <60, and ≥60mg/dl. The primary outcome for the study was the onset of end-stage renal disease (ESRD) in the recall phase or a 50% reduction of eGFR from the baseline values. ESRD was assessed as the starting of renal replacement therapy, including renal transplantation or dialysis. The development of end-stage renal disease was the secondary outcome of the study. Till the follow-up, the subjects were assessed till death, study completion, or visit.

The collected data were subjected to the statistical evaluation using SPSS software version 21 (Chicago, IL, USA) and one-way ANOVA and t-test for results formulation. The data were expressed in percentage and number, and mean and standard deviation. The level of significance was kept at $p<0.05$.

RESULTS

The present prospective clinical study was conducted to assess the correlation between the progression of chronic kidney disease and serum high-density lipoprotein cholesterol levels. The study included a total of 542 subjects from both genders within the age range of 19 years to 76 years with a confirmed diagnosis of chronic kidney disease in stages 1-5 and not undergoing dialysis. The demographic characteristics of the study subjects are listed in Table 1. The mean age of the subjects was 53.5±12.4 years. The highest age was in serum HDL-C level <30 groups with 57.4±10.6 years which decreased with increased HDL-C levels to 51.6±12.4 years in ≥60 HDL-C level which was statistically significant with $p<0.001$. The highest number of males were in the HDL-C level <30 groups with 80.4% males, whereas the least 38.7% in ≥60 HDL-C level group. For comorbidities, diabetes mellitus was also highest in serum HDL-C level <30 with 68.6% subjects and least in ≥60 HDL-C levels with 23.7%. A similar pattern was followed by hypertension, coronary artery disease, COPD, and cerebrovascular diseases with a significant difference and p-value of <0.001 except for COPD which was non-significant with $p=0.246$. The etiology of CKD was polycystic kidney disease, hypertension, diabetic nephropathy, and glomerulonephritis which all were highest in serum HDL-C level <30 level group and lowest in≥60 HDL-C levels with $p<0.001$. BMI showed a significant reduction with increased HDL-C levels with $p<0.001$. CRP, albumin, calcium, fasting glucose, and WBC levels reduced significantly with increased serum HDL-C levels ($p<0.001$), whereas fasting glucose showed a non-significant difference in different groups with $p=0.925$. For the UACR (urine albumin to creatinine ratio) assessment in the study subjects, it was seen that in the HDL-C level <30 group, UACR was 529.3mg/g which decreased with increase in HDL-C levels to 30-<40, 40-<50, 50-<60, and ≥60 where the respective values of UACR was found to be 424.7, 340.7, 315.3, and 308.2 respectively. This decrease in UACR values with increase in HDL-C levels was found to be statistically significant with $p=0.04$ (Table 1).

On assessing the clinical outcomes in the study subjects based on eGFR categories and etiology of chronic kidney diseases, overall outcomes were renal in 15.31% (n=83) subjects, was >50% reduction in eGFR in 8.30% (n=45) subjects, and end-stage renal disease in 12.54% (n=68) study subjects. Highest renal outcome was seen in eGFR category of <30-≥15 mL/min per 1.73 m^2 with 37.34% (n=31) subjects and least in 9.77% (n=53) subjects. Highest >50% reduction in eGFR was seen in category of mL/min per 1.73 m2 35.5% (n=16) subjects and least in ≥90 mL/min per 1.73 m2 group with 4.44% (n=2) subjects. End-stage renal disease was also highest in eGFR category of <30-≥15 mL/min per 1.73 m^2 with 41.17% (n=28) subjects and least in ≥90 mL/min per 1.73 m2 group with 1.47% (n=1) subject. For the etiology of chronic kidney disease, Polycystic kidney disease, hypertension, Diabetic nephropathy, and glomerulonephritis was seen in 15.68% (n=85), 0.18% (n=100), 23.43% (n=127), and 36.16^ (n=196) study subjects respectively as shown in Table 2.

For the association of HDL-C and renal outcome in the study subjects, it was seen that for HDL-C serum levels of <30, the event was seen in 9.63% (n=8) subjects which were statistically significant with p=0.003. For HDL-C value of 30-<40, there were 24% (n=130) subjects where event was seen in 19.27% (n=16) subjects. For HDL-C levels of 40-<50, there were 30% (n=162) subjects where event was seen in 15.66% (n=13) study subjects. In HDL-C levels of 50-<60, there were 21% (n=114) subjects where event was seen in 13.25% (n=11) subjects. These were statistically non-significant with p-values of 0.235 and 0.244 respectively. In ≥60 serum HDL-C levels, there were 21% (n=114) subjects where the event was seen in 14.45% (n=12) study subjects. This was statistically significant with p=0.001 as depicted in Table 3.

DISCUSSION

The present prospective clinical study was conducted to assess the correlation between the progression of chronic kidney disease and serum high-density lipoprotein cholesterol levels. The study included a total of 542 subjects from both genders within the age range of 19 years to 76 years with a confirmed diagnosis of chronic kidney disease in stages 1-5 and not undergoing dialysis. The mean age of the subjects was 53.5±12.4 years. The highest age was in serum HDL-C level <30 groups with 57.4±10.6 years which decreased with increased HDL-C levels to 51.6±12.4 years in ≥60 HDL-C level which was statistically significant with p<0.001. The highest number of males were in the HDL-C level <30 groups with 80.4% males, whereas the least 38.7% in ≥60 HDL-C level group. For comorbidities, diabetes mellitus was also highest in serum HDL-C level <30 with 68.6% subjects and least in ≥60 HDL-C levels with 23.7%. A similar pattern was followed by hypertension, coronary artery disease, COPD, and cerebrovascular diseases with a significant difference and p-value of <0.001 except for COPD which was non-significant with p=0.246. The etiology of CKD was polycystic kidney disease, hypertension, diabetic nephropathy, and glomerulonephritis which all were highest in serum HDL-C level <30 level group and lowest in ≥60 HDL-C levels with p<0.001. BMI showed a significant reduction with increased HDL-C levels with p<0.001. CRP, albumin, calcium, fasting glucose, and WBC levels reduced significantly with increased serum HDL-C levels (p<0.001), whereas fasting glucose showed a non-significant difference in different groups with p=0.925. These demographics were comparable to the studies of Kiuchi MG et al[5] in 2016 and ZewingerS et al[6] in 2016 where authors assessed the subjects with comparable demographics.

On estimating the UACR (urine albumin to creatinine ratio) in the study subjects, it was seen that in the HDL-C level <30 group, UACR was 529.3mg/g which decreased with increase in HDL-C levels to 30-<40, 40-<50, 50-<60, and ≥60 where the respective values of UACR was found to be 424.7, 340.7, 315.3, and 308.2 respectively. This decrease in UACR values with increase in HDL-C levels was found to be statistically significant with p=0.04. These results were similar to the studies of Sun K et al[7] in 2015 andEunheeJi and Yon Su Kim[8] et al in 2016 where authors also showed significant decrease in UACR was seen with increased HDL-C levels.

For the assessment of the clinical outcomes in the study subjects based on eGFR categories and etiology of chronic kidney diseases, overall outcomes were renal in 15.31% (n=83) subjects, was >50% reduction in eGFR in 8.30% (n=45) subjects, and end-stage renal disease in 12.54% (n=68) study subjects. Highest renal outcome was seen in eGFR category of <30-

≥15 mL/min per 1.73 m^2 with 37.34% (n=31) subjects and least in 9.77% (n=53) subjects. Highest >50% reduction in eGFR was seen in category of mL/min per 1.73 m2 35.5% (n=16) subjects and least in ≥90 mL/min per 1.73 m2 group with 4.44% (n=2) subjects. End-stage renal disease was also highest in eGFR category of <30-≥15 mL/min per 1.73 m^2 with 41.17% (n=28) subjects and least in ≥90 mL/min per 1.73 m2 group with 1.47% (n=1) subject. For the aetiology of chronic kidney disease, Polycystic kidney disease, hypertension, diabetic nephropathy, and glomerulonephritis was seen in 15.68% (n=85), 0.18% (n=100), 23.43% (n=127), and 36.16^ (n=196) study subjects respectively. These results were consistent with the results of the studies by Moradi H et al[9] in 2014 and Speer T et al[10] in 2015 where similar clinical outcomes were shown by the authors based on eGFR categories and etiology of chronic kidney diseases.

For the association of HDL-C and renal outcome in the study subjects, it was seen that for HDL-C serum levels of <30, the event was seen in 9.63% (n=8) subjects which were statistically significant with p=0.003. For HDL-C value of 30-<40, there were 24% (n=130) subjects where event was seen in 19.27% (n=16) subjects. For HDL-C levels of 40-<50, there were 30% (n=162) subjects where event was seen in 15.66% (n=13) study subjects. In HDL-C levels of 50-<60, there were 21% (n=114) subjects where event was seen in 13.25% (n=11) subjects. These were statistically non-significant with p-values of 0.235 and 0.244 respectively. In ≥60 serum HDL-C levels, there were 21% (n=114) subjects where the event was seen in 14.45% (n=12) study subjects. This was statistically significant with p=0.001. These results were in agreement with the findings of Yamamoto S et al[11] in 2012 and Ganda A et al[12] in 2013 where the comparable association of HDL-C and the renal outcome was shown by the authors as in the present study.

CONCLUSION

Within its limitations, the present study concluded that there was an established correlation between the levels of serum HDL-C and adverse outcomes in kidney disease progression in subjects with chronic kidney disease. The present study concludes that both high, as well as low serum HDL-C levels, are harmful in subjects with non-dialysis chronic kidney disease.Thus, determination of HDL-C levels accords significant predictive value in development and progression of kidney damage in patients with chronic kidney disease. Hence, this study evaluated that the HDL-C may be considered as an alternative to urinary albumin-creatinine ratio (UACR) measurement.However, for optimal prediction ofkidney damage and its assessment, both UACR and HDL-C should be concurrently determined during the routine health status check of CKD patients, to improve on predictive precision and efficiency.

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TABLES

Characteristics	**Total (n=542)**	**Serum HDL-C levels (mg/dl)**					**p-value**
		<30	**30-<40**	**40-<50**	**50-<60**	**≥60**	
Mean age (years)	53.5±12.4	57.4±10.6	55.5±11.4	54.3±12.3	52.8±12.7	51.6±12.4	**<0.001**
Male gender	61.4	80.4	77.2	67.4	55.3	38.7	**<0.001**
Comorbidities (%)							
Diabetes mellitus	33.7	68.6	41.3	37.2	24.8	23.7	**<0.001**
Hypertension	96.3	97.6	99.2	97.5	95.2	92.2	**<0.001**
Coronary artery disease	1.9	4.5	3.3	1.7	0.9	0.4	**<0.001**
COPD	0.8	1.3	0.6	1.3	0.4	0.4	0.246
Cerebrovascular diseases	6.2	6.7	7.5	6.2	7.2	3.7	**0.04**
Etiology of CKD (%)							
Polycystic kidney disease	16.3	5.6	1.2	11.2	22.9	25.7	**<0.001**
Hypertension	18.7	18.5	22.8	20.7	17.4	12.4	**<0.001**

Diabetic nephropathy	23.4	58.3	30.7	24.7	15.2	13.5	**<0.001**
Glomerulonephritis	36.4	16.3	30.7	36.5	40.3	41.7	**<0.001**
Others	6.2	2.3	5.6	6.9	5.2	7.7	0.235
BMI	24.4±3.6	25.6±2.6	25.6±3.4	25.2±3.5	24.6±3.7	23.4±3.6	**<0.001**
Physical activity							
<3 times/week	61.6	63.2	64.3	62.6	59.5	60.2	0.177
>3 times/week	38.4	37.2	35.7	37.4	40.9	40.2	0.177
Alcohol intake							
None	79.7	83.4	79.6	77.6	81.5	77.5	0.165
Moderate	8.5	7.7	9.6	9.6	8.5	7.5	0.683
High	11.6	9.1	10.7	12.6	9.7	15.2	**0.03**
Smoking status (%)							
Current	15.6	22.4	20.2	17.2	14.6	9.4	**<0.001**
Former	30.8	36.8	41.6	30.6	26.3	21.6	**<0.001**
Never	53.8	40.7	38.4	52.7	59.2	69.7	**<0.001**
Laboratory parameters							
CRP	0.7	0.8	0.8	0.6	0.4	0.2	**0.001**
Albumin	4.4±0.6	4.2±0.7	4.3±0.6	4.4±0.6	4.4±0.6	4.4±0.6	**0.01**
Calcium	9.3±0.7	8.8±0.5	9.2±0.7	9.4±0.7	9.4±0.7	9.4±0.8	**<0.001**
Phosphorus	3.9±0.9	3.6±0.6	3.5±0.5	3.5±0.5	3.4±0.4	3.5±0.5	0.925
Fasting glucose (mg/dl)	111.3±40.2	117.4±47.3	112.6±40.4	113.4±39.7	108.3±38.3	104.1±33.3	**<0.001**
$WBCX10^3$ µl	6.6±1.7	7.3±2.1	7.1±1.8	6.7±1.8	6.4±1.7	6.1±1.8	**<0.001**
UACR (Urine albumin to creatinine ratio mg/g	354.3	529.3	424.7	340.7	315.3	308.2	0.**044**

Table 1: Demographic characteristics of the study subjects

Outcomes	**Variables**	**Total**		**Renal outcomes**		**>50% reduction in ESR**		**ESRD**	
		%	**n=542**	**%**	**n=83**	**%**	**n=45**	**%**	**n=68**
eGFR categories at baseline, mL/min per 1.73 m2	≥90	12.73	69	3.61	3	4.44	2	1.47	1
	<90-≥60	21.58	117	6.02	5	6.66	3	2.94	2
	<60-≥45	18.26	99	16.86	14	24.4	11	8.82	6
	<45-≥30	16.97	92	12.04	10	20	9	16.17	11
	<30-≥15	20.66	112	37.34	31	35.5	16	41.17	28
	<15	9.77	53	24.09	20	8.88	4	29.41	20
Overall outcomes		100	542	15.31	83	8.30	45	12.54	68
CKD etiology	Polycystic kidney disease	15.68	85	13.25	11	17.7	8	13.23	9
	Hypertension	0.18	100	13.25	11	13.33	6	13.23	9
	Diabetic nephropathy	23.43	127	43.37	36	37.7	17	45.58	31
	Glomerulonephritis	36.16	196	26.50	22	26.6	12	25	17
	Others	6.27	34	3.61	3	4.44	2	2.94	2

Table 2: Clinical outcomes based on eGFR and etiology of CKD in the study subjects

HDL-C	**Subjects**		**Events**		**p-value**
	%	**n=542**	**%**	**n=83**	
<30	4	22	37.34	31	**0.003**
30-<40	24	130	19.27	16	0.235
40-<50	30	162	15.66	13	0.244
50-<60	21	114	13.25	11	-
≥60	21	114	14.45	12	**0.001**

Table 3: Association of HDL-C and renal outcome in the study subjects