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Evaluation of Lipid profile in patients with chronic kidney disease in Banas Medical College and Research Institute, Palanpur, Gujarat, India

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Type of study: Original Research Paper

Abstract:

Background: Patients with chronic renal failure (CRF) frequently experience dyslipidemia. Dyslipidemia is linked to cardiovascular mortality in people with CRF. This study compared the body mass index (BMI) of matched healthy control subjects with that of CRF patients to uncover lipid abnormalities and their significance.

Materials and Methods: Three fasting lipid profiles were calculated in this study using data from 280CRF patients. As controls, a healthy population of identical age, sex, and BMI was utilized. The data was documented into a master sheet and statistical analysis was carried out.

Results: Patients with CRF experience dyslipidemia. Despite the fact that the total cholesterol was higher in CRF patients than in controls, the difference was statistically insignificant. In instances with CRF, triglycerides increased statistically significantly. LDL-C levels were higher in CRF instances, but statistically speaking, the difference was not statistically significant. On the other hand, as compared to controls, the high-density lipoprotein cholesterol (HDL-C) exhibited a statistically significant decline.

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Conclusion: In CRF, lipid abnormalities are frequent. There are no statistically significant changes in total cholesterol. When compared to normal, triglycerides statistically significantly rise in CRF instances.Patients with CRF exhibit a statistically significant decline in HDL-C compared to controls.

Keywords: Dyslipidemia, lipid profile, and chronic renal disease.

Introduction: Whenever a disease process compromises the anatomical or functional integrity of the kidneys, chronic kidney disease (CKD) develops. Chronic kidney disease (CKD) leads to renal failure.Multiple lipid metabolic problems present in chronic renal illness leads to a very atherogenic phenotype. Although the nephrotic syndrome exhibits the most glaring lipid abnormalities, hyperlipidemia is a hallmark of all causes of renal illness.

Recognizing that atherosclerotic heart disease is the most frequent cause of morbidity and death in people with end-stage renal disease (ESRD), lipid abnormalities in CRF are crucial.

Due to the steadily aging of ESRD patients and the diverse constellation of uremia-associated characteristics, cardiovascular illnesses are the primary causes of mortality in this population. Renal replacement treatment, dialysis, or transplantation may be required to support life when the kidney function has declined and is no longer sufficient to do so. Therefore, it's crucial to stop chronic renal insufficiency from growing and eventually leading to ESRD.[1-4]

Inevitably, kidney disease is typically asymptomatic in the early stages. Identification of people at risk for CKD at an early stage is crucial. Type I diabetes, high blood pressure, the presence of the protein barrier, a family history of renal disease, and advancing age are major risk factors for the development and progression of CKD.Glycemic management (in diabetes), blood pressure control for individuals with excessive blood pressure, and the use of angiotensin-converting enzyme inhibitors can all decrease the course of kidney disease to the end stage.[2-4]

The leading cause of mortality in persons with CRF and ESRD is cardiovascular disease. Hypertension may contribute to the onset of atherosclerotic coronary artery disease in addition to compromising microcirculation, particularly when there are several lipid abnormalities present in ESRD.

Reduced HDL-C production and decreased reverse cholesterol pathway activity are additional variables that may play a role in atherosclerotic coronary artery disease in ESRD patients.

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Patients on dialysis and those with CKD have a different dyslipidemia spectrum from the general population. Depending on the stage of CKD, it affects all lipoprotein classes and exhibits significant differences. Additionally, a growing body of clinical experience evidence points to the possibility that lipids play a role in the onset and development of chronic renal illness. These individuals are more prone to develop ESRD since they are always present with potentially harmful lipid abnormalities.[5-9]

Therefore, it is crucial to analyze the lipoprotein subclass in CRF patients in order to evaluate the clinical outcome.

Materials and Methods:

Data Source: Prior informed consent was obtained from patients with CKD who reported to Banas Medical College & Research Institute (BMCRI), Palanpur, Gujarat.

Collection of data: 280 patients with CKD who visited BMCRI over the course of a year and were diagnosed, comprised the participants of the current cross-sectional investigation.

Inclusion criteria: CKD patients between the ages of 18 and 80; those with known CKD regardless of the cause; those receiving conservative or dialysis treatment for CKD; and those who have the disease as demonstrated by imaging (bilateral kidney shrinkage/loss of corticomedullary differentiation) or biochemistry (elevated blood urea, elevated serum creatinine) for longer than three months.

Exclusion criteria: Patients using medications that influence lipid metabolism, such as blockers, statins, steroids, and oral contraceptives, as well as those with nephrotic syndrome and acute renal failure, Pregnant females, known hypothyroidism, The following guidelines were used to evaluate these individuals using the proforma:A medical assessment and clinical history, Standard tests such as blood hemoglobin (HB)%, total count, differential count, blood sugar, and urine analysis, renal indicators such blood urea and serum creatinine, the fasting lipid profile and an abdominal ultrasound.

Within 4 to 6 hours after collection, every specimen was examined. Enzymatic measurement of the plasma's total cholesterol and triglycerides was followed by the determination of HDL-C by measuring the cholesterol in the supernatant following the precipitation of lipoproteins carrying apolipoprotein B (Apo-B). Using the Friedewald formula, the LDL-C is estimated.In clinical practice, the Friedewald formula seems to be the most useful and trustworthy way to calculate LDL-C.

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LDL-C=Totalcholesterol-[HDL-C+(Triglycerides/5)]

By dividing the plasma triglycerides by 5, which represents the ratio of cholesterol to triglyceride in VLDL particles, one may calculate the amount of very-low-density lipoprotein (VLDL). The test resolution must be acquired on fasting plasma, and the triglyceride level must be less than 350 mg/dL for this formula to be considered relatively accurate. Application of ultra centrifugation methods (Beta quantification) is necessary for the correct measurement of LDL-C in situations with triglyceride levels above this.

Results: A master chart was created to store the information gathered about all of the chosen instances. With the use of a computer, Statistical Package for Social Sciences (SPSS, 2021) and the Epidemiological Information Package (EPI, 2021), data analysis was carried out. This program was used to compute the range, frequencies, percentages, means, standard deviations, chi-square, and p-values. To determine the significance of a difference between two quantitative variables, the Kruskal-Wallis chi-square test was performed. A significant link was considered to exist when the p-value was less than 0.05.

The findings are shown in Tables 1-4 and Graphs 1 and 2.

Table 1: Age distribution

Age Distribution	Cases		Control group	
	No.	%	No.	%
41–45	5	1.8	20	16
46–50	145	51.8	60	48
51–55	95	33.9	25	20
55–59	35	12.5	20	16
Total	280	100	125	100
Range	45–57		41–59	
Mean	51.2		50.4	
Standarddeviation	3.0		4.9	
p-value	0.1536			
Notsignificant				

Table2: Sex distribution

Sex	Cases		Control group	
	No.	%	No.	%
Male	140	50	50	40
Female	140	50	75	60
p-value	0.7945			
Notsignificant				

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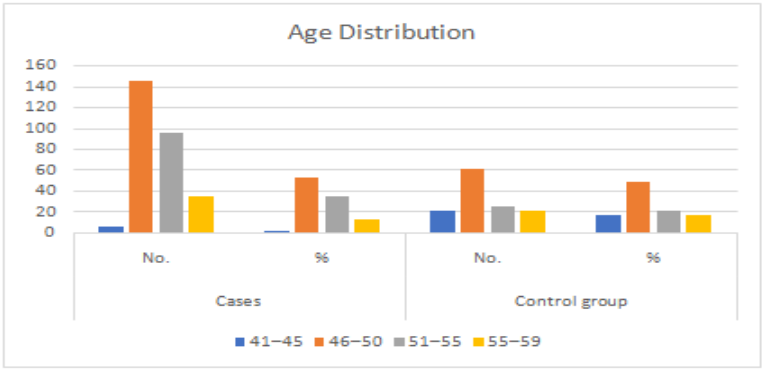
Table3: Bodymass index

BMI	Cases	Controlgroup
Range	110–130	110–130
Mean	24.55	24.76
Standarddeviation	1.08	1.09
p-value	0.3668	
Notsignificant		

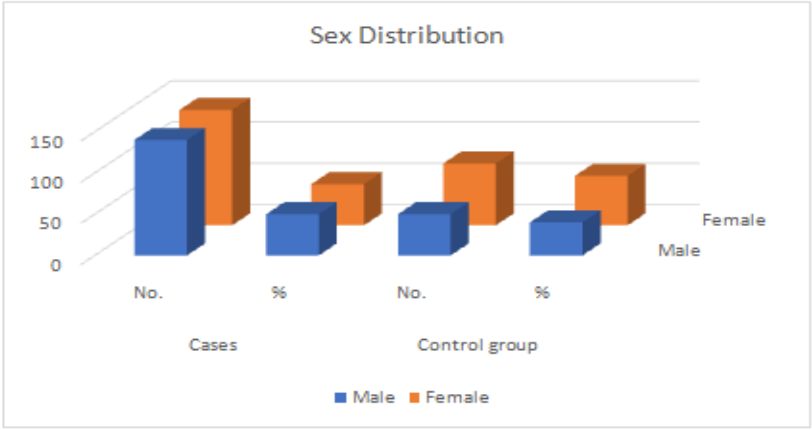
Table4: Lipid profile

	Cases			Controls			
Lipid	Range	Mean	SD	Range	Mean	SD	p-value
TC	158–269	213.6	14.9	132–245	207.8	22.1	0.1761NS
TGL	110–264	205.9	44.9	112–180	148.0	16.3	0.0001S
HDL	16–75	39	18.5	38–86	60.7	14.3	0.0001S
LDL	110–171	139.4	16.4	110–172	127.2	15.6	0.1031NS

SD:Standarddeviation;TC:Totalcholesterol;TGL:Triglyceride;NS:Notsignificant;S:Significant



Graph1: Age distribution



Graph2: Sex distribution

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Discussion: When a disease process compromises the kidneys structural or functional integrity, chronic kidney disease is the outcome. Chronic kidney disease (CKD) leads to renal failure. The leading cause of death in persons with mild to severe CKD and ESRD is cardiovascular disease. It is well known that patients with CKD display significant changes in lipoprotein metabolism, which, in their most severe form, may lead to the development of severe dyslipidemia. Dyslipidemia has been established as a well-known traditional risk factor for cardiovascular disease in the general population.

The purpose of this study was to pinpoint the lipid abnormalities seen in CRF patients admitted to RRMCH. The research comprised a total of 280 patients that met the diagnostic criteria for CRF. 125 healthy, age-, sex-, and BMI-matched controls were used to compare the lipid profile. These controls also met the inclusion and exclusion criteria.

The age range for the 280 cases was 45–59 years, with a mean age of 51.2 years. The age range for the controls was between 41 and 59 years, with a mean of 50.4 years. Regarding age, there was no discernible difference between cases and controls (p-value 0.1536). They can so be contrasted.

The research group consisted of 140 males and 140 females, an equal number of each gender. 50 of the 125 controls were men and 75 were women. Regarding sex, there was no discernible difference between the patients and controls (p-value 0.7945). The patients' average BMI was 24.55 kg/m2.

The controls' average BMI was 24.76 kg/m2. Regarding BMI, there was no discernible difference between the patients and controls.

After examining the lipid profile and contrasting the CRF patients with the controls, we discovered that triglycerides had significantly increased while HDL-C had significantly decreased. Between the patients and the controls, there was no appreciable difference in total cholesterol or LDL-C.

In the CRF patients, the mean total cholesterol level was 213.6 mg/dL, compared to 207.8 mg/dL in the controls. This parameter did not differ statistically significantly (p-value 0.1761) from other parameters. This finding was consistent with those made by Kimak et al. in their study of plasma lipoproteins in CRF patients. They also came to the conclusion that total cholesterol is not substantially higher in CRF patients.[10]

Comparing patients to controls revealed a significant rise in serum triglycerides. Mean triglycerides in cases were 205.9 mg and 148 mg/dL in controls (p-value = 0.0001). This outcome agrees with the findings of Kimak et al., who showed a considerable rise in triglyceride, LDL, and Apo-B contents. Bhagwat et al. found that CRF patients had a high

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triglyceridemia of 232 mg/dL when compared to controls in another research (p-value 0.01). Another Indian research on dyslipidemia in patients with CRF and kidney transplantation by Shah et al. showed that individuals with CRF receiving conservative therapy had significantly higher triglyceride levels. These findings demonstrate that hypertriglyceridemia is a significant lipid anomaly in CRF patients.[11-12]

According to our study, LDL-C levels rose between patients and controls (139.4 vs. 127.2 mg/dL). According to statistics, this was not significant (0.1031). This was comparable to the Bhagwat research, which discovered that LDL-C increased in CRF patients compared to controls but did not significantly differ from controls. Results from the research by Kimak et al did not match those of ours. In their study, LDL-C levels among CRF patients significantly increased when compared to controls.10 In CRF, tiny compact particles that are highly vulnerable to oxidation predominate despite the fact that the total amounts of LDL are not much higher. Compared to bigger LDL substrates, these tiny particles are hypothesized to be more atherogenic.[13]

In contrast to controls, CRF sufferers had significantly lower levels of HDL in our research (39 vs. 60.7 mg/dL, p = 0.001). This was consistent with the findings of Bhagwat et al., who discovered that HDL-C levels in CRF groups were considerably low (2011) mg/dL (p-value less than 0.001). When compared to nonuremic people, patients with CKD often have lower plasma HDL-C concentrations.

Conclusion: In CRF, lipid abnormalities are frequent. There are no statistically significant changes in total cholesterol. When compared to normal, triglycerides statistically significantly rise in CRF instances. Although LDL-C is higher in CRF patients than in controls, this difference is not statistically significant. Patients with CRF exhibit a statistically significant decline in HDL-C compared to controls.

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Conflict of interest: Nil

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