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To Analyse the Pattern of Lipid Profile in Patients of Chronic Kidney Disease.

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ABSTRACT

Background: Chronic kidney disease (CKD), is a progressive and irreversible deterioration in the function of kidney over months to years,2 which is defined as"glomerular filtration rate (GFR) lesser than 60 ml/min with protenuria for 3 months".3 It leads to various biochemical disturbances and numerous clinical symptoms and signs.4Some of the associated changes are abnormalities in haematology parameters, heart related issues, disturbances of gastrointestinal tract, neurologic disorder, osteodystrophy, skin related problems and sexual dysfunction. Dyslipidemias are a common among CRF patients. The disturbances of lipoprotein metabolism are seen in early stages of CRF and can follow decrement pattern that follows the renal function deterioration. Aim and Objectives This study was aimed to analyse the pattern of lipid profile among the dif. **Methods:** The study was cross-sectional observational study conducted on 100 patients either attending or being admitted in the department of Internal Medicine, TMMC & RC, TMU , Moradabad, U.P., INDIA, which were in line with the inclusion criteria. **Result & Conclusion:** Our results indicated that patients undergoing CRF show important abnormalities of lipid metabolism which could contribute to atherosclerosis and cardiovascular disease and may increase the morbidity and mortality in these patients.

Keywords: Chronic kidney disease, lipid profile.

INTRODUCTION

Renal failure refers to "a condition, in which there is a loss of normal function, of kidneys, due to various factors, which include infections, auto-immune diseases, diabetes and other endocrine disorders, cancer, and toxic chemicals. In chronic renal failure (CRF), the deterioration of function of kidney gradually occurs and leading to accumulation of associated problems over years. This condition cannot be reversed; however, its progress can be slowed down and eventually, leads to the development of the symptoms of end-stage renal failure. It shares many similar risk factors, with cardiovascular diseases such as Diabetes and hypertension.[1]

Chronic kidney disease (CKD), is a progressive and irreversible deterioration in the function of kidney over months to years,[2] which is defined as "glomerular filtration rate (GFR) lesser than 60 ml/min with protenuria for 3 months".[3] It leads to various biochemical disturbances and numerous clinical symptoms and signs.[4]

Some of the associated changes are abnormalities in haematology parameters, heart related issues, disturbances of gastrointestinal tract, neurologic disorder, osteodystrophy, skin related problems and sexual dysfunction.5The difference from acute disease, in that, the decrease occurs over a period of 3 months.

Dyslipidemias are a common among CRF patients. The disturbances of lipoprotein metabolism are seen in early stages of CRF and can follow decrement pattern that follows the renal function deterioration. Literature has shown that dyslipidemias can lead to Cardiovascular disease, and renal function worsening.[6]

The lipid abnormalities, characteristically, reported among CRF patients are increased triglyceride levels, normal or reduction in total cholesterol (TC), decrease in levels of HDL, normal LDL levels. Progressive CRF, can not only lead to End stage renal disease (ESRD), but is also related to higher cardiovascular morbidity & mortality. Due to significant role of plasma lipids in the pathogenesis of atherosclerosis and ischemic heart disease, it becomes worthy for studying the role of various lipid fractions in CRF patients. Main cause of death is CVD among ESRD patients and exclusively, among hemodialysis compared to transplantation patients.[6]

Abnormal amount of lipids decline lead to disturbance at a fast rate of kidney function and leads to RRT among patients with CKD.[7,8] The

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mechanism is not known precisely, but it has been suggested that mesangial cells bind and take up oxidized LDL, which, leads to injury of mesangial, epithelial and endothelial cells by favouring recruitment of inflammatory cells such as macrophages which release cytokines, chemokines and growth factors,[9,10] leading to glomerulosclerosis, thereafter.[11] Increase in cholesterol and triglycerides results in injury to podocytes and sclerosis of mesangial cells, followed by glomerulosclerosis.

Disturbance in lipoproteins, is has been a feature of Chronic renal failure, along with cholesterol and triglycerides to be raised, and PUFAs to be lesser. All these abnormalities have been identified to be a sole risk factor for atherosclerosis,[12-15] with poor results with dialysis treatment.[16] An increased plasma homocysteine concentration is more among hemodialysis patients,[17,18] and also considered to be risk factor for atherosclerotic complications of ESRD.[19-22]

CKD is associated with increase in triglyceride level, due to increase in VLDL, chylomicrons, and its remnants. Increase in triglycerides is due to delay in catabolism and the increase in triglyceride-rich lipoproteins from liver. Catabolism delay is most common mechanism for increase in triglyceride-rich lipoprotein among CKD patients, which results from reduction in action of triglyceride lipase of liver, along with peripheral lipoprotein lipase.[24,25]

Literature has shown that, more small dense LDL levels, among non-dialysis-dependent CKD patients than healthy subjects and shown that small dense LDL to be a risk factor for CVD. Due to significant modification in turn over of lipid components, lipoproteins circulation time is more. Thus, lipoproteins, are at risk of post-ribosomal modification, including glycation, oxidation, and carbamylation. These modified lipoproteins bind less likely to classic LDL receptors and scavenger receptors capture me, leading to uremia, on macrophage surface. High affinity for macrophages results in the accumulation of cholesterol and the formation of foam cells in the vascular walls, finally resulting in the development of accelerated atherosclerotic plaques.[26,27]

Abnormalities in the lipid metabolism are found to be most common among end stage renal failure patients. Triglycerides and high cholesterol components affect the blood circulation pool by formation of fat plaques on major blood vessels.[1]

Keeping this idea in the centre of present work, we conducted a hospital-based study on patients with proven CKD in a tertiary care centre, Moradabad, Uttar Pradesh, India to assess lipid profile and its effect on dialysis and non-dialysis patients.

Aim and Objectives

This study was aimed to analyse the pattern of lipid profile among the different stages of CKD in a tertiary care centre, Moradabad, U.P, India.

MATERIALS AND METHODS

Study Setting

The study was conducted on 100 patients either attending or being admitted in the department of Internal Medicine, TMMC & RC, TMU , MORADABAD, U.P., INDIA, which were in line with the inclusion criteria.

Study Design

The present study was a cross-sectional observational study.

Selection of Subjects

An informed written consent will be obtained from all the patients willing to participate in the study on an Informed consent form (ICF) as per the guidelines of Institutional Ethical Committee (IEC).To be eligible for the study the patient had to fulfill the following inclusion and exclusion criterias:

Inclusion criteria

- Glomerular Filtrartion Rate(GFR)<60 ml/min
- Age more than 18 years – 60 years

Exclusion criteria

- Patient not giving consent for the study
- Obese Patients (BMI >30)
- Known Case of Acute Kidney Injury
- Known Case of Diabetes Mellitus
- Known Case of Ischemic heart disease
- Known Case of Hypothyroidism
- Taking Drugs that effect Lipid Profile

Diagnostic criteria for dyslipidemia

- Criteria for dyslipidemia will be decided according to National Cholesterol Education Programme (ATP III) guidelines;

Total Cholesterol	<200mg/dl	200-239mg/dl	>240mg/dl
Triglyceride	<150mg/dl	150-249mg/dl	250-499/≥500mg/dl
LDL	<100/100-129mg/dl	130-159mg/dl	≥160mg/dl

RESULTS

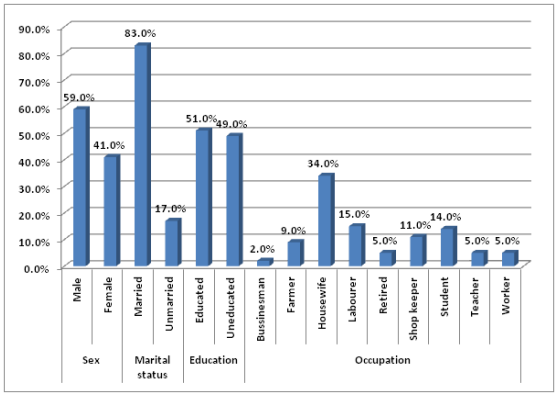


Table 1: Demographic characteristics of the study population

		Frequency	Percentage
Sex	Male	59	59.0%
	Female	41	41.0%
Marital status	Married	83	83.0%
	Unmarried	17	17.0%
Education	Educated	51	51.0%
	Uneducated	49	49.0%
Occupation	Businessman	2	2.0%
	Farmer	9	9.0%
	Housewife	34	34.0%
	Labourer	15	15.0%
	Retired	5	5.0%
	Shop keeper	11	11.0%
	Student	14	14.0%
	Teacher	5	5.0%
	Worker	5	5.0%

There were 59 (59.0%) males and 41 (41.0%) females.
There were 83 (83.0%) Married and 17 (17.0%) Unmarried subjects.
There were 51 (51.0%) educated and 49 (49.0%) Uneducated subjects.
There were 2 (2.0%) Businessman, 9 (9.0%) Farmers, 34 (34.0%) Housewives, 15 (15.0%) Labourers, 5 (5.0%) Retired subjects, 11 (11.0%) Shop keepers, 14 (14.0%) Students, 5 (5.0%) Teachers and 5 (5.0%) Workers.

Table 2: Minimum, Maximum, Mean and Standard Deviationof study population for Age (in years), Weight (in kgs), Height (in cms) and BMI

	Minimum	Maximum	Mean	Std. Deviation
Age (in years)	18	60	42.45	16.24
Weight (in kgs)	52	72	60.47	6.33
Height (in cms)	160	172	166.52	3.33
BMI	19.1	24.6	21.76	1.64

The mean Age (in years) was 42.45±16.24, Weight (in kgs) was 60.47±6.33, Height (in cms) was 166.52±3.33 and BMI was 21.76 ±1.64.

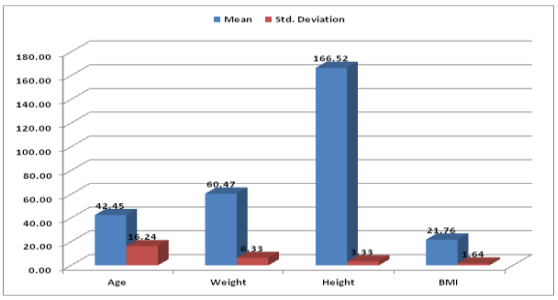


Table 3: Distribution of Urine examination among the study population

Urine examination		Frequency	Percentage
Albumin	-	73	73.0%
	+	6	6.0%
	++	3	3.0%
	+++	18	18.0%
Pus cells	0	73	73.0%
	1-2	9	9.0%
	6-8	3	3.0%
	8-10	6	6.0%
	15-20	3	3.0%
	35-40	3	3.0%
	OCC	3	3.0%

Albumin was found to be negative in 73 (73.0%) patients, + in 6 (6.0%), ++ in 3 (3.0%) patients and +++ in 18 (18.0%) patients.
Pus cells were found to be negative in 73 (73.0%) patients, 1-2 cells were found in 9 (9.0%)patients, 6-8 cells were found in 3 (3.0%)patients, 8-10 cells were found in 6 (6.0%), 15-20 cells were found in 3 (3.0%)patients, 35-40 cells were found in 3 (3.0%) patients and OCC were found in 3 (3.0%) patients.

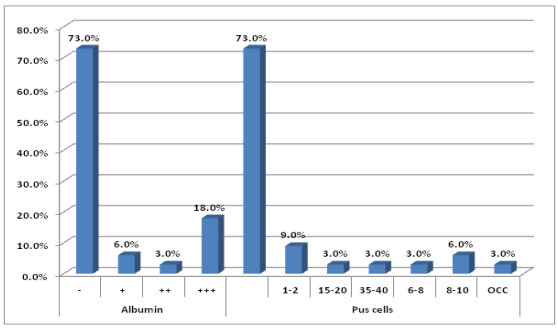


Table 4: Descriptive characteristics according toRenal function tests

Renal function test	Minimum	Maximum	Mean	Std. Deviation
Urea	64	297	157.46	52.14
Creatinine	2.7	19.7	8.34	3.63
Uric acid	3.5	135.0	10.96	22.02
Calcium	4.6	8.8	7.16	0.98
Phosphorous	3.7	108.0	10.38	17.37
Alkaline phosphatase	55	213	104.10	41.51
Sodium	116	146	133.19	8.47
Potassium	2.90	7.40	5.06	1.16
Chloride	74.0	119.0	99.83	11.83

The mean Urea level was 157.46±52.14, Creatinine level was 8.343.63, Uric acid level was 10.96±22.02, Calcium level was 7.16±0.98, Phosphorous level was 10.38±17.37, Alkaline phosphatase level was 104.10±41.51, Sodium level was 133.19±8.47, Potassium level was 5.06±1.16 and Chloride level was 99.83±11.83.

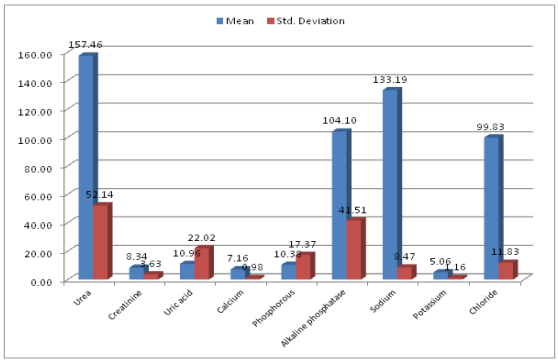
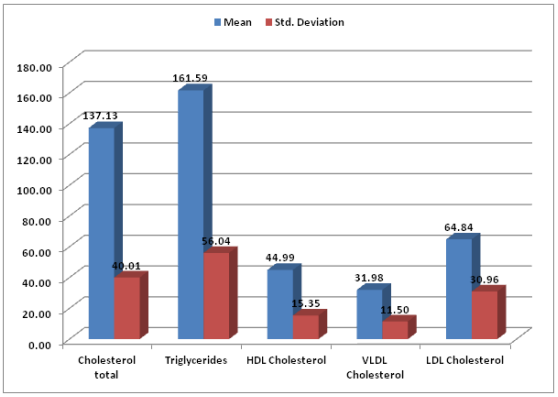


Table 5: Descriptive characteristics according to Cholesterol total, Triglycerides, HDL Cholesterol, VLDL Cholesterol and LDL Cholesterol

	Minimum	Maximum	Mean	Std. Deviation
Cholesterol total	78	227	137.13	40.01
Triglycerides	56	276	161.59	56.04
HDL Cholesterol	18	90	44.99	15.35
VLDL Cholesterol	11.2	55.2	31.98	11.50
LDL Cholesterol	19.4	135.2	64.84	30.96

The mean total Cholesterol level was 137.13 ±40.01, Triglycerides level was 161.59±56.04, HDL Cholesterol level was44.99±15.35, VLDL Cholesterol level was31.98±11.50 and LDL Cholesterol level was64.84±30.96.



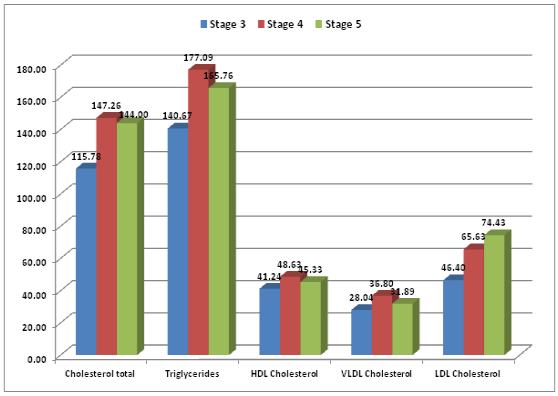


Table 6: Comparison of mean Total Cholesterol, Triglycerides, HDL Cholesterol, VLDL Cholesterol and LDL Cholesterol was compared between Stage 3, 4 and 5 chronic kidney disease

	Stage 3		Stage 4		Stage 5		F-value	p-valuea	Post-hoc comparisonsb
	Mean	SD	Mean	SD	Mean	SD			
Cholesterol total	115.78	43.00	147.26	26.77	144.00	39.79	5.840	0.004*	4,5 > 3
Triglycerides	140.67	49.56	177.09	63.73	165.76	53.36	3.018	0.044*	4,5 > 3
HDL Cholesterol	41.24	20.41	48.63	10.05	45.33	13.99	1.478	0.233	N/A
VLDL Cholesterol	28.04	10.03	36.80	13.47	31.89	10.62	3.808	0.026*	4,5 > 3
LDL Cholesterol	46.40	30.91	65.63	10.50	74.43	33.15	8.249	0.001*	4,5 > 3

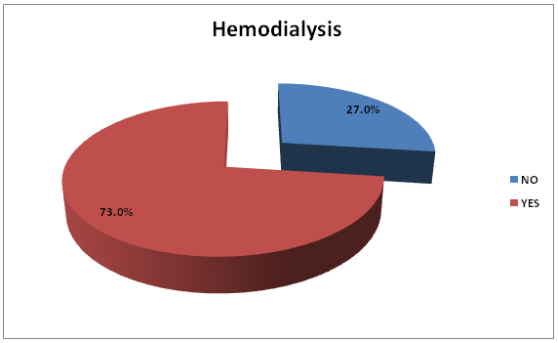
aOne-way ANOVA test bpost-hoc bonferroni test * Significant difference

The mean Total Cholesterol, Triglycerides, HDL Cholesterol, VLDL Cholesterol and LDL Cholesterol was compared between Stage 3, 4 and 5 chronic kidney disease using the One-way ANOVA test with post-hoc bonferroni test for inter-group comparisons. The mean Total Cholesterol, Triglycerides, VLDL Cholesterol and LDL Cholesterol was significantly more among Stage 4 and 5 chronic kidney disease in comparison to Stage 3 chronic kidney disease.

Table 7: Distribution of Haemodialysis among study population

Haemodialysis	Frequency	Percentage
No	27	27.0%
Yes	73	73.0%
Total	100	100.0%

Haemodialysis was found among 73 (73.0%) subjects.



DISCUSSION

This study was a hospital-based study on patients with proven CKD in a tertiary care centre, Moradabad, Uttar Pradesh, India to assess the pattern of lipid profile and its impact on dialysed and non-dialysed patients.

CRF is a health problem across the world and major cause of morbidity and mortality in developed countries. CRF patients are at more risk for cardiovascular disease and cerebrovascular disease and are more likely to die because of them. CRF leads to early atherosclerosis and increase inchances of cardiovascular morbidity and mortality.[32] Many factors are responsible for atherogenesis and CVD among CRF patients, with, most prominent being, dyslipidemias.[33] Chronic renal failure, per se, primarily affects the metabolism of high-density lipoprotein (HDL) and triglyceride (TG)-rich lipoproteins.[34]

Age-wise distribution of study population

In our study, the mean age of the study population was 42.45±16.24 years. This was similar to the study by Shah et al,[28] Group 1 which was controls were30±5 years old, Group 2 which was CRF patients treated conservatively were 49±17 years old, Group 3 which was ESRD patients on HD for at least 3 months were 53±9 years old, Group 4 which was 3 months post-renal transplant patients were31±11 years old and Maheshwari et al,[30] maintenance haemodialysis (MHD) and control groups had a mean age of 47.88 ± 13.92 and 54.56 ± 11.16 years respectively.

Gender-wisedistribution of study population

The present study included 59.0% males and 41.0% females with more number of males than females.The study by Maheshwariet al30 included 50 patients with end-stage renal disease on maintenance hemo-dialysis (MHD) which comprised of 31 males and 19 females.

Distribution of the patients according to presence or absence of haemodialysis

Our study included 73.0% patients with haemodialysis and 27% patients without hemodialysis. The study by Henning et al included 40 non-dialyzed patients with chronic renal disease and in 50 patients with end-stage renal disease requiring chronic maintenance hemodialysis.[29]

Mean Cholesterol total, Triglycerides, HDL Cholesterol, VLDL Cholesterol and LDL Cholesterol

In the present study, the mean total Cholesterol level was 137.13±40.01, Triglycerides level was 161.59±56.04, HDL Cholesterol level was 44.99±15.35, VLDL Cholesterol level was 31.98±11.50 and LDL Cholesterol level was 64.84±30.96. Almost similar results were reported by Mohanraj et al,[31] mean triglyceride of the study group was found to be 197.26 mg/dl (178.18 mg/dl. control group) and Lodh et al,[35] the lipid profile was also dearranged with statistically significant elevation in total cholesterol and triglyceride levels and significantly lower HDL levels as compared to the controls.

"Decreased triglyceride clearance from the plasma due to inhibition of both hepatic and lipoprotein lipase may lead to hypertriglyceridemia.[37] The reduced catabolism is likely due to the decreased activity of LPL and hepatic triglyceride lipase, which cleaves triglycerides into Free Fatty Acid (FFA) for energy production or storage".

"Hypertriglyceridemia leads to alteration of size and composition of HDL and LDL. Hypertriglyceridemia can lead to an increase in VLDL secretion from liver which leads to activation of Cholesteryl Ester Transfer Protein (CETP). CETP transfers triglycerides to LDL and HDL leading to formation of triglyceride rich LDL and HDL. Hepatic triglyceride lipase hydrolyses the triglyceride content of the HDL and LDL particles leading to formation of a subfraction called small dense LDL and HDL. Small dense LDL (sdLDL) has low affinity for LDL receptor, can penetrate arterial wall easily and are more susceptible to oxidation. Oxidized sdLDL is highly atherogenic which increases the risk of cardiovascular diseases".[38]

"Increase in VLDL cholesterol in CKD are mainly due to their reduced clearance as well as insulin resistance driven over production of VLDL.[34,39] Maheshwari et al.,[30] reported hypertriglyceridemia and low HDLc and elevated lipoprotein-a, which could contribute to atherosclerosis and cardiovascular disease that may increase the morbidity and mortality in patients on maintenance haemodialysis".

"One of the commonly seen impairment of lipid metabolism in the CRF patient group, which also includes the HD patients, is a decrease in HDL cholesterol and impaired HDL metabolism appear in the form of decreased Apo AI, impair HDL maturation.[36,40] Another reason for lowered HDL and impairment in its metabolism is Lecithin-Cholesterol acyl transferase (LCAT deficiency)".[41,42]

Mean Total Cholesterol, Triglycerides, HDL Cholesterol, VLDL Cholesterol and LDL Cholesterol was compared between Stage 3, 4 and 5 chronic kidney disease

In our study, the mean Total Cholesterol, Triglycerides, VLDL Cholesterol and LDL Cholesterol was significantly more among Stage 4 and 5 chronic kidney disease in comparison to Stage 3 chronic kidney disease.

This was similar to the findings by Rao et al,[40] there was hypertriglyceridemia in group 1 (stage I and II patients with a GFR between 60 and 119 ml/min/1.73m2) and group 2 (stage III and IV patients with a GFR between 15 and 59 ml/min/1.73m2) patients when compared to controls and Mohanraj et al,[31] there were elevated levels of serum total and LDL cholesterol in stage 3 and 4 CKD.

Elevated triglyceride levels are due to impaired activity lipoprotein lipase (LPL) and direct inhibitory effect of various uremic 'toxins' on the enzymes involved in lipid metabolism,[43,44] represent the most important pathophysiological mechanisms underlying the development of hypertriglyceridemia in renal failure.

Studies in India, does not show higher cholesterol in ESRD early stages, cholesterol being at usual or lesser level, whereas western studies, CKD groups to be having higher cholesterol levels. This could be due to difference in dietary habits.[45] In Our study, we could see that higher cholesterol was significantly reported in Stage 4 and 5 chronic kidney disease. Whereas Michael et al found contrasting results with low levels of cholesterol in ESRD patients,[46] statistically significantly.

"Because of hepatic apo AI genes being down-regulated, Apo AI, which mainly activates lecithin cholesterol acyltransferase, in CKD is lesser causing LACT working to be reduced, which leads to esterification of cholesterol being lesser and maturation of HDL is disturbed. HDL levels being lesser is due to non-LACT activity in CKD.[47] In the study by Rao et al, non-significantly HDL cholesterol was statistically lesser in groups 2 and 3 than controls".

"Disturbances of lipids in CKD may have huge effect, chances for CVD at a special note.[49] The study by Khatiwada et al,[48] suggested the significant association between the presence of cardiovascular disease with hypercholesterolemia and high LDL cholesterol in CKD patients. Our study reveals that CKD progression is strongly associated with CVD prevalence. Stage 4 and stage 5 patients had higher risk for having CVD as compared to stage 3 patients. This study did not report any significant increase in dyslipidemia cases during CKD progression. The independent role of

dyslipidemia on cardiovascular morbidity and mortality in the general population has been reported in numerous epidemiologic studies".[7]

"But, the role of dyslipidemia in the pathophysiology of atherosclerotic disease in patients with CKD remains controversial. Some studies have shown a positive association between cholesterol values and the risk for cardiovascular events in CKD individuals, whereas others failed to find any significant correlation. Although the precise causes of this significant deviation from what is observed in the general population have not been established, it has been proposed that the presence of phenomena such as inflammation or protein energy wasting (conditions very common in ESRD patients) may significantly confound the relationship between the traditional risk factors for CVD and mortality in CKD patient population".[50]

"The study by Chen et al. also demonstrated that some level of dyslipidaemia were found to be independently associated with renal replacement therapy and rapid renal progression in CKD. Thus, assessment of lipid profile in CKD patients may help identify high risk groups with adverse renal outcomes".[7]

CONCLUSION

There were 59.0% males and 41.0% females with 51.0% educated and 49.0% Uneducated subjects. There were 2.0% Businessman, 9.0% Farmers, 34.0% Housewives, 15.0% Labourers, 5.0% Retired subjects, 11.0% Shop keepers, 14.0% Students, 5.0% Teachers and 5.0% Workers.

The mean Age of the study population was 42.45±16.24 years, mean weight was 60.47±6.33kgs, mean height was 166.52±3.33 cms and BMI was 21.76±1.64.

Viral markers were found to be negative in all the patients.

Albumin was found to be negative in 73.0% patients, + in 6.0%, ++ in 3.0% patients and +++ in 18.0% patients.

Pus cells were found to be negative in 73.0% patients, 1-2 cells were found in 9.0% patients, 6-8 cells were found in 3.0% patients, 8-10 cells were found in 6.0%, 15-20 cells were found in 3.0% patients, 35-40 cells were found in 3.0% patients and OCC were found in 3.0% patients.

The mean Systolic Blood Pressure was 158.40±21.35, Diastolic Blood Pressure was 85.30±10.77 and Pulse rate was 95.48±20.24.

The mean Fasting blood sugar level was 89.76±10.75, Post-prandial blood sugar level was 114.35±14.24 and HbA1c level was 5.04%±0.54%.

The mean Hb level was 7.43±2.27, TLC level was 8180.70±4069.56 and Platelet count (in lakhs) was 1.85±0.64.

The mean total Cholesterol level was 137.13 ±40.01, Triglycerides level was 161.59±56.04, HDL Cholesterol level was 44.99±15.35, VLDL Cholesterol level was 31.98±11.50 and LDL Cholesterol level was 64.84±30.96.

The mean Total Cholesterol, Triglycerides, VLDL Cholesterol and LDL Cholesterol was significantly more among Stage 4 and 5 chronic kidney disease in comparison to Stage 3 chronic kidney disease.

Haemodialysis was done among 73.0% subjects.

Patients of Chronic renal failure with and without hemodialysis are at higher risk of developing dyslipidemias, which is characterized by hypertriglyceridemia, elevated VLDL and decreased HDL levels.

"Disturbances in lipoprotein metabolism (mainly accumulation of intact or partially metabolized apolipoproteinB-containing particles as well as reduced concentrations of HDL-cholesterol) are evident even at the early stages of CKD and usually follow a decreasing pattern that parallels the deterioration in renal function. Since several intrinsic (genetic, primary kidney disease) or exogenous (drugs, method of renal replacement) factors can influence the phenotypic expression of these alterations, the precise knowledge of the pathophysiological mechanisms that underlie their development is of paramount importance".

"Hemodialysis is the process which can effectively reduce the accumulation of nitrogenous waste products but fails to clear dyslipidemias generated during the course of CRF. But still the patients on hemodialysis are still exposed to several of the metabolic consequences of renal failure. On the basis of the findings of the present study, it is further suggested that prescribing lipid lowering treatment in CRF patients with dyslipidemias for preventing future episode of cardiovascular events could help and will also preserve renal function. A strict monitoring lipid profile and lipoproteins can reduce the morbidity and mortality rate and will also improve the quality of life of CRF patients".

"Our results indicated that patients undergoing CRF show important abnormalities of lipid metabolism which could contribute to atherosclerosis and cardiovascular disease and may increase the morbidity and mortality in these patients".

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