



CHRONIC KIDNEY DISEASE INDUCED CHANGES IN LIPID PROFILE, SERUM PROTEINS, ELECTROLYTES IN PRE-DIALYSIS PATIENTS IN TERTIARY CARE HOSPITAL IN WESTERN MAHARASHTRA.

Clinical Biochemistry

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ABSTRACT

Chronic Kidney Disease (CKD) represents a significant public health concern globally, with its prevalence steadily increasing. This study aimed to investigate the alterations in lipid profile, serum proteins, and electrolytes in pre-dialysis CKD patients at a tertiary care hospital in Pune, Maharashtra. A retrospective analysis was conducted on data collected from pre-dialysis CKD patients attending the nephrology department between [insert timeframe]. Lipid profile parameters including total cholesterol, HDL cholesterol, LDL cholesterol, and triglycerides were measured using standard biochemical assays. Serum protein levels were assessed, and electrolyte imbalances, including sodium, potassium, calcium, and phosphorus, were analyzed. Preliminary findings revealed significant alterations in lipid profile parameters among pre-dialysis CKD patients compared to healthy individuals. Elevated levels of total cholesterol, LDL cholesterol, and triglycerides, along with decreased HDL cholesterol levels, were observed. Furthermore, disruptions in serum protein levels, such as albumin and globulin, were noted, indicative of kidney dysfunction. Electrolyte disturbances, particularly hyperkalemia and hyperphosphatemia, were prevalent in this cohort. This study underscores the intricate relationship between CKD and metabolic derangement, emphasizing the need for comprehensive management strategies to mitigate cardiovascular risk factors and delay disease progression in pre-dialysis CKD patients. Further prospective studies are warranted to elucidate the underlying mechanisms driving these alterations and evaluate the effectiveness of targeted interventions in improving clinical outcomes in this population. **Methods:** 150 CKD adult patients were studied and compared with 150 controlled group. Lipid profile was studied after a minimum 12 hour fasting. About 10 ml blood was collected from median cubital vein and centrifuged at 5000 rpm for ten minutes then lipid profile was done by 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 dyslipidaemia 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 proved that, dyslipidaemia progress with CKD. Early monitoring of lipid profile may help to control the progression of CKD and avoid morbidity and mortality of CKD patients.

KEYWORDS

Dyslipidaemia, cardiovascular, chronic kidney disease, Vitro slide method, Lipid Profile

INTRODUCTION

CKD typically stands for chronic kidney disease (CKD), a condition characterized by gradual loss of kidney function over time. It's often associated with aging, high blood pressure, diabetes, or other chronic conditions. Managing CKD usually involves lifestyle changes, medications, and sometimes dialysis or kidney transplantation in advanced stages.

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, anemia, hypertension, vascular calcification, endothelial dysfunction and depressed nitric oxide availability⁽²⁾.

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 apolipoproteins (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 centrifuge 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. These include: **Total cholesterol:** This is a measure of all the cholesterol in your blood, including both "good" (HDL) and "bad" (LDL) cholesterol. **HDL cholesterol:** High-density lipoprotein cholesterol, often referred to as "good" cholesterol, helps remove LDL cholesterol from the bloodstream, thus lowering the risk of heart disease **cholesterol:** Low-density lipoprotein cholesterol, often referred to as "bad" cholesterol, can build up in the walls of arteries, leading to atherosclerosis and increasing the risk of heart disease and stroke. **Triglycerides:** Triglycerides are a type of fat found in your blood. Elevated triglyceride levels are also associated with an increased risk of heart disease.

A lipid profile is typically done as part of a routine check-up to assess your risk of cardiovascular disease. It helps your healthcare provider determine if you have unhealthy cholesterol levels and if you're at risk for heart disease or stroke. Based on the results, your healthcare provider may recommend lifestyle changes, such as diet and exercise, or medications to help manage your lipid levels.

MATERIALS AND METHODS

Materials: 150 adult patients admitted at Bharati Vidyapeeth Medical College hospital, Pune-411043 Maharashtra were studied.

Inclusion Criteria:

The patients confirmed having CKD (chronic kidney disease) and above the age of 18 years either gender.

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 12 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 5000 rpm for 10 minutes. The supernatant clear serum was then pipetted out and stored in dry thin-walled vials at 40°C. The samples were analysed on the same day. Study of lipid profile was done by VITROS slide method. 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 Creatinin – 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 – 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 – 5.62 (± 82.10) in CKD group, 14.10 (± 4.30) in controlled group, t test 28.06 and $p < 0.001$
- Serum Calcium – 8.38 (± 1.26) in CKD group, 8.95 (± 0.75) in controlled group, t test 4.47 and $p < 0.001$
- Serum Phosphorous – 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-2: Prevalence of individual and overall study of Dyslipidaemia in both CKD and controlled group.

Lipid profile parameter

- TC – CKD $< 260 - 76$ (50.6%), 74 in controlled, CDD > 200 74 (49.3%), 26 in controlled group.
- TG $< 150 - 48$ (32%) in CKD, 68 in controlled group, > 150 102 (68%) in CKD, 32 in controlled group
- HDL $< 40 - 110$ (73%) in CKD group, 22 in controlled group, 40-60 40 (26.6%) in CKD group, 78 in controlled group
- LDL $< 130 - 87$ (58%) in CKD group, 69 in controlled group, $> 130 - 63$ (42%) in CKD group, 31 in controlled group
- 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 Dyslipidaemia, 6 in controlled group, Absent in 123 (82%) in CKD and 45 in controlled group.

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-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) ml/min/1.73 m²

DISCUSSION

Current study of lipid profile in CKD patients of Maharashtra population. In the comparison of biochemical parameters in CKD patients and controlled groups except serum sedum all values were highly significant ($p < 0.001$) (Table-1). Study of prevalence of Individual and overall study of dyslipidaemia in both controlled group and CKD patients 27 (18%) had presences dyslipidaemia and 23 (82%) had absence of dyslipidaemia (Table-2). The study of lipid profile in CKD patients at various stage.

Stage III had 9 (6%), Stage IV had 45 (30%), Stage V had 96 (64%) CKD patients with elevated lipid profile (Table-3). In correlation of lipid profile parameters with GFR – TR, HDL and VLDL have significant correlation with GFR mean GFR = 11.75 (± 7.93) ml/min/1.73 m² (Table-4). These findings are more or less agreement with previous studies⁽⁵⁾⁽⁶⁾⁽⁷⁾.

Hyperlipidaemic 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⁾.

Second factor is accumulation of lipoproteins in glomerular mesangium can promote matrix production and glomeruli-sclerosis⁽⁹⁾. In addition to this impaired HDL mediated 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 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 cholesterol transport results in progressive renal disease (11).

It is reported that, consumption of high fat diet exacerbates hyperlipidaemia whereas correction of hyperlipidaemia attenuates the severity of glomeruli sclerosis and tubule- 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

Dyslipidaemia is a common cardio vascular risk factor CKD in adult patients. Some lipid abnormalities such as reduced HDL elevated TG and atherogenic risk tends 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 level but further studies are necessary to evaluate their potential application to CKD patients in order to improve clinical outcomes because exact pathogenesis of dyslipidaemia 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 Pune-411043 (Maharashtra)
- No conflict of Interest
- Self-Funding

Table – 1

No. of CKD patients: 150

Comparison of Biochemical parameters in CKD patients and controlled group

Sl. No	Biochemical parameters	CKD group 150	Controlled group 100	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 Dyslipidaemia in both CKD group and controlled group

Lipid profile parameter	CKD group with % 150			Controlled group with 100%
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 lipid profile in CKD patients at various stages

CKD Stage	No. of cases	TC	TG	HDL	LDL	VLDL
I	0	--	--	--	--	--
II	0	--	--	--	--	--
III	9 (6%)	194.6 (±22.20)	146.2 (±55.60)	40.30 (±5.40)	115.2 (± 17.48)	26.40 (±10.62)
IV	45 (30%)	198.4 (±42.25)	152.4 (±66.49)	39.16 (±6.80)	119.6 (±36.12)	32.46 (±14.80)
V	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 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 m²

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