Content available at: https://www.ipinnovative.com/open-access-journals

International Journal of Clinical Biochemistry and Research

Journal homepage: https://www.ijcbr.in/

Original Research Article

Lipoprotein abnormalities: A potential consequence of chronic kidney disease

Sandeep Singh[1], Umesh Kumar[2], Rajinderjit Singh Ahi[1]*, Basharat Azhar Paul[3]

[1]*Dept. of Biochemistry, Adesh Institute of Medical Sciences and Research, Bathinda, Punjab, India*
[2]*Dept. of Biochemistry, Pandit Bhagwat Dayal Sharma Post Graduate Institute of Medical Sciences, Rohtak, Haryana, India*
[3]*Dept. of Biochemistry, MM Institute of Medical Science and Research, Mullana, Haryana, India*

ARTICLE INFO

Article history:
Received 06-07-2024
Accepted 27-07-2024
Available online 21-08-2024

Keywords:
Chronic kidney disease
Glomerular filtration rate
Very-low density lipoprotein
High-density lipoprotein
Intermediate-density lipoprotein

ABSTRACT

Background: Chronic kidney disease (CKD) is marked by kidney damage or a glomerular filtration rate (GFR) of less than 60 mL/min/1.73 m^2 for at least three months, regardless of the underlying etiology. When a variety of renal problems are present, albuminuria defined as an albumin-to-creatinine ratio >30 mg/g in two out of three spot urine samples can serve to determine kidney failure.The estimated global rate of CKD is 13.4%. Nearly every aspect of biological life involves lipids. A few of these include acting as hormones or as precursors to hormones, providing energy, storing function and metabolic fuels, acting as functional and structural molecules in bio-membranes and forming insulation to aid in nerve transmission or prevent heat loss. The blood contains a variety of lipoproteins. They are chylomicrons, very-low density lipoprotein (VLDL), intermediate-density lipoprotein (IDL), low-density lipoprotein (LDL) and high-density lipoprotein (HDL), in descending sequence of increasing density. Less dense lipoproteins result from lower protein/lipid ratios. Although some lipoproteins carry cholesterol in its natural "free" alcohol form (the cholesterol-OH group facing the water surrounding the particles), all lipoproteins contain identical amounts of cholesterol. Other lipoproteins carry cholesterol in the form of fatty acyl esters, sometimes referred to as cholesterol esters. Triacylglycerol and cholesterol, which were not employed in the production of bile acids, are converted by the liver into VLDL molecules.

Aims: The aim of the study was to assess the Lipid levels in CKD and study the correlation between eGFR (which is a marker of severity of CKD) and lipid levels in CKD.

Materials and Methods: The present study was an observational study. The study was conducted over a period of six months on 180 patients. Blood samples were obtained in Becton Dickinson's commercially available red-capped tubes vacutainers (BD). After that, blood samples were left undisturbed at room temperature for 15-30 minutes to coagulate. For 5 minutes, the tubes were centrifuged at 3000 rpm. After centrifugation, the sample solution (serum) was transferred to a fresh polypropylene tube with a Pasteur pipette. Lipid profile and serum creatinine were done on fully automated SYSMEX BX-3010.

Results: Our results show that mean and standard deviation of serum cholesterol, serum triglycerides, HDL, VLDL and LDL with *p*-value between males and females in the different stages of CKD shows a statistically significant difference between stage II, III B, IV and stage V.

Conclusion: The present study highlights the progressive increase in serum cholesterol, serum triglycerides, VLDL, LDL levels as CKD advances through its stages. A progressive decline in HDL levels as CKD also advances through its stages. The correlations between eGFR and various biomarkers in the stages of CKD shed light on the complex interactions between renal function, lipid metabolism.

https://doi.org/10.18231/j.ijcbr.2024.018

1. Introduction

This global initiative's justification for tackling the issue is straightforward and obvious. Throughout the world, CKD is common. The harmful effects of CKD are well known, as are the underlying scientific and evidence-based methods for prevention, detection, evaluation, and therapy. Utilizing current knowledge and resources is crucial for improving chronic disease care and outcomes internationally, even though risk factors and care resources differ locally.[1]

CKD is marked by kidney damage or a glomerular filtration rate (GFR) of less than 60 mL/min/1.73 m^2 for at least three months, regardless of the underlying etiology. When a variety of renal problems are present, albuminuria, defined as an albumin-to-creatinine ratio >30 mg/g in two out of three spot urine samples, can serve to determine kidney failure.[2] The estimated global rate of CKD is 13.4%.[3] In India, one of the most widespread illnesses that is not transmissible. CKD has a significant morbidity, mortality rate, and financial impact.

The three most typical CKD causes are glomerulonephritis, diabetes mellitus and hypertension. One in three persons with diabetes and one in five adults with hypertension both have CKD.[4]The etiology of CKD also known as chronic kidney disease of undetermined etiology (CKDu), is uncertain in some cases.[5] Initially, there are usually no apparent symptoms, but later signs and symptoms could include confusion, exhaustion, nausea, and leg edema.[6]

Nearly every aspect of biological life involves lipids. A few of these include acting as hormones or as precursors to hormones, providing energy, storing function and metabolic fuels, acting as functional and structural molecules in bio-membranes and forming insulation to aid in nerve transmission or prevent heat loss.[7]Impaired kidney function can have an impact on a variety of tissues, cells and organs involved in lipid metabolism, including the liver, gut, plasma, macrophages, and vascular endothelium. Cholesterol is only marginally hydrophilic or soluble in water, as a single molecule. Additionally, bile acid, vitamin D, and steroid hormones are all biosynthesized using cholesterol as a precursor.[8]

The blood contains a variety of lipoproteins. They are chylomicrons, very-low density lipoprotein (VLDL), intermediate-density lipoprotein (IDL), low-density lipoprotein (LDL) and high-density lipoprotein (HDL), in descending sequence of increasing density. Less dense lipoproteins result from lower protein/lipid ratios. Although some lipoproteins carry cholesterol in its natural "free" alcohol form (the cholesterol-OH group facing the water surrounding the particles), all lipoproteins contain identical amounts of cholesterol. Other lipoproteins carry cholesterol in the form of fatty acyl esters, sometimes referred to as cholesterol esters.[9] Triacylglycerol and cholesterol, which were not employed in the production of bile acids, are converted by the liver into VLDL molecules.

An excessive level of lipids in the blood, such as triglycerides, cholesterol, and/or fat phospholipids, is known as dyslipidemia. A risk factor for the emergence of chronic renal disease is dyslipidemia.[10]Dyslipidemia develops in CKD patients as a result of altered postprandial lipoprotein and other triglyceride-rich lipoprotein metabolism, modifications to lipoprotein structure and reverse cholesterol transport.

So, the present study has been planned to study the severity of CKD with serum lipid levels.

2. Aims and Objectives

The aim of the study was to assess the Lipid levels in CKD and study the correlation between eGFR (which is a marker of severity of CKD) and lipid levels in CKD.

3. Materials and Methods

For lipid profile testing, all blood samples taken in plain vacutainer tubes were sent to the Biochemistry laboratory of the Biochemistry Department, AIMSR. The investigation lasted six months (October 2022 to March 2023) after receiving clearance from the AIMSR Research Committee and the Ethics Committee of Biomedical and Health Research, Adesh University, Bathinda.

3.1. Inclusion criteria

All the diagnosed cases of chronic kidney disease (all the patients were evaluated for chronic kidney disease as per the K/DOQI criteria by the National Kidney Foundation for diagnosis of CKD).[11]

3.2. Exclusion criteria

1. All HIV-positive individuals.
2. All the patients have a history of gout and /or hyperuricemia.
3. Patients who are taking anti-tubercular drugs and thiazide diuretics.

3.3. Methodologies and experimental strategy

3.3.1. Serum preparation

Blood samples were obtained in Becton Dickinson's commercially available red-capped tubes vacutainers (BD). After that, blood samples were left undisturbed at room temperature for 15-30 minutes to coagulate. For 5 minutes, the tubes were centrifuged at 3000 rpm. After centrifugation, the sample solution (serum) was transferred to a fresh polypropylene tube with a Pasteur pipette.

* *Corresponding author*.
E-mail address: umeshgothwal98@gmail.com (R. S. Ahi).

3.3.2. Specimen storage and handling during testing

During testing, the temperature of the specimens was kept between 20 and 25 °C. Specimens were kept at 4-8°C for a maximum of 8 days. If the samples required to be preserved for a longer amount of time, they were put in a deep freezer at -20°C.

3.3.3. Procedure

All the samples were collected from the patient attending IPD and OPD Department of Adesh Institute of Medical Science and Research, Bathinda. Serum creatinine, lipid profile (total cholesterol, triglycerides and HDL) was done on fully automated SYSMEX BX-3010.

3.3.4. Parameters

The following parameters were assessed in the following study:

Parameters	Methods	Biological reference
Serum creatinine	Jaffe's method	0.9-1.3 mg/dL (for males) 0.6-1.1 mg/dL (for females)
Serum cholesterol	CHOD-PAP Enzymatic photometric test	<200 mg/dL
Serum triglycerides	Glycerol-3-phosphateoxidase (GPO)	<200 mg/dL
HDL	Immuno FS	≥35 mg/dL
VLDL	TG/5	<40 mg/dL
LDL	Friedewald's formula	<130 mg/dL

3.3.5. Statistically analysis

The data analysis was done by using suitable software like MS EXCEL 2021. A t-test was used for enumeration data, which were expressed as mean and standard deviation. Pearson's analysis was employed to correlate the variables. Statistical method $p<0.05$ was considered to indicate a statistically significant.

4. Results

This study was conducted in Adesh Institute of Medical Sciences and Research, Bathinda. During a six-month period, 180 serum samples of CKD patients were tested for lipid profile. The five stages of CKD were determined by the estimated glomerular filtration rate (eGFR). The eGFR was calculated using the level of creatinine. In the present study, males and females were distributed in the stages based on the eGFR because of their different biological references of serum creatinine in males and females.

The study's findings are as follows.

Table 1: Age distribution amongst CKD patients

Age Group	No. of Patients	%
20-30	13	7.2
31-40	25	13.8
41-50	26	14.4
51-60	51	28.3
61-70	65	36.1
TOTAL	**180**	**100**
Range	20-70	
Mean ± SD	52.8 ± 12.8	

The above table shows the age distribution among 180 chronic kidney disease patients. The age of patients ranged from 20-70 years. The mean± SD for age was 52.8±12.8 years. Only 7.2% of patients were in the age group of 20-30 years., 13.8% of the patients were the age between 31-40 years, and 28.3% of the patients were the age between 51-60. The patients between 41 and 50 years of age were 14.4%. Maximum patients, that is, 36.1%, were in the age group between 61-70 years.

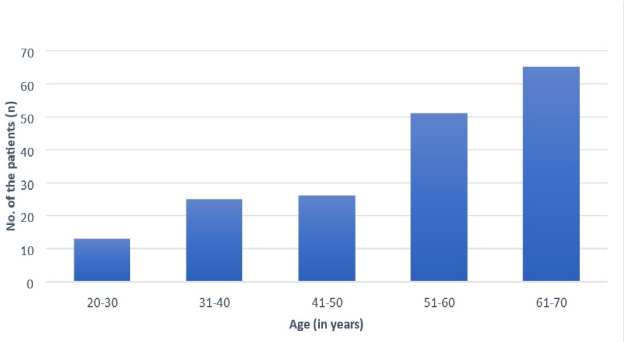

Figure 1: Age distribution in CKD patients

Table 2: Gender distribution amongst CKD patients

Gender	No. of Patients	Percentage
Male	126	67.7
Female	54	32.2
Total	**180**	**100**

As evident from the above table, there were 126 males (67.7%) and 54 females (32.2%) among total of 180 CKD patients. So, in the present study, the analysis according to the gender showed marked male preponderance.

Table 3: OPD/IPD wise distribution

OPD/IPD	No. of Patients	Percentage
OPD	32	17.7
IPD	148	82.2
TOTAL	**180**	**100**

IPD patients had a higher proportion of cases (82.2%) than IPD patients (17.7%).

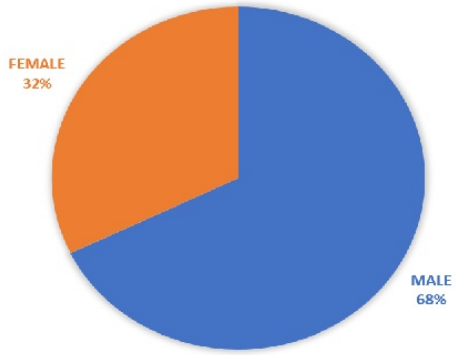

Figure 2: Gender distribution in CKD patients

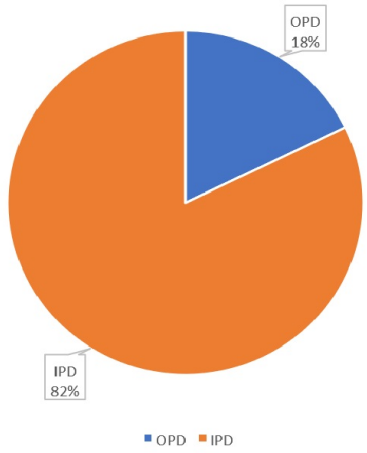

Figure 3: OPD/IPD wise distribution of CKD patients

Table 4: Patients distribution in the stages of CKD based on eGFR

Stages	eGFR value ml/ min/1.73 m^2	No. of patients	Male	Female
I	>90	10	7	3
II	60-89	21	13	8
III A	45-59	18	13	5
III B	30-44	27	20	7
IV	15-29	46	32	14
V	<15	58	41	17

Above table shows the distribution of patients in the stages of CKD. There were minimum number of male and female present in the stage I and maximum number of male and female present in the stage V.

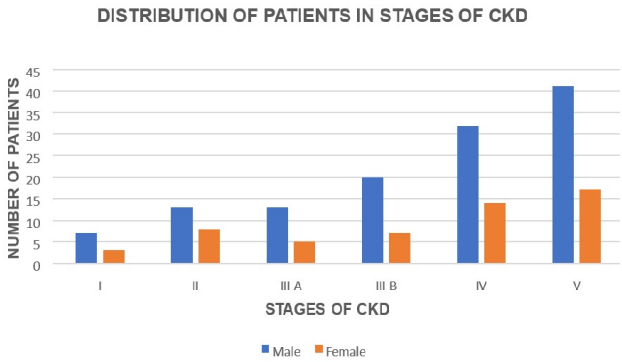

Figure 4: Patients distribution in the stages of CKD

Table 5: Distribution of serum cholesterol in the various stages of CKD and their mean and standard deviation with *p*-value

Stages	Mean ± Standard Deviation in males	Mean ± Standard Deviation in females	*p*-value
Stage I	176.30±25.45	172.8±9.44	=0.8276
Stage II	181.30±19.32	177.66±11.48	=0.6364
Stage III A	191.71±12.88	183.64±12.79	=0.2504
Stage III B	214.7±27.46	206.57±17.72	=0.4740
Stage IV	243.68±26.81	215.71±19.71	=0.0011*
Stage V	276.15±20.85	251.34±19.04	=0.0001*

*(p<0.05)=significant

Table 5 shows the mean and standard deviation of serum cholesterol with a *p*-value between males and females in the different stages of chronic kidney disease, which shows a statistically significant difference between stage IV and stage V (p=0.0011 and 0.0001, respectively).

Table 6: Distribution of serum triglycerides in the various stages of CKD and their mean and standard deviation with *p*-value

Stages	Mean ± Standard deviation in males	Mean ± Standard deviation in females	*p*-value
Stage I	169.71±11.27	165.83±7.65	=0.6063
Stage II	189.8±14.21	174.61±17.45	=0.0418*
Stage III A	202.76±22.74	191.87±13.65	=0.3355
Stage III B	234.6±24.14	227.14±19.65	=0.4698
Stage IV	249.25±22.15	229.14±16.00	=0.0038*
Stage V	261.26±28.56	257.93±26.89	=0.6827

*(p<0.05)=significant

Table 6 shows the mean and standard deviation of serum triglycerides with *p*-value between males and females in the different stages of CKD which shows a statistically significant difference between stage II and stage IV (p=0.0418 and 0.0038, respectively).

Table 7: Distribution of HDL in the various stages of CKD and their mean and standard deviation with *p*-value

Stages	Mean ± Standard deviation in males	Mean ± Standard deviation in females	*p*-value
Stage I	36.46±2.43	35.90±1.95	=0.7355
Stage II	35.78±2.29	34.6±1.67	=0.2227
Stage III A	35.5±1.93	33.57±1.71	=0.0685
Stage III B	35±2.30	32±2.64	=0.0084*
Stage IV	34.12±2.33	31.85±1.99	=0.0028*
Stage V	30.41±1.97	29.76±1.96	=0.2569

*(p<0.05)=significant

Table 7 shows the mean and standard deviation of HDL with p-value between males and females in the different stages of chronic kidney disease, which shows a statistically significant difference between stage III B and stage IV (p=0.0084 and 0.0028, respectively).

Table 8: Distribution of VLDL in the various stages of CKD and their mean and standard deviation with *p*-value

Stages	Mean ± Standard deviation in males	Mean ± Standard Deviation in females	*p*-value
Stage I	34.14±2.19	33.36±1.37	=0.5905
Stage II	36.8±2.28	35.61±4.43	=0.4242
Stage III A	38.37±2.73	37.50±3.59	=0.5853
Stage III B	48.32±3.97	45.82±3.20	=0.1466
Stage IV	49.85±4.43	46.91±2.47	=0.0250*
Stage V	52.90±5.07	52.83±5.47	=0.9629

*(p<0.05)=significant

Table 8 shows the mean and standard deviation of VLDL with *p*-value between males and females in the different stages of CKD, which shows a statistically significant difference in stage IV (p=0.0250).

Table 9 shows the mean and standard deviation of LDL with *p*-value between males and females in the different stages of CKD, which shows a statistically significant difference in stage IV (p=0.0019).

Table 10 shows the correlation of eGFR with various parameters (cholesterol, triglycerides, HDL, VLDL, and LDL) in stage I of CKD. The correlation of eGFR with triglycerides, HDL, VLDL, and LDL in males was found to be significant with *p*-values 0.01173, 0.003228, 0.01173, and 0.021431, respectively, whereas the correlation of eGFR with Cholesterol in males was found to be nonsignificant with *p*-values 0.07151. The correlation of eGFR with cholesterol, triglycerides, HDL, VLDL, and LDL in females was found to be nonsignificant with *p*-values 0.731755, 0.403256, 0.117554, 0.403256, and 0.780499, respectively.

Table 9: Distribution of LDL in the various stages of CKD and their mean and standard deviation with *p*-value

Stages	Mean ± Standard deviation in males	Mean ± Standard deviation in females	*p*-value
Stage I	84.07±7.90	82.2±4.60	=0.7171
Stage II	113.97±12.19	112.13±9.93	=0.7236
Stage III A	118.37±10.40	114.67±13.86	=0.5448
Stage III B	157.99±13.84	152.78±12.63	=0.3899
Stage IV	202.73±25.74	177.48±18.55	=0.0019*
Stage V	203.45±14.89	201.90±19.73	=0.7447

*(p<0.05)=significant

Table 11 shows the correlation of eGFR with various parameters (cholesterol, triglycerides, HDL, VLDL, and LDL) in stage II of CKD. The correlation of eGFR with cholesterol, triglycerides, VLDL, and LDL in males was found to be significant with *p*-values 0.000012, 0.007975, 0.007975, and 0.014293, respectively, whereas the correlation of eGFR with HDL in males was found to be nonsignificant with p values 0.05901. The correlation of eGFR with cholesterol in females was found to be significant with a *p*-value of 0.040191, whereas the correlation of eGFR with triglycerides, HDL, VLDL, and LDL in females was found to be nonsignificant with a value of 0.130131, 0.292625, 0.130131, and 0.302758, respectively.

Table 12 shows the correlation of eGFR with various parameters (cholesterol, triglycerides, HDL, VLDL, and LDL) in stage III A of CKD. The correlation of eGFR with cholesterol, triglycerides, and VLDL in males was found to be significant with *p*-values 0.024981, 0.001514, and 0.001514, respectively, whereas the correlation of eGFR with HDL and LDL in males was found to be nonsignificant with *p*-values 0.055697 and 0.05624. The correlation of eGFR with triglycerides, HDL, and VLDL in females was found to be significant with *p-values* 0.017919, 0.030399, and 0.017919, whereas the correlation of eGFR with cholesterol and LDL in females was found to be nonsignificant, with *p*-value 0.084817 and 0.078435 respectively.

Table 13 shows the correlation of eGFR with various parameters (cholesterol, triglycerides, HDL, VLDL, and LDL) in stage III B of CKD. The correlation of eGFR with cholesterol, triglycerides, HDL, VLDL, and LDL in

Table 10: Correlation of eGFR with various parameters (cholesterol, triglycerides, HDL, VLDL and LDL) in the stage I of CKD

Parameters	*r*-value male	*p*-value	*r*-value female	*p*-value
eGFR vs. Cholesterol	-0.71448	=0.07151	-0.40977	=0.731755
eGFR vs. Triglycerides	-0.86664	=0.01173*	-0.80671	=0.403256
eGFR vs. HDL	-0.92129	=0.003228*	-0.98317	=0.117554
eGFR vs. VLDL	-0.86664	=0.01173*	-0.80671	=0.403256
eGFR vs. LDL	-0.82844	=0.021431*	-0.3389	=0.780499

*(p<0.05)=significant

Table 11: Correlation between eGFR and parameters (cholesterol, triglycerides, HDL, VLDL and LDL) in the stage II of CKD

Parameters	*r*-value in male	*p*-value	*r*- value in female	*p*-value
eGFR vs. Cholesterol	-0.91411	=0.000012*	-0.72961	=0.040191*
eGFR vs. Triglycerides	-0.69835	=0.007975*	-0.58226	=0.130131
eGFR vs. HDL	-0.53678	=0.05901	-0.42682	=0.292625
eGFR vs. VLDL	-0.69835	=0.007975*	-0.58226	=0.130131
eGFR vs. LDL	-0.65903	=0.014293*	-0.418	=0.302758

*(p<0.05)=significant

Table 12: Correlation between eGFR and parameters (cholesterol, triglycerides, HDL, VLDL, and LDL) in the stage III A of CKD

Parameters	*r*-value in male	*p*-value	*r* -value in female	*p*-value
eGFR vs. Cholesterol	-0.61631	=0.024981*	-0.82634	=0.084817
eGFR vs. Triglycerides	-0.78431	=0.001514*	-0.93932	=0.017919*
eGFR vs. HDL	-0.54264	=0.055697	-0.91385	=0.030399*
eGFR vs. VLDL	-0.78431	=0.001514*	-0.93932	=0.017919*
eGFR vs. LDL	-0.54112	=0.05624	-0.83597	=0.078435

*(p<0.05)=significant

Table 13: Correlation between eGFR and parameters (cholesterol,triglycerides, HDL, VLDL and LDL) in the stage III B of CKD

Parameters	*r*-value in male	*p*-value	*r*-value in female	*p*-value
eGFR vs Cholesterol	-0.9007	<0.00001*	-0.91624	=0.003753*
eGFR vs Triglycerides	-0.84139	<0.00001*	-0.83185	=0.020543*
eGFR vs HDL	-0.5727	=0.008409*	-0.89601	=0.00633*
eGFR vs VLDL	-0.84139	<0.00001*	-0.83185	=0.020543*
eGFR vs LDL	-0.55037	=0.01199*	-0.50091	=0.25317

*(p<0.05)=significant

males was found to be significant with *p-values* <0.00001, <0.00001, 0.008409, <0.00001 and 0.01199, respectively. The correlation of eGFR with cholesterol, triglycerides, HDL, and VLDL in females was found to be significant with *p*-values 0.003753, 0.020543, 0.00633, and 0.020543 whereas the correlation of eGFR with LDL in females was found to be nonsignificant with *p*-value 0.25317.

Table 14 shows the correlation of eGFR with various parameters (cholesterol, triglycerides, HDL, VLDL, and LDL) in stage IV of CKD. The correlation of eGFR with cholesterol, triglycerides, HDL, VLDL, and LDL in males was found to be significant with *p-values* <0.00001, 0.000011, 0.000413, 0.000011, <0.00001 and 0.000091, respectively. The correlation of eGFR with cholesterol, triglycerides, HDL, VLDL, and LDL in females was found to be significant with *p*-values of 0.006,0.001572, 0.00875, 0.001572, and 0.011855, respectively.

Table 15 shows the correlation of eGFR with various parameters (cholesterol, triglycerides, HDL, VLDL, and LDL) in stage V of CKD. The correlation of eGFR with cholesterol, triglycerides, HDL, VLDL, and LDL in males was found to be significant with *p*-values <0.00001, 0.000107, <0.00001, 0.000107, and <0.00001, respectively. The correlation of eGFR with cholesterol, triglycerides,

Table 14: Correlation between eGFR and parameters (cholesterol, triglycerides, HDL, VLDL and LDL) in the stage IV of CKD

Parameters	*r* -value in male	*p*-value	*r*- value in female	*p*-value
eGFR vs. Cholesterol	-0.79004	<0.00001*	-0.69389	=0.006*
eGFR vs. Triglycerides	-0.69473	=0.000011*	-0.76179	=0.001572*
eGFR vs. HDL	-0.58734	=0.000413*	-0.67064	=0.00875*
eGFR vs. VLDL	-0.69473	=0.000011*	-0.76179	=0.001572*
eGFR vs. LDL	-0.75887	<0.00001*	-0.65075	=0.011855*

*(p<0.05)=significant

Table 15: Correlation between eGFR and parameters (cholesterol, triglycerides, HDL, VLDL and LDL) in the stage V of CKD

Parameters	*r*-value in male	*p*-value	*r*- value in female	*p*-value
eGFR vs. Cholesterol	-0.90054	<0.00001*	-0.86373	<0.00001*
eGFR vs. Triglycerides	-0.56892	=0.000107*	-0.63917	=0.005755*
eGFR vs. HDL	-0.79254	<0.00001*	-0.87169	<0.00001*
eGFR vs. VLDL	-0.56892	=0.000107*	-0.63917	=0.005755*
eGFR vs. LDL	-0.72319	<0.00001*	-0.59498	=0.011929*

*(p<0.05)=significant

HDL, VLDL, and LDL in females was found to be significant with p-values <0.00001,0.005755, <0.00001, 0.005755, and 0.011929, respectively.

4.1. Stage I

Figures 5, 6, 7, 8 and 9.

4.2. Stage II

Figures 10, 11, 12, 13 and 14.

4.3. Stage III A

Figures 15, 16, 17, 18 and 19.

4.4. Stage III B

Figures 20, 21, 22, 23 and 24.

4.5. Stage IV

Figures 25, 26, 27, 28 and 29.

4.6. Stage V

Figures 30, 31, 32, 33 and 34.

5. Discussion

CKD is a prevalent health condition that affects millions of individuals worldwide.[12] It is a progressive and irreversible disease that results in the gradual deterioration of kidney function over time. CKD can have various causes, the most common being diabetes and high blood pressure.[13] The progression of CKD occurs in stages, each characterized by the level of kidney function and the presence of symptoms. The five stages of CKD are determined by the estimated glomerular filtration rate (eGFR) which measures

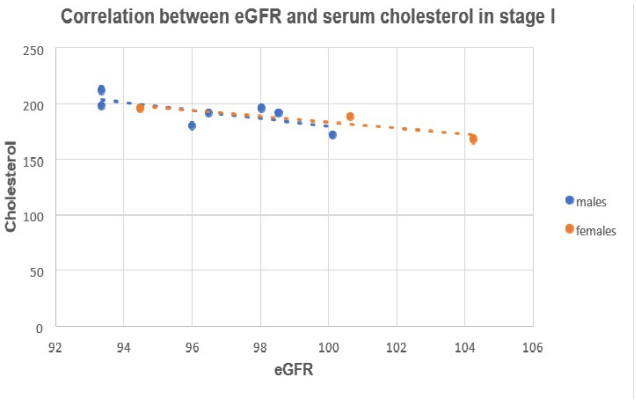

Figure 5: Showing correlation of eGFR and serum cholesterol in stage I

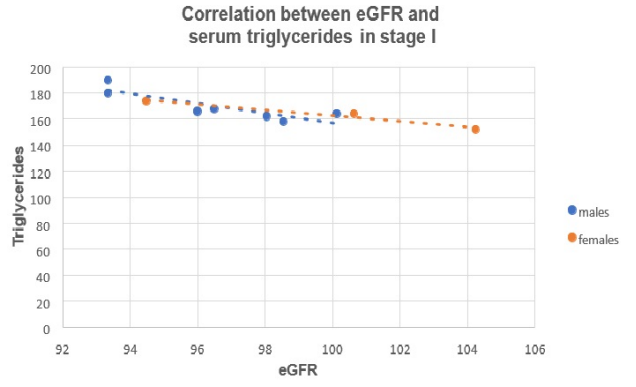

Figure 6: Showing correlation of eGFR and serum triglycerides in stage I

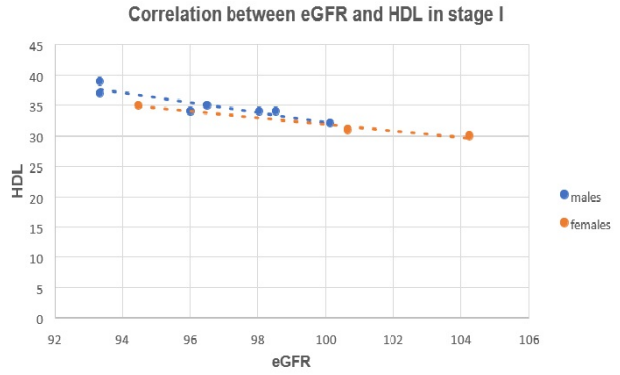

Figure 7: Showing correlation of eGFR and serum HDL in stage I

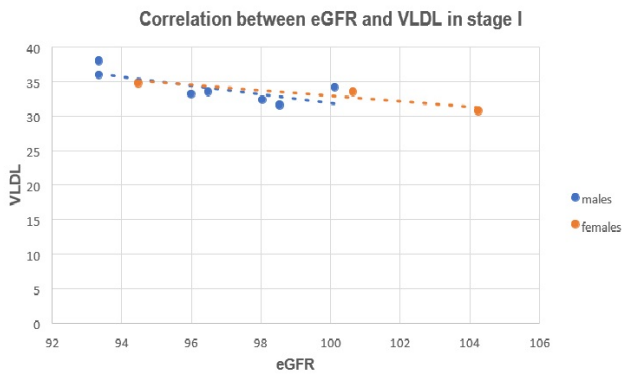

Figure 8: Showing correlation of eGFR and VLDL in stage I

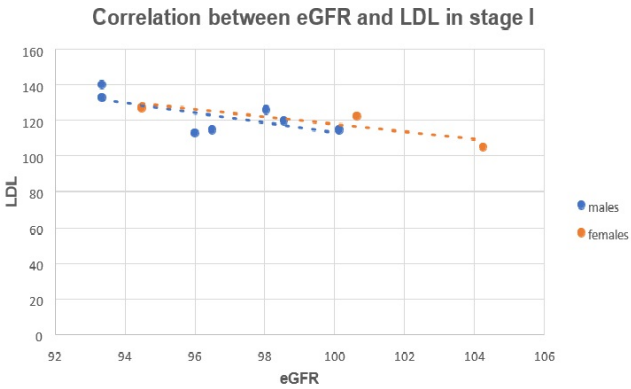

Figure 9: Showing correlation of eGFR and LDL in stage I

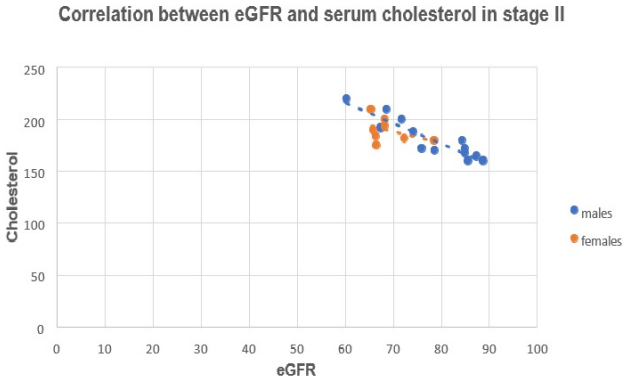

Figure 10: Showing correlation of eGFR and serum cholesterol in stage II

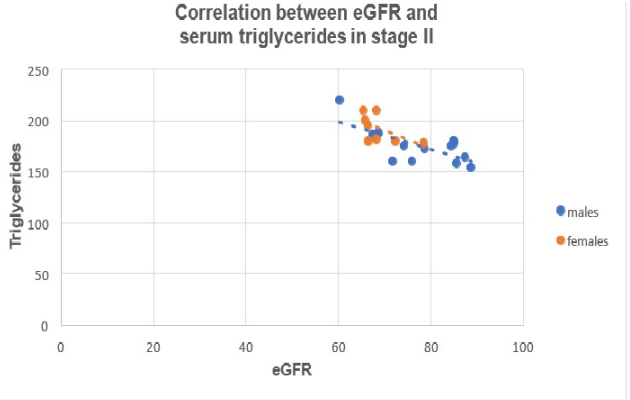

Figure 11: Showing correlation of eGFR and serum triglycerides in stage II

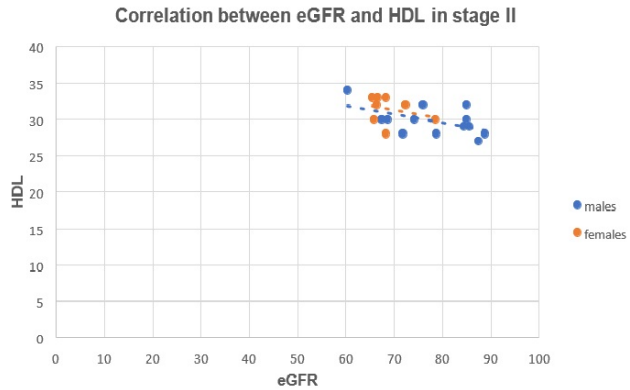

Figure 12: Showing correlation of eGFR and serum HDL in stage II

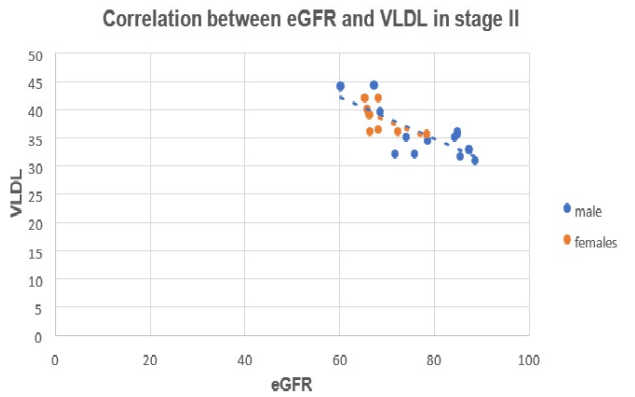

Figure 13: Showing correlation of eGFR and VLDL in stage II

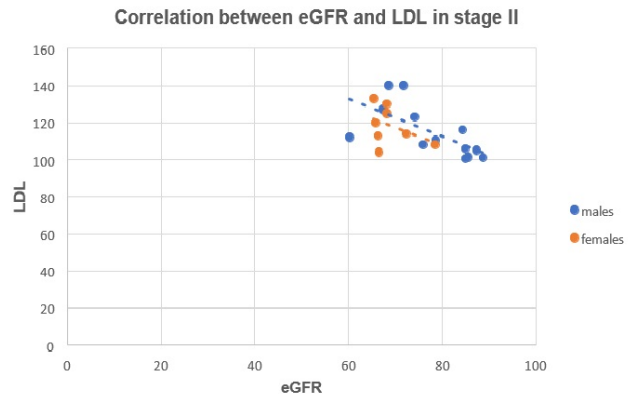

Figure 14: Showing correlation of eGFR and LDL in stage II

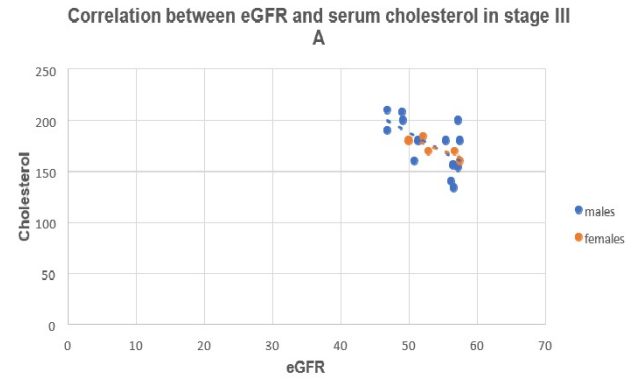

Figure 15: Showing correlation of eGFR and serum cholesterol in stage III A

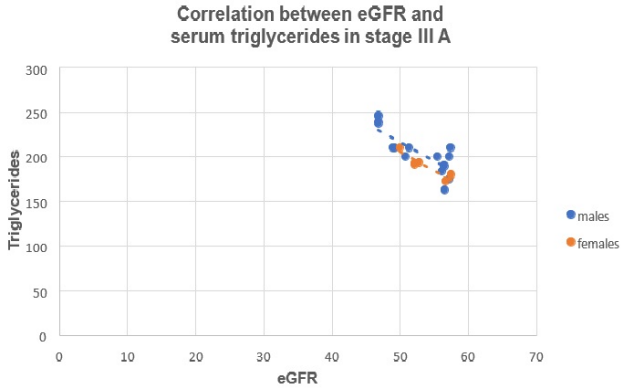

Figure 16: Showing correlation of eGFR and serum triglycerides in stage III A

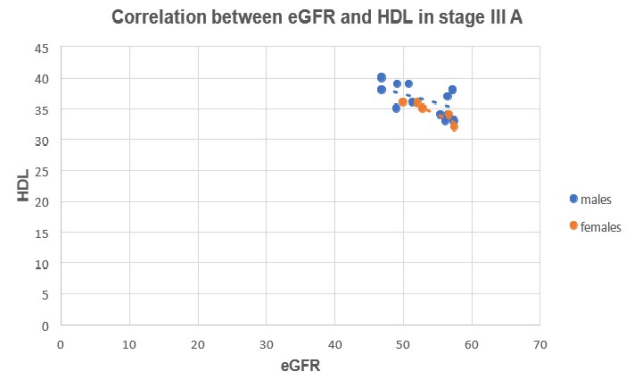

Figure 17: Showing correlation of eGFR and serum HDL in stage III A

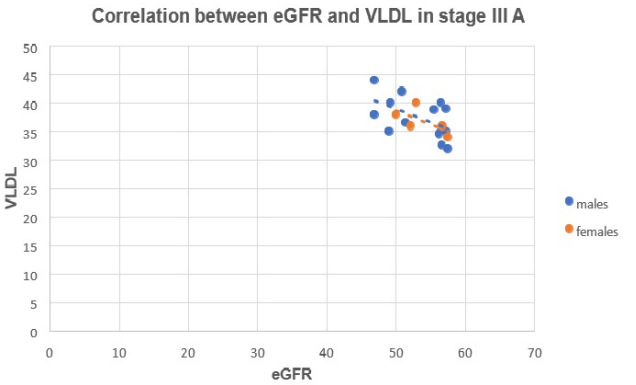

Figure 18: Showing correlation of eGFR and VLDL in stage III A

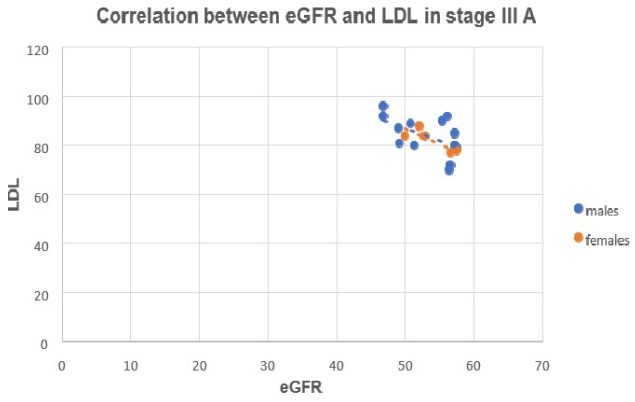

Figure 19: Showing correlation of eGFR and LDL in stage III A

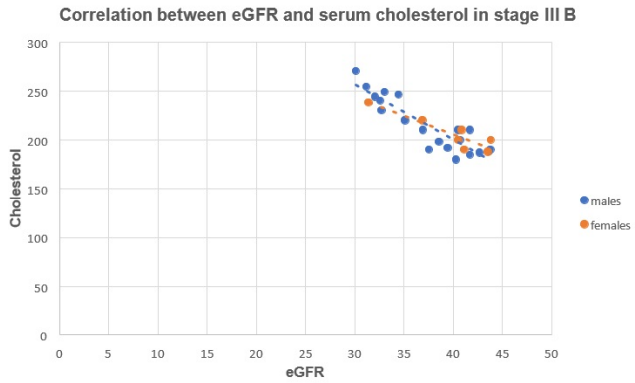

Figure 20: Showing correlation of eGFR and serum cholesterol in stage III B

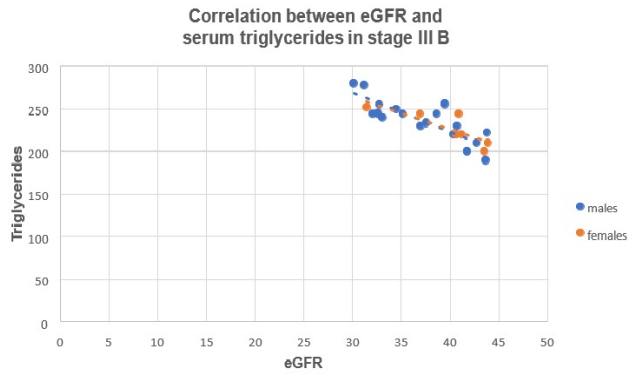

Figure 21: Showing correlation of eGFR and serum triglycerides in stage III B

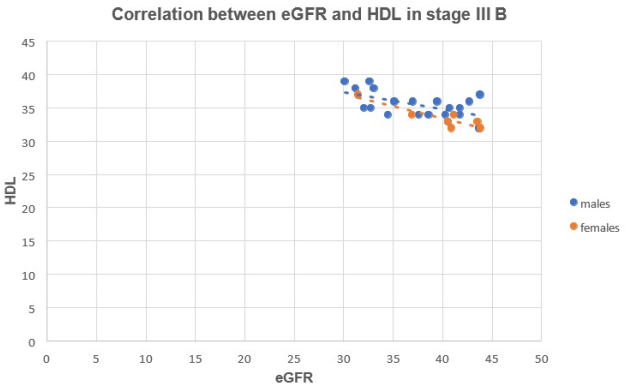

Figure 22: Showing correlation of eGFR and serum HDL in stage III B

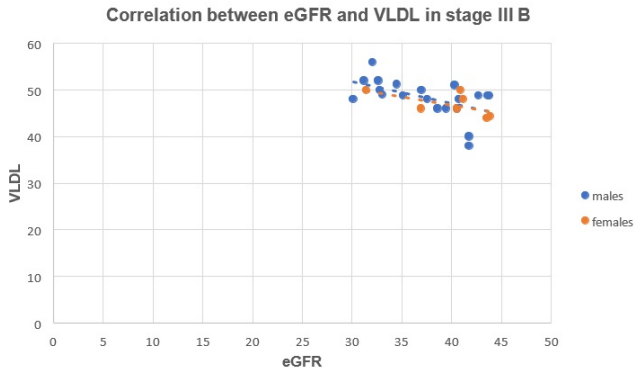

Figure 23: Showing correlation of eGFR and VLDL in stage III B

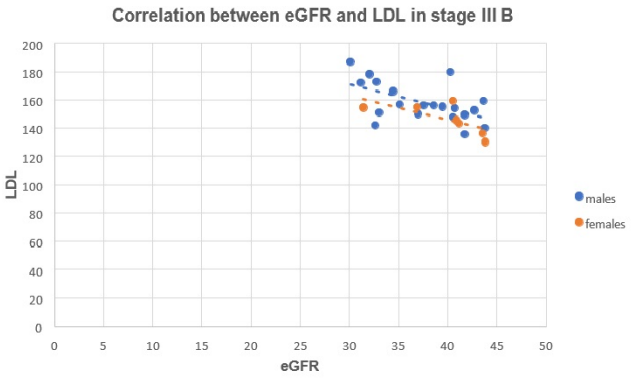

Figure 24: Showing correlation of eGFR and LDL in stage III B

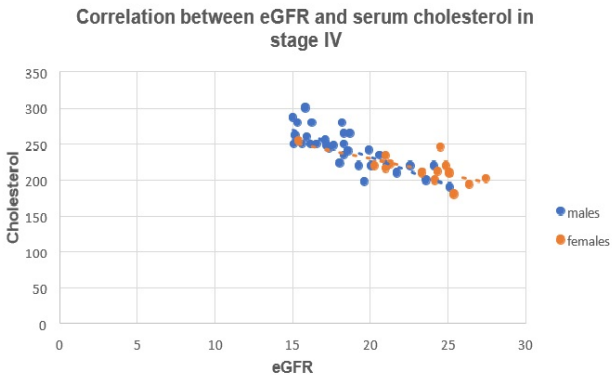

Figure 25: Showing correlation of eGFR and serum cholesterol in stage IV

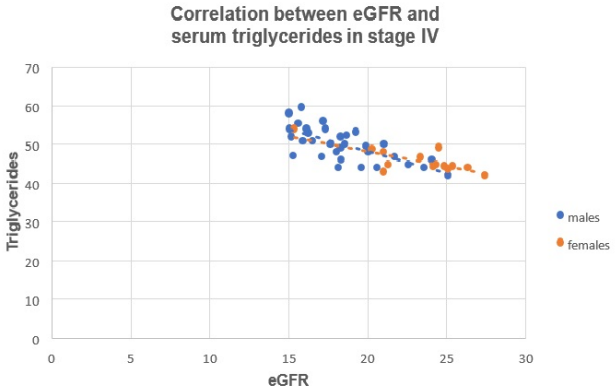

Figure 26: Showing correlation of eGFR and serum triglycerides in stage IV

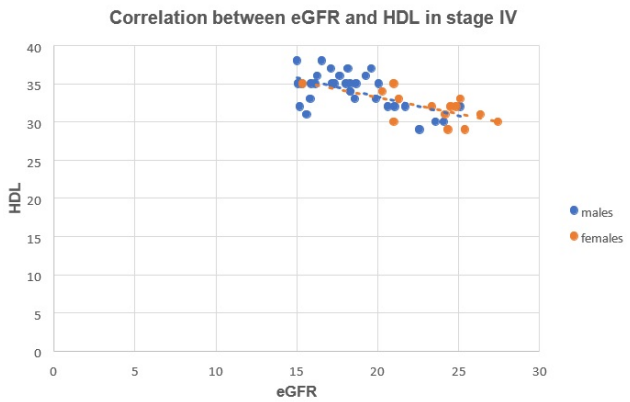

Figure 27: Showing correlation of eGFR and serum HDL in stage IV

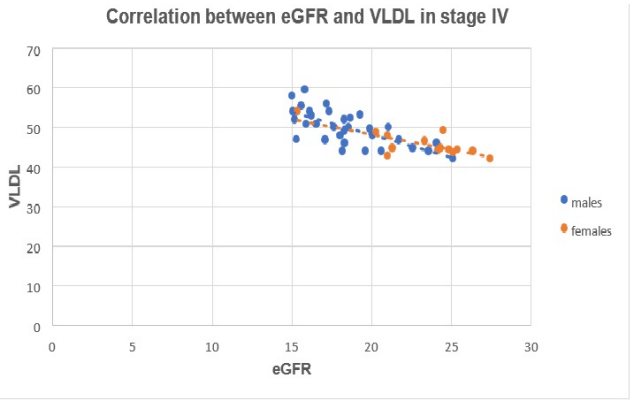

Figure 28: Showing correlation of eGFR and VLDL in stage IV

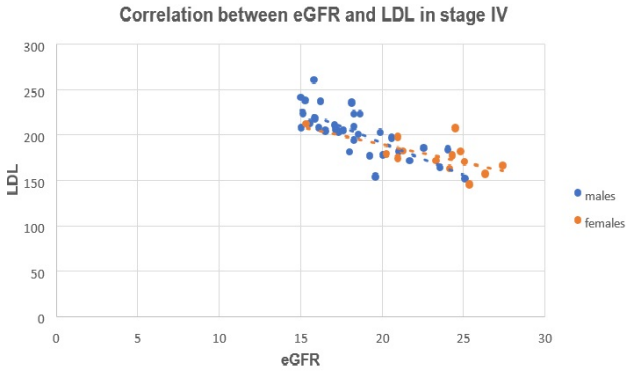

Figure 29: Showing correlation of eGFR and LDL in stage IV

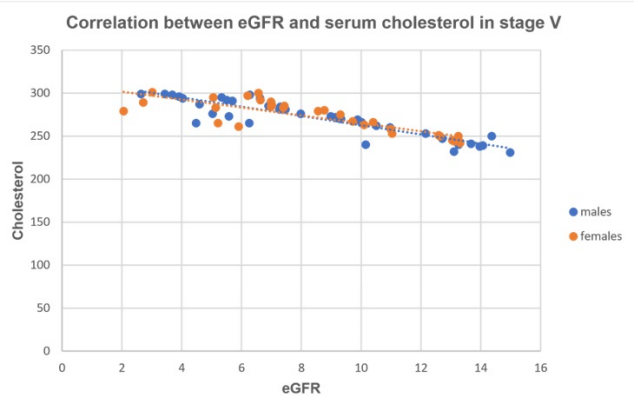

Figure 30: Showing correlation of eGFRand serum cholesterol in stage V

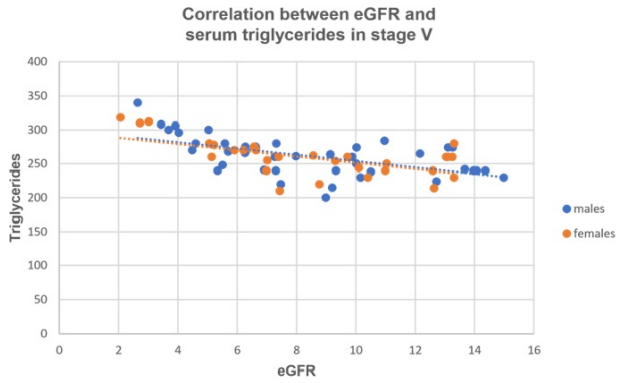

Figure 31: Showing correlation of eGFR and serum triglycerides in stage V

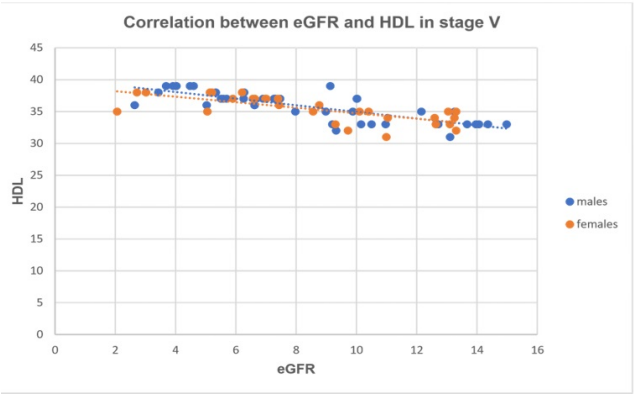

Figure 32: Showing correlation of eGFRand serum HDL in stage V

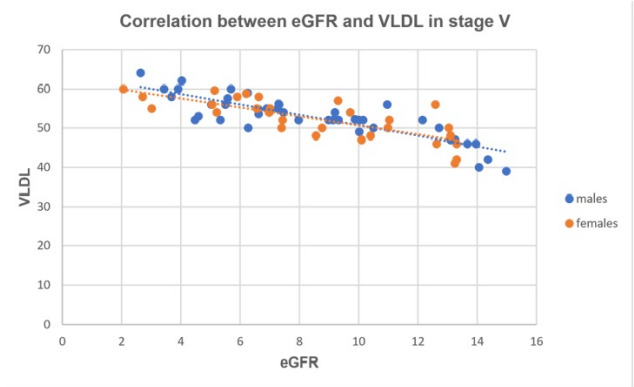

Figure 33: Showing correlation of eGFRand VLDL in stage V

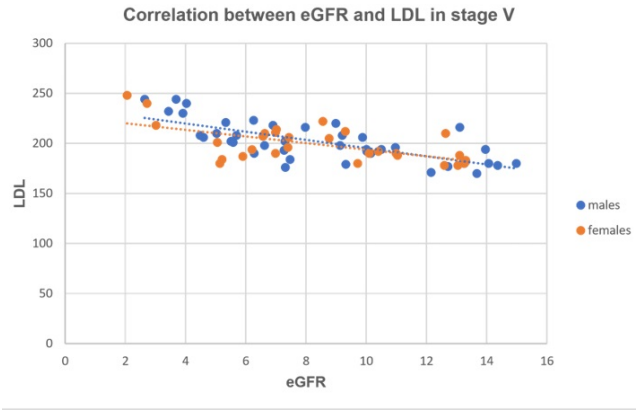

Figure 34: Showing correlation of eGFR and LDL in stage V

how well the kidneys are filtering waste. Diagnosing CKD involves various tests and assessments. The eGFR mentioned earlier is calculated using a blood test that measures the level of creatinine, a waste product produced by muscle metabolism.[14]

This might be the first observational study conducted to assess serum uric acid, serum C-reactive protein, and lipid levels in chronic kidney disease patients. To achieve the objectives of this study, 180 participants attending outpatient departments (OPD) and inpatient departments (IPD) of medicine at AIMSR hospital, Bathinda, were enrolled. The participants were further classified into the stages of chronic kidney disease for correlation and to identify the status of biochemical parameters.

CKD can develop at any age and various conditions can lead to CKD (Mary Mallappallil et al., 2014).[15] Previously study, Ji Cheng Lv et al., 2019 have reported, CKD becomes more common with increasing age.[16] For every decade above age 40 years, GFR declines by 10 ml/min such that by age 70 years, the GFR has declined by about 30 ml/min. The aging process, compounded by risk factors as well as hemodynamic and nonhemodynamic consequences of activation of the renin-angiotensin system, makes the elderly susceptible to CKD. Therefore, in this present study, in order to verify the impact of age for the outcomes of CKD participants were categorized into different age groups, which indicate;7.2% of cases were in the age groups of 20 to 30 years,13.8% of cases were in the age groups of 31 to 40 years,14.4% of cases were in the age groups of 41 to 50, 28.3% of cases were in age groups of 51 to 60 years, while 36.1% of the cases were in the age groups of 61 to 70 years suggested that highest number of cases were in the older age groups (Table 4). This clearly indicated that increase in the age may have direct impact on the CKD. The age of patients in the present study ranged from 20-70 years. The mean± SD for age was 52.8±12.8 years. Maximum patients, that is, 36.1% were in the age group between 61-70 years. The other studies like those done by Vandana Menon et al.,

2003, Madero et al., 2009 and Zhibin li et al., 2013 were had the ages of the patients as 52±12, 52±12 and 58.7± 12 respectively, which was almost similar to the present study.[17–19] On the contrary, a study by Kentaro Kohagura et al., 2013 had mean± SD of the age of patients as 42.4± 18.5, which was lesser than the present study and that by Liu et al., 2021, Sebastjan bevc et al., 2017, Ailing zahang et al., 2021 and Yin et al., 2013 was having mean± SD of age as 63.5 ±13.5, 72.5±5, 73.63 ±10.26, 73.63 ±10.26 respectively which was higher than the present study group.[11,20–23]

In the present study, there were 122 males (67.7%) and 58 females (32.2%) among total of 180 CKD patients. So, in the present study, the analysis according to the gender showed marked male preponderance. As the enrolment of patients in the present study was random so a greater number of males could be due to high prevalence of kidney disease in males as compare to females because of differences in hormones levels. Higher testosterone levels in men may cause a loss in kidney function. On the other hand, men's kidneys may not be protected by estrogen, which is higher in women until menopause. This was in accordance with other studies by Mohamed E. Suliman et al., 2006 and Oluseyi A. Adejumo et al., 2016who were also having a greater number of males in their studies in their studies as compared to females.[24,25] Also, H.K. Aggarwal et al., 2018were having a greater number of males as was there in the present study.[26] Only the study by Sebastjan Bevc et al., 2017[22] had a greater number of females than males.[22]

Over time, CKD became roughly twice as likely to develop in people with high cholesterol. People who have kidney disease are more likely to experience heart issues. In the previous studies, Matthew B Lanktree et al., 2017analysed data from prospective cohorts to investigate the association between cholesterol levels and the development of CKD over time.[27] It reported an increased risk of CKD in individuals with higher cholesterol levels and Liu miao et al., 2021also examined the relationship between cholesterol levels and the incidence of kidney disease.[28] It was found that higher cholesterol levels were associated with an increased risk of incident kidney disease. In this study, we examined the variations in serum cholesterol levels across different CKD stages and explored potential gender-based differences in these levels. Our findings highlight a significant pattern in serum cholesterol levels as CKD progresses through its different stages. Notably, there is a steady increase in serum cholesterol levels with advancing CKD stages. The rise in cholesterol levels becomes more pronounced in the later stages of CKD, with stages IV and V demonstrating substantially higher cholesterol levels compared to the earlier stages (Stage I, Stage II, and Stage III) (Table 9). This escalating trend suggests a potential association between advanced CKD stages and elevated serum cholesterol levels. In agreement with the current study, Oluseyi A Adejumo et al., 2016 explored the association between cholesterol levels and the risk of developing kidney dysfunction.[26] It found that higher total cholesterol levels were associated with an increased risk of developing renal dysfunction. In agreement to these statements, Chang et al., 2015 also examined the relationship between serum lipid levels and the risk of incident CKD.[29] It found that abnormal lipid profiles were associated with an increased risk of developing CKD. Renal dysfunction in later stages of CKD may disrupt the clearance of lipoproteins and contribute to dyslipidemia, ultimately resulting in elevated serum cholesterol levels. Interestingly, we also assessed potential gender-based variations in serum cholesterol levels within each CKD stage. While there were variations between males and females, statistical significance was not consistently achieved across all stages. This suggests that, within the scope of this study, gender might not be a primary factor influencing serum cholesterol levels in CKD patients. The increase in cholesterol concentrations in current study might due to impaired renal function, alcohol consumption, altered lipoprotein metabolism and hormonal disfunction.

Triglyceride levels also rise in CKD. Early in the progression of chronic renal illness, triglyceride levels in serum start to rise and peak in stage IV (Takaaki Kosugi et al., 2021.[30] In the previous studies, Kanno et al., 2015investigated the link between triglyceride levels and the progression of CKD in a Japanese population. It found that higher triglyceride levels were associated with a higher risk of CKD progression and Naraneethan et al., 2014 assessed the impact of triglyceride levels on CKD progression and mortality in a cohort of patients participating in the Kidney Early Evaluation Program.[31] It found that higher triglyceride levels were associated with a greater risk of CKD progression and mortality. In this study, we examined variations in serum triglyceride levels across various CKD stages and explored potential gender-based differences in these levels. Our findings reveal a discernible pattern in serum triglyceride levels as CKD progresses through its stages. There is a consistent upward trend in triglyceride levels with advancing CKD stages. Notably, stages II, IV, and V exhibited significantly higher mean serum triglyceride levels compared to earlier stages (Stage I and Stage III A) (Table 10). This observation suggests a potential link between advanced CKD stages and elevated serum triglyceride levels.

In agreement to the current study, Yanni wang et al., 2016. This study explored the relationship between triglycerides and the development of diabetes in individuals with CKD, as diabetes is a leading cause of CKD.[32] The increasing triglyceride levels seen with CKD progression could be attributed to multifactorial factors. Renal dysfunction and impaired glomerular filtration can influence lipid metabolism, leading to altered triglyceride clearance. Additionally, hormonal changes and

inflammation associated with CKD could contribute to dyslipidemia. Interestingly, our study also explored gender-based differences in serum triglyceride levels within each CKD stage. While some variations were noted, statistical significance was not consistently achieved. This suggests that, within the scope of this study, gender might not be a primary determinant of serum triglyceride levels in CKD patients. The increase in triglycerides concentrations in current study might due to impaired renal function and also by some medications like corticosteroids and immunosuppressants which lead to hypertriglyceridemia.

High-density lipoprotein cholesterol (HDL-C) is a traditional protective factor for CVD, which was thought to achieve its protective effect by mediating the reverse cholesterol transport pathway, but it is known to decrease significantly in CKD patients (Matthew B Lanktree et al., 2017).[27] In the previous studies, JA Lamprea et al., 2005 examined the association between HDL cholesterol levels and the prevalence of CKD in a multi-ethnic population.[33] It found that low HDL cholesterol levels were independently associated with a higher prevalence of CKD and JM Mora et al., 2011explored the relationship between HDL cholesterol levels and the risk of CKD as a secondary outcome.[34] In this study, we examined the variations in HDL levels across different CKD stages and explored potential gender-based differences in these levels. Our findings reveal a notable trend in HDL levels as CKD progresses through its stages. There is a general downward trajectory in HDL levels as CKD advances, with the later stages of CKD showing significantly lower HDL levels compared to the earlier stages (Stage I and Stage II) (Table 11). This observation raises important questions about the potential impact of declining HDL levels on the cardiovascular risk profile of CKD patients.

In agreement to the current study, Hiroo Sonoda et al., 2017investigated the association between HDL cholesterol levels and the decline of kidney function in elderly individuals.[35] It is reported that lower HDL cholesterol levels were linked to a greater decline in kidney function over time. Our study also examined gender-based differences in HDL levels within each CKD stage. While variations were observed, statistical significance was only achieved in certain stages. This suggests that gender might have a nuanced influence on HDL levels in CKD patients, with differences possibly arising from hormonal and metabolic variations. Reduced HDL levels are considered a risk factor for cardiovascular diseases, which are highly prevalent in CKD patients. The declining trend in HDL levels throughout CKD stages underscores the need for comprehensive cardiovascular risk management strategies in CKD patients.

VLDL levels also correlated with CKD (Jhi kai huang et al., 2022).[36] In the previous studies, Mora et al., 2007 examined the relationship between VLDL cholesterol and various health outcomes, including CKD and Klag et al., 2000 investigated the relationship between serum lipids, including VLDL cholesterol, and the risk of nephropathy in individuals with type 2 diabetes, which is a common cause of CKD.[37,38] In this study, we explored variations in VLDL levels across different CKD stages and examined potential gender-based differences in these levels. Our findings reveal an intriguing pattern in VLDL levels as CKD progresses through its stages. While not consistently significant across all stages, there is a noticeable upward trend in VLDL levels as CKD advances. Notably, stages IV and V exhibited significantly higher VLDL levels compared to earlier stages (Stage I and Stage III B) (Table 12). This suggests a possible association between advanced CKD stages and elevated VLDL levels. Elevated VLDL levels are often associated with an increased risk of cardiovascular diseases due to their role in transporting triglycerides. The observed increase in VLDL levels as CKD advances could potentially contribute to the heightened cardiovascular risk commonly observed in CKD patients. In agreement to the current study, Van der velde et al., 2010.[39] This study investigated the predictive value of apolipoprotein B (a component of VLDL) for renal function decline in patients. Mechanistically, altered lipid metabolism and reduced renal function might contribute to the observed changes in VLDL levels. Our study also explored gender-based differences in VLDL levels within each CKD stage. While some variations were noted, statistical significance was not consistently achieved. This suggests that gender might not be a primary determinant of VLDL levels in CKD patients within the scope of this study. Elevated VLDL levels are associated with dyslipidemia and increased cardiovascular risk. The observed trend in VLDL levels throughout CKD stages underscores the importance of comprehensive cardiovascular risk management strategies for CKD patients, particularly those in advanced stages.

LDL modifications in the CKD increase cardiovascular risk (Charles J. Ferro et al., 2018).[40] In the previous studies, Takaaki kosugi et al., 2021examined the association between LDL cholesterol levels and the risk of kidney function decline in older adults.[30] It is found that higher LDL cholesterol levels were independently associated with an increased risk of kidney function decline and Mora et al., 2007 investigated the relationship between serum cholesterol levels, including LDL cholesterol, and the progression of CKD in patients without diabetes.[39] It is suggested that high LDL cholesterol levels might contribute to the progression of kidney disease. In this study, we examined variations in LDL levels across different CKD stages and explored potential gender-based differences in these levels. Our findings demonstrate a significant pattern in LDL levels as CKD progresses through its stages. There is a clear and consistent increase in LDL levels with advancing CKD stages. Notably, stages IV and V exhibit significantly

higher mean LDL levels compared to the earlier stages (Stage I, Stage II, and Stage III A) (Table 13). This observed trend underscores a potential association between advanced CKD stages and elevated LDL levels. In agreement to the current study, Chang et al., 2019 examined the relationship between LDL cholesterol levels and the risk of developing CKD in a prospective cohort.[29] It was found that higher LDL cholesterol levels were associated with an increased risk of incident CKD. In agreement to these statements, Kamanna and Kashyap et al., 2013 discusses the importance of managing LDL cholesterol levels in individuals with CKD to reduce cardiovascular risk and potentially slow CKD progression.[41] Elevated LDL levels are recognized as a major risk factor for cardiovascular diseases, which are common comorbidities in CKD patients. The increasing LDL levels observed as CKD progresses could further contribute to the elevated cardiovascular risk in this population. Altered lipid metabolism, inflammation, and reduced renal function in later stages of CKD could all play a role in the observed changes in LDL levels. Interestingly, our study also evaluated gender-based differences in LDL levels within each CKD stage. While variations were noted, statistical significance was only achieved in Stage IV. This suggests that, within the scope of this study, gender might not consistently influence LDL levels in CKD patients.

In this study, we explored the correlations between eGFR and lipid parameters in the stages of CKD patients. The correlations between eGFR and lipid parameters demonstrate varying strengths and significance. In males, eGFR is negatively correlated with cholesterol, triglycerides, HDL, VLDL and LDL. In females, the relationships are generally weaker and not statistically significant for most parameters. These correlations highlight the intricate interplay between renal function and lipid metabolism. As eGFR decreases in stages of CKD, adverse changes in lipid profiles, particularly decreased HDL and increased VLDL, could potentially contribute to the progression of renal dysfunction and the heightened cardiovascular risk commonly associated with CKD.

It's important to acknowledge some limitations of our study, including the relatively small sample size and the observational design. The observational design restricts our ability to establish causal relationships between lipid levels and CKD progression. Additionally, our study did not consider factors such as dietary habits, lifestyle, infections, comorbidities and medication use, which could influence lipid levels.

The link between elevated lipid levels and CKD progression carries clinical implications. Elevated cholesterol, triglycerides, VLDL, LDL levels and declining HDL levels are recognized risk factors for atherosclerosis and cardiovascular diseases, which are common comorbidities in CKD patients. The association between high cholesterol, triglycerides, VLDL, LDL levels and declining HDL levels and cardiovascular risk highlights the importance of managing lipid profiles in CKD patients, especially as the disease advances.

Future research could benefit from longitudinal designs that track changes in lipid levels as CKD progresses. Additionally, investigating the mechanisms behind dyslipidemia in CKD, including factors related to altered lipid metabolism and renal dysfunction, could provide deeper insights into the observed patterns.

6. Conclusion

CKD is one of the non-communicable diseases that have had an increase in deaths related to them over the previous two decades. The present study highlights the progressive increase in serum cholesterol, serum triglycerides, VLDL, LDL levels as CKD advances through its stages. A progressive decline in HDL levels as CKD also advances through its stages. The correlations between eGFR and various biomarkers in the stages of CKD shed light on the complex interactions between renal function, lipid metabolism. The negative correlations with lipid parameters suggest that even in early CKD stages, there are indications of altered physiological processes.

The correlations underscore the importance of considering multiple biomarkers in assessing CKD progression and cardiovascular risk.

7. Ethics Declaration

This study was approved by the AIMSR Research Committee and the Ethics Committee of Biomedical and Health Research, Adesh University, Bathinda.

8. Consent

Written informed consent was obtained from all participants for the use of their blood samples for this research and the publication of any findings in the scientific literature.

9. Source of Funding

None.

10. Conflict of Interest

None.

References

1. Kovesdy CP. Epidemiology of chronic kidney disease: An update 2022. *Kidney Int Suppl (2011)*. 2022;12(1):7–11.
2. Wanner C, Tonelli M. KDIGO clinical practice guideline for lipid management in CKD: Summary of recommendation statements and clinical approach to the patient. *Kidney Int*. 2014;85(6):1303–9.
3. Hill NR, Fatoba ST, Oke JL, Hirst JA, O'callaghan CA, Lasserson DS, et al. Global prevalence of chronic kidney disease: A systematic review and meta-analysis. *PLoS One*. 2016;11(7):e0158765.
4. Disease G, Incidence I, Collaborators P, S. Global, regional, and national incidence, prevalence, and years lived with disability for 310

diseases and injuries, 1990-2015: A systematic analysis for the Global Burden of Disease Study 2015. *Lancet*. 2016;388(10053):1545–1602.
5. Rizvi SM, Muhit MM, Tao S, Kalam A, Rahman ML, Alom MK, et al. Treatment and prevention of chronic kidney disease. *N Am Acad Res*. 2023;6(1):186–219.
6. Wesseling C, Crowe J, Hogstedt C, Jakobsson K, Lucas R, Wegman DH. The epidemic of chronic kidney disease of unknown etiology in Mesoamerica: a call for interdisciplinary research and action. *Am J Public Health*. 2013;103(11):1927–30.
7. Kazancioğlu R, Kidney Int Suppl. Risk factors for chronic kidney disease: An update. *Kidney Int Suppl*. 2013;3(4):368–71.
8. Bursill D, Taylor WJ, Terkeltaub R, Kuwabara M, Merriman TR, Grainger R, et al. The gout, hyperuricemia, and crystal-associated disease network consensus statemen regarding labels and definitions for disease elements in gout. *Arthritis Care Res (Hoboken)*. 2019;71(3):427–34.
9. Nehring SM, Goyal A, Patel BC. C reactive protein. 2023;Available from: https://www.ncbi.nlm.nih.gov/books/NBK441843/.
10. Giollabhui NM, Ellman LM, Coe CL, Byrne ML, Abramson LY, Alloy LB. To exclude or not to exclude: Considerations and recommendations for C-reactive protein values higher than 10 mg/L. *Brain Behav Immun*. 2020;87:898–900.
11. Jha V, Garcia-Garcia G, Iseki K, Li Z, Naicker S, Plattner B, et al. Chronic kidney disease: Global dimension and perspectives. *Lancet*. 2013;382(9888):260–72.
12. Nacak H, Van Diepen M, Qureshi AR, Carrero JJ, Stijnen T, Dekker FW, et al. Uric acid is not associated with decline in renal function or time to renal replacement therapy initiation in a referred cohort of patients with Stage III, IV and V chronic kidney disease. *Nephrol Dial Transplant*. 2015;30(12):2039–45.
13. Ye M, Hu K, Jin J, Wu D, Hu P, He Q. The association between time-mean serum uric acid levels and the incidence of chronic kidney disease in the general population: A retrospective study. *BMC Nephrol*. 2018;19(1):190.
14. Phukan RR, Goswami RK. Unusual dyslipidemia in patients with chronic kidney diseases. *J Clin Diagn Res*. 2017;11(1):BC01–4.
15. Saini M, Vamne A, Kumar V, Chandel MS. The study of pattern of lipid profile in chronic kidney disease patients on conservative management and hemodialysis: A comparative study. *Cureus*. 2022;14(1):e21506.
16. Miao L, Min Y, Qi B, Zhu CM, Chen JH, Deng GX, et al. Causal effect between total cholesterol and HDL cholesterol as risk factors for chronic kidney disease: A mendelian randomization study. *BMC Nephrol*. 2021;22(1):35.
17. Kovesdy CP, Anderson JE, Kalantar-Zadeh K. Inverse association between lipid levels and mortality in men with chronic kidney disease who are not yet on dialysis: Effects of case mix and the malnutrition-inflammation-cachexia syndrome. *J Am Soc Nephrol*. 2007;18(1):304–11.
18. Wang Y, Qiu X, Lv L, Wang C, Ye Z, Li S, et al. Correlation between serum lipid levels and measured glomerular filtration rate in Chinese patients with chronic kidney disease. *PLoS One*. 2016;11(10):e0163767.
19. Zhang L, Yuan Z, Chen W, Chen S, Liu X, Liang Y, et al. Serum lipid profiles, lipid ratios and chronic kidney disease in a Chinese population. *Int J Environ Res Public Health*. 2014;11(8):7622–35.
20. Arnold R, Issar T, Krishnan AV, Pussell BA. Neurological complications in chronic kidney disease. *JRSM Cardiovasc Dis*. 2016;5:2048004016677687.
21. Cravedi P, Remuzzi G. Pathophysiology of proteinuria and its value as an outcome measure in chronic kidney disease. *Br J Clin Pharmacol*. 2013;76(4):516–23.
22. Nehus E. Obesity and chronic kidney disease. *Curr Opin Pediatr*. 2018;30(2):241–46.
23. Olsen E, Van Galen G. Chronic renal failure: Causes, clinical findings, treatments and prognosis. *Vet Clin North Am Equine Pract*. 2022;38(1):25–46.
24. Kazancioglu R. Risk factors for chronic kidney disease: An update. *Kidney Int Suppl*. 2011;3(4):368–71.
25. Zhang L, Zhang P, Wang F, Zuo L, Zhou Y, Shi Y, et al. Prevalence and factors associated with CKD: A population study from Beijing. *America J Kidney Dis*. 2008;51(3):373–84.
26. Nivedita A, Kumar A, Sinha A, Mitra J, Sinha R. Uric acid levels in chronic kidney disease: A hospital based cross-sectional study in RIMS, Ranchi, Jharkhand. *Int J Res Med Sci*. 2021;9(2):569–72.
27. Kugler E, Cohen E, Goldberg E, Nardi Y, Levi A, Krause I, et al. C reactive protein and long-term risk for chronic kidney disease: A historical prospective study. *J Nephrol*. 2015;28(3):321–7.
28. Menon V, Wang X, Greene T, Beck GJ, Kusek JW, Marcovina SM, et al. Relationship between C-reactive protein, albumin, and cardiovascular disease in patients with chronic kidney disease. *Am J Kidney Dis*. 2003;42(1):44–52.
29. Son M, Seo J, Yang S. Association between dyslipidemia and serum uric acid levels in Korean adults: Korea National Health and Nutrition Examination Survey. *PLoS One*. 2020;15(2):e0228684.
30. Nakano T, Tanaka S, Tsuruya K, Kitazono T. Relationship between serum lipid concentrations and impaired renal function in patients with chronic kidney disease: The Fukuoka Kidney Disease Registry Study. *Clin Exp Nephrol*. 2021;25(4):385–93.
31. Bevc S, Hojs R, Ekart R, Završnik M, Gorenjak M, Puklavec L. Simple cystatin C formula for estimation of glomerular filtration rate in overweight patients with diabetes mellitus type 2 and chronic kidney disease. *Exp Diabetes Res*. 2012;2012:179849.
32. Aggarwal HK, Jain D, Chauda R, Bhatia S, Sehgal R. Assessment of malnutrition inflammation score in different stages of chronic kidney disease. *Pril (Makedon Akad Nauk Umet Odd Med Nauki)*. 2018;39(2-3):51–61.
33. Goicoechea M, De Vinuesa S, Verdalles U, Ruiz-Caro C, Ampuero J, Rincón A, et al. Effect of allopurinol in chronic kidney disease progression and cardiovascular risk. *Clin J Am Soc Nephrol*. 2010;5(8):1388–93.
34. Li Z, Liu Q, Mao H, Li Z, Dong X, Liu Y, et al. Gender difference in the association of hyperuricemia with chronic kidney disease in southern China. *Kidney Blood Press Res*. 2013;36(1):98–106.
35. Liu M, Li XC, Lu L, Cao Y, Sun RR, Chen S, et al. *Eur Rev Med Pharmacol Sci*. 2014;18(19):2918–26.
36. Zhang A, Deng W, Zhang B, Ren M, Tian L, Ge J, et al. Association of lipid profiles with severity and outcome of acute ischemic stroke in patients with and without chronic kidney disease. *Neurol Sci*. 2021;42(6):2371–8.
37. Johnson RJ, Nakagawa T, Jalal D, Sánchez-Lozada LG, Kang DH, Ritz E. Uric acid and chronic kidney disease: Which is chasing which? *Nephrol Dial Transplant*. 2013;28(9):2221–8.
38. Lora CM, Daviglus ML, Kusek JW, Porter A, Ricardo AC, Go AS, et al. Chronic kidney disease in United States Hispanics: A growing public health problem. *Ethn Dis*. 2009;19(4):466–72.
39. Fox CS, Matsushita K, Woodward M, Bilo HJ, Chalmers J, Heerspink HJ, et al. Associations of kidney disease measures with mortality and end-stage renal disease in individuals with and without hypertension: a meta-analysis. *Lancet*. 2012;380(9854):1662–73.
40. Tonelli M, Wiebe N, Culleton B, House A, Rabbat C, Fok M, et al. Chronic kidney disease and mortality risk: a systematic review. *J America Soc Nephrol*. 2006;17(7):2034–47.
41. Zubovic SV, Kristic S, Prevljak S, Pasic IS. Chronic kidney disease and lipid disorders. *Med Arch*. 2016;70(3):191–2.

Author biography

Sandeep Singh, M.Sc. Student https://orcid.org/0009-0001-1082-5319

Umesh Kumar, Tutor https://orcid.org/0000-0003-1936-7630

Rajinderjit Singh Ahi, Professor and Head

Basharat Azhar Paul, M.Sc. Biochemistry Student

Cite this article: Singh S, Kumar U, Ahi RS, Paul BA. Lipoprotein abnormalities: A potential consequence of chronic kidney disease. *Int J Clin Biochem Res* 2024;11(2):108-124.