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Research Article

ANALYTICAL STUDY OF LIPID PROFILE, SERUM PROTEINS, ELECTROLYTES IN CHRONIC KIDNEY DISEASE IN PRE-DIALYSIS PATIENTS IN TERTIARYCARE HOSPITAL IN PUNE MAHARASHTRA

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ABSTRACT

Background: Chronic Kidney Disease (CKD) causes irreversible damage to renal tissue resulting in decreased kidney function. It will affect the cardio-vascular system and leads to morbidity and mortality. Study of lipid profile in chronic kidney disease in pre-dialysis patients

Method: 150 CKD adult patients were studied nearly 2:1 M/F sex and gender matched and compared with 150 controlled group. Lipid profile was studied after a minimum 14 hour fasting. About 10 ml blood was collected from median cubital vein and centrifuged at 7000 rpm for fifteen minutes then lipid profile, Serum total cholesterol CHO-PAP, Serum triglycerides GPO-PAP, HDL Direct enzymatic, VlDL & LDL calculated, serum urea Urease (glutamate dehydrogenase (GLD), serum creatinine, (kinetic alkaline picrate) Serum Total Protein Biuret method, Albumin Bromocresyl Green (BCG), (Abbott C Chemistry integrated platform) on Artect Fully Automatic Machin. Electrolytes sodium (Na^+), Serum potassium (K^+) Calcium (Ca^{++}) done by Alinity ICT module indirect ion selective electrode method. Haemogram done with Modified cynmeth haemoglobin 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 27 (18%) CKD and absent in 123 (82%) CKD patients out 150, 9 (6%) patients were in 3rd stage, 45 (30%) were at stage-IV, 96 (64%) were at Vth stage. In correlation of lipid profile with GFR, TG, HDL and VLDL had significant p value ($p<0.001$).

Conclusion: Present pragmatic study interprets that, dyslipidemia progress with CKD. Early monitoring of lipid profile may help to control the progression of CKD and avoid morbidity and mortality of Chronic Kidne Disease patients.

INTRODUCTION

Chronic kidney disease (CKD) is associated with premature atherosclerosis and increased incidence of cardio vascular morbidity and mortality [(1)]. Several factors contribute to atherogenesis and cardio vascular disease in patients with CKD. The main risk factors are lipid disorders oxidative stress inflammation, physical inactivity, anaemia, hypertension, vascular calcification, endothelial dysfunction and depressed nitric oxide availability [(2)].

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Numerous studies have been conducted to compare the features and mechanism of CKD – induced dyslipidemia. In the plasma, lipid are carried by water soluble particles known as lipoproteins, which consists of nonpolar lipid core (triglycerides, cholesterol esters) surrounded by an envelope composed of specific apolipopratiens (apo) phosphorus lipid and other polar lipids. The plasma lipoproteins are commonly classified as either high density (HDL), low density (LDL), immediate or very low density (VLDL), lipoproteins according to their ultra centrifugation characteristics. Chylomicrons and VLDL serve of vehicles to transport triglycerides and cholesterol from the sites of absorption from intestine, liver. In contrast HDL serves as a vehicle to transport excess cholesterol from peripheral tissues to the liver for disposal. Hence attempt is made to evaluate the lipid profile to know the severity of CKD.

MATERIAL AND METHOD

150 adult patients admitted at Bharati Vidyapeeth Medical College tertiary care hospital, Pune-411043, Maharashtra population were studied.

Inclusive Criteria: The patients confirmed having CKD (chronic kidney disease) and above the age of 18 years.

Exclusion Criteria: Patients of HIV, hepatitis, terminal stage of cancer patient, below 18 years patients and stage of renal disease (CKD-stage V) on haemodialysis patients having Diabetes mellitus patients already on lipid lowering drug therapy.

Methods: 150 chronic kidney disease patients were compared with 150 normal healthy volunteers (controlled group). Blood samples were drawn cubital fossa after a maximum of 14 hour fasting. About 10ml of blood was drawn and transfused to dried glass plain vials serum was separated within 2 hours after collection and centrifuged at 7000 rpm for 10 minutes. The supernatant clear serum was then pipetted out and stored in dry thin walled vials at 40^{o}C. The samples were analysed on the same day. Study of lipid profile was done by Abbot Alinity C Chemistry integrated platform method. as per NCEP guidelines. Serum total cholesterol CHO-PAP, Serum triglycerides GPO-PAP, HDL Direct enzymatic, VlDL & LDL calculated, serum urea Urease (glutamate dehydrogenase (GLD), serum creatinine, (kinetic alkaline picrate) serum total protein, biuret method, albumin bromocresyl Green (BCG), (Abbott C Chemistry integrated platform). Electrolytes sodium (Na^{+}), serum potassium (K^{+}) calcium (Ca^{++}) done by Alinity ICT module indirect ion selective electrode method, Haemogram done with Modified cynmeth haemoglobin method The obtained results in both groups were noted and compared. The duration of study was January-2019 to January-2023.

Statistical analysis

Various parameters were compared in Chronic Kidney Disease and controlled group (normal group) with t test and GFR was correlated with Pearson co-efficient regression method. The statistical analysis was carried out in SPSS method. The ratio of male and female was 2:1.

Observation and Results

Table-1: Comparison of Biochemical parameters in CKD patients and controlled groups

- Blood Urea – 206.73 (±82.10) in CKD group, 14.10 (± 4.30) in controlled group, t test 28.06 and p<0.001
- Serum creation – 8.60 (±4.30) in CKD group, 0.75 (± 0.23) in controlled group, t test 22.2 and p<0.001
- Serum total protein – 6.06 (±0.60) in CKD group, 6.82 (± 0.44) in controlled group, t test 11.42 and p<0.001
- Serum Albumin – 3.36 (±0.52) in CKD group, 4.22 (± 0.32) in controlled group, t test 10.1 and p<0.001
- Serum. Sodium (Na^{+})- 139.60 (±6.11) in CKD group, 140.45 (± 5.18) in controlled group, t test 1.18 and p<0.23 (p value is insignificant)
- Serum Potassium (K^{+})- 5.62 (±82.10) in CKD group, 14.10 (± 4.30) in controlled group, t test 28.06 and p<0.001
- Serum Calcium (Ca^{++})- 8.38 (±1.26) in CKD group, 8.95 (± 0.75) in controlled group, t test 4.47 and p<0.001
- Serum Phosphorous (P) – 7.32 (±2.14) in CKD group, 3.65 (± 0.82) in controlled group, t test 19.01 and p<0.001
- Haemoglobin – 7.60 (±1.50) in CKD group, 12.05 (± 1.60) in controlled group, t test 22.08 and p<0.001

Note: (except serum sodium) all parameters have significant p value (p<0.001)

Table 1 Comparison of Biochemical parameters in CKD 150 and control !50 group patients

Sl. No	Biochemical parameters	CKD group 150	Controlled group 150	t test	p value
1	Blood Urea	206.73 (± 82.10)	14.10 (± 4.30)	28.6	P<0.001
2	Serum creatinine	8.60 (± 4.30)	0.75 (± 0.28)	22.2	P<0.001
3	Serum total protein	6.06 (± 0.60)	6.82 (± 0.44)	11.42	P<0.001
4	Serum Albumin	3.36 (± 0.52)	4.22 (± 0.32)	16.1	P<0.001
5	Serum sodium	139.60 (± 6.11)	140.45 (± 5.18)	1.18	p>0.23
6	Serum potassium	5.62 (± 1.28)	4.26 (± 0.70)	10.8	P<0.001
7	Serum Calcium	8.38 (± 1.26)	8.95 (± 0.75)	4.47	P<0.001
8	Serum phosphorous	7.32 (± 2.14)	3.65 (± 0.82)	19.01	P<0.001
9	Haemoglobin	7.60 (± 1.50)	12.05 (± 1.60)	22.08	P<0.001

Except serum sodium all value have significant p value (p<0.001)

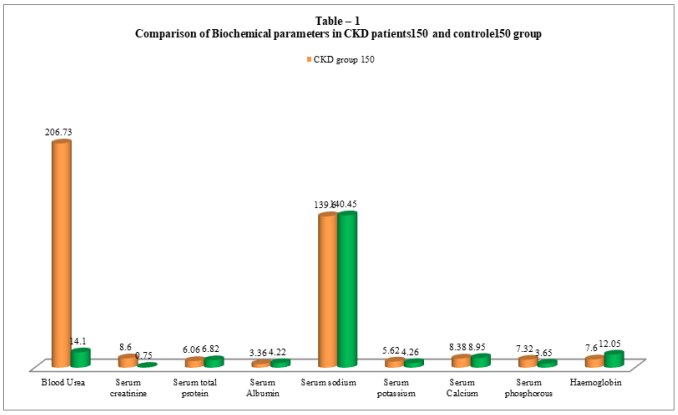

Table 2 Prevalence of individual and overall study of Dyslipidemia in both CKD and controlled group.

Lipid profile parameter

- Total Cholesterol (TC) – CKD<260 – 76 (50.6%), 74 in controlled, CDD>200 74 (49.3%), 26 in controlled group

- Triglycerides (TG) <150 – 48 (32%) in CKD, 68 in controlled group, >150 102 (68%) in CKD, 32in controlled group
- High Density Lipoprotein Good Cholestrol HDL <40 – 110 (73%) in CKD group, 22 in controlled group, 40-60 40 (26.6%) in CKD group, 78 in controlled group
- Low Density Lipoprotein Bad Cholesterol (LDL) <130 – 87 (58%) in CKD group, 69 in controlled group, >130 – 63 (42%) in CKD group, 31 in controlled group
- Very Low Density Lipoproteins (VLDL) <30 – 48 (32%) in CKD group, 89 in controlled group, >30 102 (68%) in CKD group, 11 in controlled group

Overall N (present) 27 (18%) in CKD Dyslipedemia, 6 in controlled group, Absent in 123 (82%) in CKD and 45 in controlled group.

Table 2 Prevalence of Individual and overall study of Dyslipidemia in both CKD Group and Control Group

Lipid profile parameter		CKD group with 150		Controlled group with 150
TC	<200	76	506	74
	>200	74	49.3	26
TG	<150	48	32	68
	>150	102	68	32
HDL	<40	110	73	22
	40-60	40	266	78
LDL	<130	87	58	69
	>130	63	42	31
VLDL	<30	48	32	89
	>30	102	68	11
Overall prevalence	N	27	18	55
	Ab	123	82	45

Table 3 Study of profile in CKD patients at various stage
Stage-I – NIL, Stage-II – NIL

Stage-III – Number of patients 9 194.6 (± 55.6) TG, 40.30 (± 5.40) HDL, 115.2 (±17.4) LDL, 26.4 (±10.62) VLDL
Stage-IV – Number of patients 45 – 198.4 (± 42.2) TC, 152.4 (±66.4) TG, 39.16 (± 6.80) HDL, 119 (±36.12 LDL, 32.46 (± 14.8) VLDL
Stage-V – 96 patients – 208 v42.15) TC, 192.8 (± 56.68) TG, 35.63 (± 4.82) HDL, 126.8 (±36.29) LDL, 39.22 (± 12.15) VLDL

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

CKD stage	No. of cases	TC	TG	HDL	LDL	VLDL
1	0	--	--	--	--	--
2	0	--	--	--	--	--
3	9 (6%)	194.6 (±22.20)	146.2 (±55.60)	40.30 (±5.40)	115.2 (± 17.48)	26.40 (±10.62)
4	45 (30%)	198.4 (±42.25)	152.4 (±66.49)	39.16 (±6.80)	119.6 (±36.12)	32.46 (±14.80)
5	96(±64%)	208 (±42.15)	192.8 (±56.68)	35.63 (±4.82)	126.8 (±36.29)	39.22 (±12.15)

Table-4: Correlation of lipid profile parameters with GFR

0.100 TC, 0.306 TG, 0.325 HDL, 0.108 LDL, 0.275 VLDL
P value was significant in TG, HDL and VLDL
Mean GFR 1175 (± 7.93) m1/min/1.73 m^2

Table 4 Correlation of lipid profile parameter with GFR

		GFR	TC	TG	HDK	LDL	VLDL
GFR	Pearson correlation	1	0.100	0.306	0.325	0.108	0.275
	P value		0.26	0.001	0.001	0.242	0.002

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

DISCUSSION

Present study of lipid profile in CKD patients from tertiary care hospital of pune Maharashtra population. In the comparison of biochemical parameters in CKD patients and controlled groups except serum sodum Na^+ all values were highly significant ($p<0.001$) (Table-1). Study of prevalence of Individual and overall study of dyslipidemia in both controlled group and CKD patients 27 (18%) had presences dyslipidemia and 23 (82%) had absence of dyslipidemia (Table-2). The study of lipid profile in CKD patients at various stage.

3^{rd} stage had 9 (6%), 4^{th} stage had 45 (30%), V^{th} stage had 96 (64%) CKD patients with elevated lipid profile (Table-3). In correlation of lipid profile parameters with GFR – TG, HDL and VLDL have significant correlation with GFR mean GFR = 11.75 (± 7.93) m1/,in/1.73 m2 (Table-4). These findings are more or less agreement with previous studies [(5)(6)(7)].

Hyperlipidemic can potentially accelerate progression of renal disease by several mechanisms. First resorption of fatly acids phospholipids, and cholesterol contained in the filtered proteins (albumin and lipoproteins) by tubular epithelial cells can stimulate tubule-interstial inflammation, foam cell formation and tissue injury [(8)]. Second factor is accumulation of lipoproteins in glomerular mesangium can promote matrix production and glomerulo-sclerosis [(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 phospho lipid burden. In fact low plasma HDL had been identified as an independent risk factor for progression of renal disease [(10)]. Moreover of hereditary Lecithin cholesterol acyltransferase (LCAT) deficiency, this is associated with marked reduction in HDL: cholesterol and impaired HDL mediated reverse cholesteroll transport results in progressive renal disease (11).

It is reported that, consumption of high fat diet exacerbates hyper-lipidemia where as correction of hyperlipidemia attenuates the severity of glomerulo sclerosis and tubuto intestinal 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 nephrectomised rats [(15)]. Numerous factors contribute to atherogenic diathesis and high risk of cardio vascular disease in CKD. These include oxidative stress inflammation, hypertension and altered metabolism of lipids carbohydrate Nitric oxide calcium and phosphate in CKD patients.

SUMMARY AND CONCLUSION

Dyslipidemia is a common cardio vascular risk factor CKD in adult patients. Some lipid abnormalities such as reduced HDL elevated TG and atherogenic risk leads to increase with worsening renal function. Statin exert positive effects in CKD and renal transplanted patients, where no advantage have been revealed in end stage renal disease patients in terms of survival or cardio vascular morbidities. New hypolipidemic therapies lead to an additional lowering cholesterol levels but further studies are necessary to evaluate their potential application to

CKD patients in order to improve clinical outcomes because exact pathogenesis of dyslipidemia is still un-clear.

Limitation of study – Owing to tertiary location of research centre, small number of patients and lack of latest technologies, we have limited findings and results.

- This research paper is approved by Ethical committee of Bharati Vidyapeeth (deemed to be university) medical college & Hospital Pune-411043 (Maharashtra)
- No conflict of Interest.
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