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Research Article

STUDY OF LIPID PROFILE IN PATIENTS WITH CHRONIC KIDNEY DISEASE

Dr. Devpriya Shukla, Dr. R.K.Jha and Dr. Chetan Mathur

Department of General Medicine Sri Aurobindo Medical College & PG Institute-Indore (M.P.)

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Background and Objective: Chronic kidney disease is diagnosed by US national kidney foundation disease quality outcomes initiative classification (K/DOQI) as GFR less than 90ml/min/ 1.73m2 for more than 3 months. These patients have found to have varied lipid profiles depending on the co-morbid conditions and also the treatment given. The lipid profiles of these patients in the western and the Indian subcontinent have widely varied in different studies. This study was undertaken to note the lipid profile in CKD patients in our hospital. **Method:** This study is a cross sectional descriptive study which has been done over a period of one year in sixty patients with CKD. The lipid profile was done on these patients. **Results:** The fasting lipid profile was collected in sixty patients with CKD and the values were analysed using students t test. It was found that the mean values of only triglycerides will elevated however the p value showed no statistically significant correlation. No correlation was found with the co morbid conditions and the lipid profile. **Conclusion:** this study has found that the mean values of the lipid profile in sixty CKD patients only the triglycerides were elevated. However, the statistical analysis showed that it was not significant. This study however did not include a detailed dietary and caloric history. Also the study group was heterogeneous .It would hence be beneficial to undertake the study after eliminating the limitation and also including a larger group.

INTRODUCTION

Chronic kidney disease (CKD) an inevitable terminal event of chronic renal parenchymal disease due to various causes is known more for its morbidity than for its mortality. The effects of the altered functioning of the renal system are reflected in every organ system of the body. The severity of the consequences of CKD has however undergone profound changes since the advent of dialysis. Cardiovascular disease is a major cause of mortality and morbidity among patients with CKD. More than 50 percent of patients with CKD die due to cardiovascular complications[1]. In recent times dyslipidemia has been identified as a major risk factor for coronary artery disease[2].This has renewed interest in the identification and management of abnormalities in the plasma lipids and lipoproteins[2]. An association between lipids and kidney disease was first noted by Virchow[3] who described fatty degeneration of renal epithelium in Bright's disease in 1860. The magnitude of the problem has become more apparent in the recent years as a result of an increase in the life span of the patients due to the advent of haemodialysis .The incidence of coronary artery disease is seen in 26 percent of dialysis patients[4].In chronic kidney disease the most prevalent lipid abnormalities which have been noted are hypertriglyceridemia and decreased HDL concentration[5].

Corresponding author:* **Dr. Devpriya Shukla
Department of General Medicine Sri Aurobindo Medical College & PG Institute-Indore (M.P.)

The LDL levels are usually found to be normal or marginally increased. Increased levels of atherogenic lipoproteins, especially LDL and possibly chylomicrons remnants, contribute to the development of atherosclerosis .Increased plasma concentration and reduced diameter favour sub endothelial accumulation of these lipoproteins .Following chemical modifications such as oxidation, the lipoproteins are no longer cleared by normal mechanisms. They trigger a self-perpetuating inflammatory response during which they are taken up by macrophages to form foam cells –a hallmark of the atherosclerotic process. Atherogenic lipoproteins also have an adverse effect on the endothelial function[6]. The arterial narrowing that follows impairs the blood supply to various organs.

Aims

To study the lipid profile in patients with chronic kidney disease

Background

Chronic kidney disease (CKD), a terminal event of chronic renal parenchymal disease due to various causes has been known for its paramount disability to the renal and also the secondary involvement of the various organ systems of the body.

The presentation and the clinical features seen in renal disease have been noted by physicians since the time of Hippocrates.

In 1847, Piorry P.A coined the term "uraemia"[7] which he attributed to the retention of urine in the blood. This theory persisted till the early part of the 20th century. An association between lipids and kidney disease was first noted by Virchow[3] who described fatty degeneration of the renal epithelium in cases of chronic kidney disease in 1860. The criteria used to include patients as a case of CKD is described by structural or functional abnormalities of the kidney lasting for more than 3 months with or without decreased glomerular filtration rate (GFR) and manifested by pathological abnormalities or markers of kidney damage (i.e. abnormalities in the composition of the blood, urine or abnormalities in the imaging studies). CKD can also be defined by using the US national kidney foundation disease quality outcome classification (K/DOQI) as GFR less than 90ml/min/1.73m2 for more than three months[8]. CKD affects all groups irrespective of the age, sex, religion, race or climatic condition. In 8-10% of the cases the aetiology for chronic kidney disease is not known.

Causes of chronic kidney disease

Glomerulopathies

1. Primary glomerular diseases: Focal and Segmental glomerulosclerosis. Membranous nephropathy. Membranoproliferative glomerulonephritis. IgA nephropathy. Idiopathic crecentric glomerulonephritis, Others
2. Secondary glomerular diseases: Diabetes Mellitus. Amyloidosis. Post infectious glomerulonephritis. Heroin abuse nephropathy. Collagen vascular diseases. Sickle cell glomerulopathy

Tubulointerstitial renal diseases: Nephrotoxic drugs e.g. antibiotic, NSAID's, heavy metals, diuretics. Reflux/chronic pyelonephritis. Renal tuberculosis .Myeloma kidney. Lymphoma/leukaemia. Multisystem disorder e.g. sarcoidosis

Hereditary diseases: Polycystic kidney disease. Alport's syndrome. Medullary cystic disease. Fabry's disease

Vascular diseases: Renal artery obstruction.Hypertensive nephrosclerosis. Chronic radiation nephritis
5. Obstructive nephropathy: Prostatic diseases. Nephrolithiasis. Retroperitoneal fibrosis/tumour

Amongst all the causes of CKD the most common causes in India are diabetic nephropathy (28.7%), chronic glomerulonephritis (22.4%) and chronic tubulointestinal disease (22.4%)[9]. The kidney has the innate ability to maintain the GFR by hyper filtration and compensatory hypertrophy of the remaining healthy nephrons. This adaptability of the nephrons allows for the continued normal clearance of plasma solutes. Hence, the levels of creatinine and urea will start to show significant rise only after the GFR has decreased by approximately fifty percent. All healthy humans also exhibit an age dependant physiological decline in the renal function amounting to an average of 0.75ml/min loss in the GFR per year after the age of forty years[10]. In CKD, accelerated renal decline supervenes irrespective of the underlying cause, indicating a final common pathway after the initial insult. Different theories have been proposed to the pathogenesis of CKD. The most accepted among them are the trade off theory, the middle molecular theory and the theory of uraemic toxins. These theories are interrelated and together help to explain the different clinical and biochemical changes seen in patients of CKD.

Lipid Metabolism: The term lipid refers to substances with poor water solubility. These include biologically important materials such as sterols, including cholesterol, that are composed of hydrocarbon rings and glycerides such as triglycerides and phospholipids, that are chiefly composed of hydrocarbon chains. The most common fats of diet are the neural fats also known as triglycerides. Each molecule of triglycerides is composed of a glycerol nucleus and three fatty acids. This neutral fat is found in food of both animal and plant origin.

Dyslipidemia in chronic kidney disease: Progressive renal failure especially when associated with proteinuria is accompanied by abnormalities of lipoprotein transport. Typically, the dyslipidemia is reflected predominantly in increased serum levels of triglycerides with high levels of VLDL, apoB and pre HDL and low levels of HDL and of apoA. Cholesterol levels may be very high in proteinuric patients[11].

This pattern of abnormalities is due to several mechanisms. Urinary protein loss stimulates an increased LDL synthesis by the liver. It is likely that proteinuria with the resultant hypoalbuminemia leads to an up regulation of 3-hydroxy-3-methyglutaryl CoA reductase with a consequent hypercholesterolemia[12]. Conversely, low HDL with a poor maturation of HDL-3 to cholesterol rich HDL-2 is due to acquired lecithin-cholesterol acyltransferase deficiency secondary to abnormal urinary losses of the enzyme[13]. Impaired clearance of chylomicrons and VLDL has emerged as the dominant factor for the increased serum triglyceride concentration. Lipoprotein lipase (LPL) is the rate-limiting enzyme in lipolysis of chylomicrons and VLDL. LPL binds to heparan sulphate proteoglycans on the cell surface of endothelium. In proteinuric renal diseases, a down regulation of LPL protein and enzymatic activity was found. These events are largely responsible for profound abnormalities in lipoprotein metabolism in nephrotic syndrome and chronic kidney disease thereby rendering these lipoproteins more atherogenic. The importance for the study of dyslipidemia in patients with CKD is emphasised by the high mortality seen in these patients due to cardiovascular or cerebrovascular disease .Hyperlipidemia has also been shown to increase the progression of the renal disease itself. Studies conducted in the western countries have demonstrated the presence of hypertriglyceridemia and a lower level of HDL in most of the cases, irrespective of the therapeutic approach. However the studies in the Indian subcontinent have shown varied results. This has been attributed to various factors namely the diet, the genetic factors, the economic condition and thereby varied availability of food types to name a few have been said to play a role for the varied results. However the recent studies conducted in India as described above have shown results similar to the western studies, though the levels of triglycerides was not as elevated as found in the western studies. Due to the varied results it should prove to be worthwhile to study the lipid profile in our patients as the corrective measures, if there is hyperlipidemia, may prove to be life saving.

MATERIAL AND METHODS

Source of Data: Diagnosed cases of CKD attending the outpatient department or admitted in SAIMS HOSPITAL INDORE.

Sample size: Sixty patients with chronic kidney disease were included using purposive sampling technique.

Design of the study: Cross sectional descriptive study.

Duration of Study: 1 year.

Procedure: Patients presenting to the hospital and diagnosed with CKD were included in the study after obtaining informed consent until sixty cases were collected. The history of the onset, progression, duration of various symptoms, drug and diet history was noted. The patient's were clinically examined for signs of hyperlipidemia. Laboratory investigations like basic blood profile, blood urea, serum creatinine, serum cholesterol, serum triglyceride, serum LDL, serum HDL and ultrasound abdomen will be done. No follow up will be done.

Inclusion Criteria

1. Patients diagnosed as chronic kidney disease on conservative treatment or hemodialysis irrespective of other coexisting disease.
2. Age more than fifteen years.
3. Male and female patients.

Exclusion criteria: Renal transplant patients. Patients on lipid lowering drugs

Statistical Analysis: Mean, standard deviation and confidence interval was calculated and the same represented by graphs. Student's t test was used to calculate the significance between means.

RESULTS

This study was conducted on sixty patients with chronic kidney disease over a period of one year. The data from each patient was obtained on a proforma and analysed. The results are as follows. Among the 60 patients included in this study 72% were male and 28% were females. The male to female ratio was 2.5:1.

Table 1 Sex Distribution

	No. of patients	Percent
Female	17	28.3
Male	43	71.7
Total	60	100.0

The study sample included majority 31.7 percent of the patients in the age group of 55 -64 years followed by 22 percent in the age group of 50-59 years.

Table 2 Age in years

	No. of patients	Percent
25 - 34	7	11.7
35 - 44	3	5.0
45 - 54	11	18.3
55 - 64	19	31.7
65 - 74	13	21.7
75 - 84	7	11.7
Total	60	100.0

	N	Minimum	Maximum	Mean	Std. Deviation
AGE yrs	60	25	84	57.53	14.912

The study group was analysed with the risk factors associated with increased cardiovascular mortality. It was found that patients with diabetes comprised 65 percent of the study population while the remaining 35 percent were non diabetic.

Table 3 Diabetes Mellitus

	No. of patients	Percent
No	21	35.0
Yes	39	65.0
Total	60	100.0

Among the sixty patients included in the study 68 percent (41 patients) had hypertension as a co morbid condition.

Table 4 Hypertension

	No. of Patients	Percent
No	19	31.7
Yes	41	68.3
Total	60	100.0

In this study population only 35 percent were on haemodialysis while the remaining 65 percent were managed conservatively.

Table 5 Haemodialysis

	No. of Patients	Percent
No	39	65
Yes	21	35
Total	60	100

A detailed general physical examination was done on the patients included in this study group with the importance to the dermatological manifestation of hyperlipidemia (xanthelasma, extensor digitorum xanthomas, Achilles tendon xanthomas or eruptive xanthomas). It was found that only 36.7 percent of the studied patients had skin markers of hyperlipidemia.

Fasting lipid profile was obtained in all sixty patients and the mean values for each fraction was calculated and is depicted in the following bar diagram.

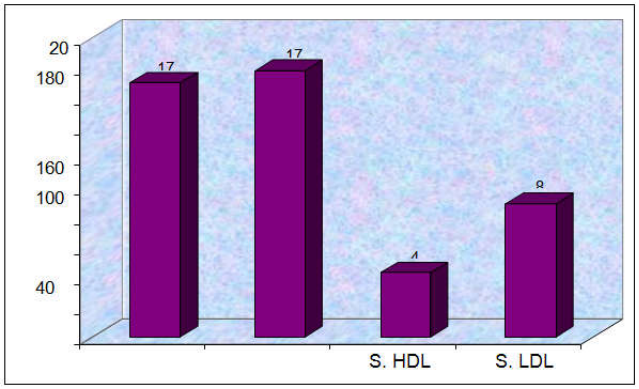

Fig 7 Mean values of fasting lipid profile in sixty patients of CKD

As depicted in the graph, it can be seen that the mean value of Serum Triglycerides is 170 mg/dl, Serum cholesterol is 178 mg/dl, Serum HDL is 43 mg/dl and Serum LDL is 89 mg/dl. They are all within normal range. The mean values of the different fractions were obtained with respect to the co-morbid conditions present in the study sample. The comparative mean values of the different fractions of lipid in the hypertensive subjects with those who were not hypertensive is depicted in bar diagram below.

The mean values of the lipoprotein fractions were also compared between males and female patients. There was no significant difference found.

Table 6 Group Statistics

SEX		N	Mean	Std. Deviation
S.TRI mg/dl	Female	17	164.71	58.092
	Male	43	173.30	121.594
S.CHOLESTEROL mg/dl	Female	17	182.65	45.678
	Male	43	176.93	40.616
S.HDL mg/dl	Female	17	50.47	13.852
	Male	43	41.28	13.888
S.LDL mg/dl	Female	17	89.65	33.160
	Male	43	89.84	37.022

This study group was analysed and the triglycerides, cholesterol, LDL and HDL values were compared to the age distribution in the group. The p values were found to be insignificant.

	P Value	
S.Triglyceride	0.46	NS
S.Cholesterol	0.93	NS
S.HDL	0.38	NS
S.LDL	0.10	NS

The values were also compared using students t test .The values were found to be insignificant

DISCUSSION

This study is a cross sectional descriptive study which included sixty patients of chronic kidney disease who were treated as inpatients or outpatients. The cases were collected over a period of one year. The patients included those who were managed with hemodialysis and those on conservative treatment.

The patients were interviewed for a detailed medical history including the presence of co-morbid conditions. They were examined in for the presence of markers of hyperlipidemia (xanthelasma, tendon xanthomas, or eruptive xanthomas). They tested for serum lipid profile after overnight fasting. Relevant laboratory investigations like hemoglobin, total count, random fasting sugar, serum urea, serum creatinine, electrolytes and ultrasound abdomen for the kidney size were done. The glomerular filtration rate was calculated using the Cockroft- Gault formula.

GFR in males= (140-age) x body weight (kilograms)
72 x S .Creatinine

In females GFR = (140-age) x body weight (kilograms) x 0.85
72 x S .Creatinine

The patients fasting lipid profile was compared to the values by the National cholesterol education program extent panel on detection, evaluation and treatment of high blood cholesterol in Adults (NCEP-ATP)

The lipid fractions were also compared to the albuminuria in patients with CKD to evaluate if there was any correlation with the protein loss and the lipoprotein fraction. The p value of the data was insignificant. The p values for S.Triglycerides, S.Cholesterol, S.HDL, and S.LDL as analyzed with the albuminuria were 0.98, 0.85, 0.53 and 0.31 respectively. These were found to be non significant.

Chan CM [14] studied the lipid abnormalities in patients with renal failure due to nephrotic syndrome and also due to causes other than nephrotic syndrome. He found that the prevalence of hypertriglyceridemia and the elevation of HDL cholesterol were proportional to the severity of renal impairment. He however noticed that the diabetic patients had increased triglycerides and lower HDL suggesting that diabetes itself exacerbated lipid abnormalities. In this study the p value of the CKD patients with diabetes and without diabetes were compared. The p values of S.Triglyceride is 0.50, S .Cholesterol is 0.81, S.HDL is 0.49 and S.LDL is 0.26 which was found to be non significant and not in accordance with the lipoprotein abnormalities seen in the study by Chan CM. In a study by Attman PO and colleagues[15], it was found that all the patients with the moderate to advanced renal failure had elevated triglycerides. They also found that the cholesterol was elevated and HDL reduced. No change was seen in the LDL level. This study showed that the patients had triglyceride and cholesterol levels within normal range and that the HDL value was not reduced.

In a study by Ravichandran[16] and Shah and colleagues [17] patients managed with hemodialysis and those on conservative management they found that patients had marginally elevated levels of triglycerides and the level of dyslipidemia had no statistical correlation to the caloric intake in both these groups. The lipoprotein fractions were also compared between those on hemodialysis and those on conservative management and no statistically significant correlation was found. The p values of S. Triglyceride are 0.87, S. Cholesterol is 0.91, S.HDL is 0.80 and S.LDL is 0.09 which is not significant. In this study though the triglyceride level was found to be marginally elevated as in the above mentioned studies there was no statistical correlation found . This study did not compare the caloric intake in the study population and the triglyceride levels. This could be attributed to low calories derived from carbohydrates and the high intake of polyunsaturated fatty acid in the diet of most of the people residing in this region. Hence it can be seen that the degree of hypertriglyceridemia in our population is less although the type of hyperlipoproteinemia is the same as that in the Western population. This may be related to the dietary pattern in the form of high intake of polyunsaturated fatty acids. In this study though the triglyceride level was found to be marginally elevated as in the above mentioned studies there was no statistical correlation found . This study did not compare the caloric intake in the study population and the triglyceride levels.

In a study by Nayak and colleagues[18] they found that the lipid profile in diabetic and non diabetic patients with CKD had elevated triglycerides, LDL cholesterol and VLDL. They found no statistically significant correlation between diabetic and the non diabetic patients. However the study group in this study was heterogeneous hence the data collected should probably have included a larger group.

CONCLUSION

This study has found that the mean values of the lipid profile in sixty CKD patients only the triglycerides were elevated. However, the statistical analysis showed that it was not significant. This study however did not include a detailed dietary and caloric history. Also the study group was heterogeneous. It would hence be beneficial to undertake the study after eliminating the limitation and also including a larger group.

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