Section: Biochemistry

Original Research Article

STUDY OF LIPID PROFILE IN CHRONIC KIDNEY DISEASE IN PRE-DIALYSIS PATIENTS OF KERALA – RETROSPECTIVE STUDY

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Abstract

Background: Chronic kidney diseases (CKD) encompass a spectrum of different pathophysiological processes associated with abnormal kidney function and progressive decline in GFR (glomerular filtration rate). In CKD, elevated atherosclerosis and the risk of increased dyslipidemia cause CVD morbidity and mortality. **Materials and Methods:** 120 CKD adult patients were studied and compared with 100 controlled group. Lipid profile was studied after a minimum 12-hour fast. About 10 ml of blood was collected from the median cubital vein and centrifuged at 5000 rpm for ten minutes, then a lipid profile was done by the VITROS slide method. The obtained results in both groups were noted and compared. **Result:** Biochemical parameters had significant p values ($p<0.001$) except serum sodium. Overall, dyslipidemia was present in 22 (18.3%) CKD and absent in 98 (81.6%). Of 120 CKD patients, 8 (6%) were in the 3rd stage, 36 (30%) were at stage 4th, and 76 (64%) were at the 5th stage. In correlation of lipid profile with GFR, TG, HDL, and VLDL had significant p-value ($p<0.001$); dialysis will prevent the complications of both CKD and CVD. **Conclusion:** It is confirmed that elevation of dyslipidemia in CKD patients is a bad prognosis because it drastically affects CVD and leads to morbidity and mortality. Early dialysis will prevent the complications of both CKD and CVD.

INTRODUCTION

Chronic kidney disease (CKD) encompasses a spectrum of different pathophysiologic processes associated with abnormal kidney function and glomerular filtration rate (GFR).[1] Cardiovascular diseases (CVD) are the leading cause of death irrespective of race and ethnicity and are mostly caused by cardiometabolic risk factors and chronic kidney disease.[2]

It is reported that the prevalence of CVD in CKD patients reaches up to 63%; hence, CVD and CKD are directly correlated, and CVD accounts for 63% of dialysis patients.[3] The prevalence of hypercholesterolemia ranges from 48% in CKD stages 1-2 to 80% in CKD stages 3-4. In addition to this, lipid abnormalities are principally present in CKD in hyperglyceridemia along with increased very low-density lipoprotein cholesterol (VLDL-C) and decreased HDL-C. Abnormal lipids and lipoproteins are more concentrated in CKD. Elevated triglyceride (TG) due to impaired activity of lipo-protein lipase (LPL) and direct inhibitory effect of various uremic toxins on enzymes involved in lipid metabolism Dyslipidemia has worsened CKD, which has increased CVD morbidity and mortality.[4] Hence an attempt is made to study the lipid profile in CKD in pre-dialysis patients.

MATERIALS AND METHODS

120 adult patients admitted at Al-Azhar Medical College and Super Specialty Hospital, Ezhalloor, Thodupuzha, Kerala-685605 were studied.

Inclusive Criteria

The patients confirmed having CKD (chronic kidney disease) and being above the age of 18 years and gave their consent in writing for study were selected for study.

Exclusion Criteria

Patients with HIV, hepatitis, terminal-stage cancer, or below 18 years of age and patients with stage V renal disease (CKD) on hemodialysis or patients with diabetes mellitus already on lipid-lowering drug therapy.

Methods: 120 chronic kidney disease patients were compared with 100 normal healthy volunteers (control group). Blood samples were drawn from the cubital fossa after a maximum of 12 hours of fasting. About 10 ml of blood was drawn and transfused to dried glass plain vials of serum were separated within 2 hours after collection and centrifuged at 5000 rpm

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for 10 minutes. The supernatant clear serum was then pipetted out and stored in dry, thin-walled vials at 40°C. The samples were analyzed on the same day. Study of lipid profile was done by the VITROS slide method.

The duration of the study was from June 2024 to January 2025.

Statistical Analysis: Various parameters were compared in chronic kidney disease and the controlled group (normal group) with the t-test, and GFR was correlated with the Pearson coefficient regression method. The statistical analysis was carried out using the SPSS method. The ratio of male and female was 2:1.

RESULTS

[Table 1] Comparison of Biochemical parameters in CKD patients and controlled groups

- Blood Urea: 204.62 (± 80.8) in CKD group, 14.8 (± 4.28) in controlled group, t test 23.4 and p<0.001
- S. creation: 8.62 (± 4.28) in CKD group, 0.75 (± 0.26) in controlled group, t test 18.3 and p<0.001
- Serum total protein: 6.05 (±0.58) in CKD group, 6.80 (± 0.42) in controlled group, t test 11.2 and p<0.001.
- Serum albumin: 3.38 (±0.50) in CKD group, 4.20 (± 0.30) in controlled group, t test 14.3 and p<0.001.
- Serum sodium: 138.50 (±6.10) in CKD group, 140.34 (± 5.16) in controlled group, t test 2.38 and p<0.001.
- Serum potassium: 5.60 (± 1.26) in CKD group, 4.24 (± 0.68) in controlled group, t test 9.68 and p<0.001.
- Serum calcium: 8.36 (± 1.24) in CKD patients, 8.96 (± 0.72) in controlled group, t test 4.2 and p<0.001.
- Sodium phosphorous: 7.33 (±2.12) in CKD patients, 3.64 (± 0.80) in controlled group, t test 16.4 and p<0.001.
- Haemoglobin: 7.58 (±1.30) in CKD group, 12.08 (± 1.62) in controlled group, t test 22.08 and p<0.001

[Table 2] Prevalence of individual and overall study of dyslipidemia in both CKD and controlled group

- TC: < 200 in 60 (50%), 73% in controlled, > 200 in 60 (50%) and 27 in controlled group.
- TG: <150 in 38 (31.6%) in CKD, 67% in controlled group, >150 82 (68.3%) in CKD, 33% in controlled group.
- HDL: <40, 88 (73.3%) in CKD group, 21% in controlled group, 40-60 in 32 (26.6%) in CKD group, 79% in controlled group.
- LDL: <130 in 70 (58.3%) in CKD group, 68% in controlled group, >130 in 50 (41.6%) in CKD group, 32% in controlled group.
- VLDL: <30 in 39 (32.5%) in CKD group, 88% in controlled group, >30 81 (67.5%) in CKD group, 12% in controlled group.

[Table 3] Study of profile in CKD patients at various stage

- 3rd Stage: Number of patients 8 (6.6%), 192.3 (± 20.2) TC, 114.2 (± 53.3) TG, 40.28 (± 4.40) HDL, 113.2 (±16.4) LDL, 26.30 (± 9.38) VLDL.
- 4th Stage: Number of patients 36 (30%) – 196.2 (± 40.23) TC, 150.2 (±65.49) TG, 38.15 (± 6.78) HDL, 117.5 (±35.12) LDL, 32.25 (± 12.40) VLDL.
- 5th Stage: Number of patients 76 (63.3%) – 206 (± 40.12) TC, 190.6 (±54.6) TG, 35.33 (± 4.8) HDL, 124.4 (±35.2) LDL, 38.11 (± 11.13) VLDL.

[Table 4] Correlation of lipid profile parameters with GFR –

- TG, HDL, VLDL, have significant correlation with GFR but TC, LDL, have insignificant correlation.

Table 1: Comparison of Biochemical parameters in CKD patients and controlled group.

Sl. No	Biochemical parameters	CKD group (120)	Controlled group (100)	t test	p value
1	Blood Urea	204.62 (± 80.8)	14.8 (± 4.28)	23.4	P<0.001
2	Serum creatinine	8.62 (± 4.28)	0.75 (± 0.26)	18.3	P<0.001
3	Serum total protein	6.05 (± 0.58)	6.80 (± 0.42)	11.2	P<0.001
4	Serum Albumin	3.38 (± 0.50)	4.20 (± 0.30)	14.3	P<0.001
5	Serum sodium	138.50 (± 6.10)	140.34 (± 5.16)	2.38	P<0.001
6	Serum potassium	5.60 (± 1.26)	4.24 (± 0.68)	9.68	P<0.001
7	Serum Calcium	8.36 (± 1.26)	8.96 (± 0.72)	4.2	P<0.001
8	Serum phosphorous	7.33 (± 2.12)	3.64 (± 0.80)	16.4	P<0.001
9	Haemoglobin	7.58 (± 1.30)	12.08 (± 1.62)	22.8	P<0.001

Table 2: Prevalencqe of Individual and overall study of Dyslipidemia in both controlled and CKD group

Lipid profile parameter	CKD group with percentage (120)			Controlled (100)
	Level	No. of patients	Percentage (%)	
TC	< 200	60	50	73
	> 200	60	50	27
TG	< 150	38	31.6	67
	> 150	82	68.3	33
HDL	< 40	88	73.3	21
	40-60	32	26.6	79
LDL	< 130	70	58.3	68
	> 130	50	41.6	32
VLDL	< 30	39	32.5	88

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	> 30	81	67.5	12
Overall prevalence	N	22	18.3	54
	Abs	98	81.6	46

Table 3: Study of lipid profile in CKD patients at various stages

CKD stage	No. of cases (120)	TC	TG	HDL	LDL	VLDL
1	0	--	--	--	--	--
2	0	--	--	--	--	--
3	8 (6.6%)	192.3 (± 20.2)	144.2 (± 53.30)	40.28 (± 4.40)	113.2 (± 16.4)	26.30 (± 9.38)
4	36 (± 30%)	196.2 (± 40.23)	150.2 (± 65.44)	38.15 (± 6.78)	117.5 (± 35.12)	32.25 (± 12.40)
5	76 (63.3%)	206 (± 40.12)	190.6 (± 54.6)	35.33 (± 4.8)	124.4 (± 35.2)	38.11 (± 11.13)

Table 4: Correlation of lipid profile parameter with GFR

		GFR	TC	TG	HDK	LDL	VLDL
GFR	Pearson correlation	1	0.100	0.304	0.322	0.106	0.273
	P value		0.25	0.001	0.001	0.244	0.001

TG, HDL and VLDL have significant correlation with GFR but TC, LDL have insignificant correlation

Note: Mean GFR = 11.75+7.93 ml/min/1.73 m^2

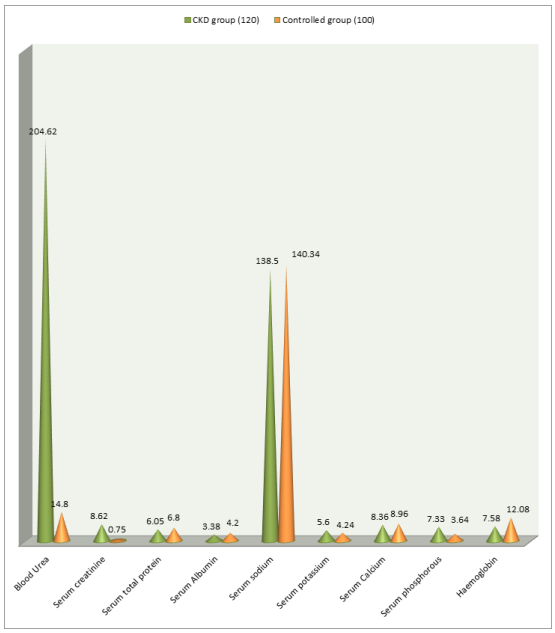

Figure 1: Comparison of Biochemical parameters in CKD patients and controlled group

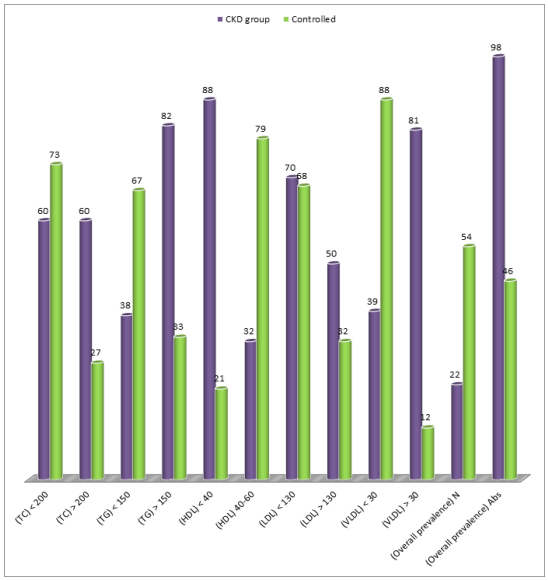

Figure 2: Prevalencqe of Individual and overall study of Dyslipidemia in both controlled and CKD group

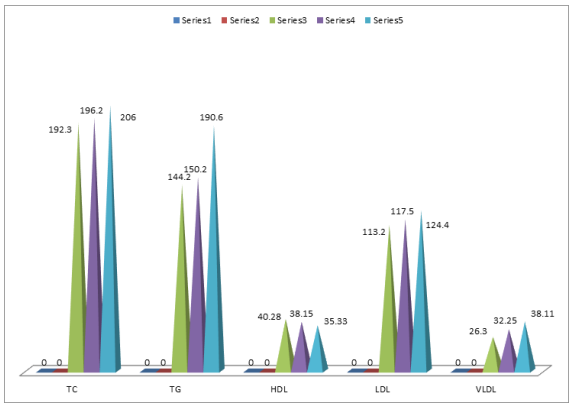

Figure 3: Study of lipid profile in CKD patients at various stages

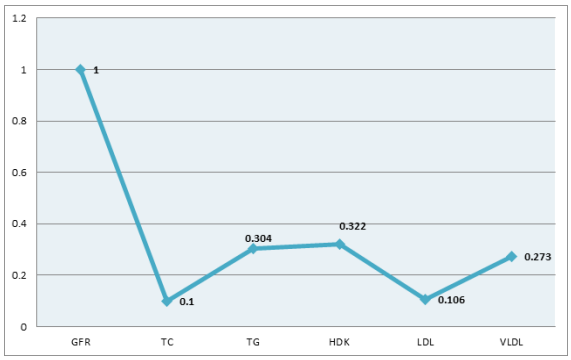

Figure 4: Correlation of lipid profile parameter with GFR

DISCUSSION

Present study of lipid profile in CKD in pre-dialysis patients of the Kerala population. The comparative study of biochemical parameters in CKD and healthy (controlled) groups had a significant p-value ($p<0.001$) [Table 1]. In the prevalence of individual and overall study, dyslipidemia was present in 22 (18.3%) patients and absent in 98 (81.6%) patients [Table 2]. In the study of lipid profile in CKD patients at various stages, 8 (6.6%) patients were in the 3rd stage, 36 (30%) in the 4th stage, and 76 (63.3%) in the 5th stage [Table 3]. In correlation of lipid profile parameters with GFR, TR, HDL, and VLDL have

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significant correlation with GFR, but TC and LDL have insignificant correlation [Table 4]. These findings are more or less in agreement with previous studies.[5-7]

Hyperlipidemia can potentially accelerate progression of renal disease by several mechanisms. First, resorption of fatty acids, phospholipids, and cholesterol contained in the filtered proteins (albumin and lipoproteins) by tubular epithelial cells can stimulate tubule-interstitial inflammation, foam cell formation, and tissue injury.[8] The second factor is that the accumulation of lipoproteins in the glomerular mesangium can promote matrix production and glomerulosclerosis.[9] In addition to this impaired HDL, medicated reverse cholesterol transport can further contribute to tissue injury by limiting the unloading of the excess cellular cholesterol and phospholipid burden. In fact, low plasma HDL had been identified as an independent risk factor for the progression of renal disease.[10] Moreover, hereditary lecithin cholesterol acyltransferase (LCAT) deficiency is associated with a marked reduction in HDL cholesterol, and impaired HDL-mediated reverse cholesterol transport results in progressive renal disease.[11]

It is reported that consumption of a high-fat diet exacerbates hyperlipidemia, whereas correction of hyperlipidemia attenuates the severity of glomerulosclerosis and tubulointestinal fibrosis in animal studies.[12] Moreover, pharmacological intervention aimed at normalization of HDL metabolism per se with no change in serum total cholesterol has been shown to retard the progression of renal disease in 5/6 nephrectomized rats.[13] Numerous factors contribute to atherogenic diathesis and high risk of cardiovascular disease in CKD. These include oxidative stress, inflammation, hypertension, and altered metabolism of lipids, carbohydrates, nitric oxide, calcium, and phosphate in CKD patients.

CONCLUSION

Dyslipidemia is a common cardiovascular risk factor for CKD in adult patients. Some lipid abnormalities, such as reduced HDL, elevated TG, and atherogenic risk, tend to increase with worsening renal function. Statins exert positive effects in CKD and renal transplant patients, where no advantage has been revealed in end-stage renal disease patients in terms of survival or cardiovascular morbidities. New hypolipidemic therapies lead to an additional lowering of cholesterol levels, but further studies are necessary to evaluate their potential application to CKD patients in order to improve clinical outcomes because the exact pathogenesis of dyslipidemia is still unclear.

Limitation of study: Owing to remote location of research centre, small number of patients lack of latest techniques we have limited finding and results.

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