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Original Research Article

Fasting Lipid Profile in Chronic Kidney Disease Patients: A Hospital Based Case Control Study

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Abstract:

Background: Cardiovascular disease (CKD), the leading cause of death for people with chronic kidney disease (CKD), is largely caused by dyslipidemia. Therefore, to reduce morbidity and mortality in CKD patients, it is imperative to investigate their lipid profile.

Methods: Subjects each of 50 in number are grouped into healthy controls (group-1), CKD patients without hemodialysis (group-2), CKD patients with hemodialysis (group-3). After fasting of 12 hours, lipid profile is assessed in all cases.

Results: In this study, there is increase in Total cholesterol (TC), Low Density lipoprotein (LDL), very Low-Density lipoprotein (VLDL) and Triglycerides (TG) and decrease in High Density Lipoprotein (HDL) in all CKD patients compared to healthy controls (p-value for each parameter <0.001). There is increase in TC, TG and VLDL in diabetic CKD patients compare to non-diabetic CKD patients and p-value for each parameter is <0.05. It was found that TG and VLDL increase and HDL decrease in group-3 compare to group-2 is statistically significant (p-value for each <0.05) and no significant variation in TC and LDL in these groups.

Conclusions: The present study showed that dyslipidemia exists in CKD patients regardless of their management strategy, but that the group with hemodialysis had significantly more dyslipidemia and is therefore at higher risk of cardiovascular disease. Starting lipid-lowering medication is preferable since it slows the progression of the disease and dyslipidemia.

Keywords: Chronic kidney disease, Cardiovascular disease, Hemodialysis, Lipid profile.

Introduction

More is known about the morbidity of chronic kidney disease (CKD) than its mortality. CRD is an inevitable terminal outcome of chronic renal parenchymal disease resulting from numerous causes. The severity of the effects of chronic kidney disease has changed significantly since the introduction of dialysis. Cardiovascular disease (CVD) is a major cause of mortality in patients with mild to moderate chronic kidney disease (CKD) and endstagerenal disease (ESRD). [1]

Irrespective of its agents, ultimately it leads to structural and functional hypertrophy of surviving nephrons. Clinically the patients are asymptomatic, with the progression of disease process and with the increasing amount of nephron losses leads to the end stage of renal disease (ESRD) which depicts the prolonged signs and symptoms of uremia. In order to reduce the burden of ESRD, research area should focus on clinical trials to slow the progression of kidney disease. Since, the availability of management aspects towards primary kidney diseases is meagre. Besides, therapies directed towards slowing the progression of kidney disease via controlling hypertension by using angiotensin converting enzyme inhibitors (ACEI's) and angiotensin receptor blockers (ARB's) are recommended management therapies. [2]

Dyslipidemia has been identified as an independent risk factor for the progression of kidney disease. The deleterious effect of hyperlipidemia on the progression of kidney disease is based on a number of lines of evidence. Hyperlipidemia has been clearly shown to accelerate the progression of kidney disease. There is extensive evidence for the processes involved in lipid induced kidney damage, where multiple mechanisms appear to be involved. In chronic kidney disease the most prevalent lipid abnormalities which have been noted are hypertriglyceridemia and decreased HDL concentration. The LDL levels are usually found to be normal or increased. [3] An association between

lipids and kidney disease was first noted by Virchow 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 hemodialysis. The incidence of coronary artery disease is seen in 26 percent of dialysis patients. [4]

Material and Methods

This hospital based case control study conducted in chronic kidney patients of age group 18 to 80 years in Department of Medicine, Sri Krishna Medical College and Hospital, Muzaffarpur, Bihar from July 2019 to June 2020. Total 150 patients are divided in to three groups. Group 1 was healthy control patient (n=50), group 2 was CKD patients without hemodialysis (n=50) and group 3 was CKD patients on hemodialysis (n=50).

Patients are included with established chronic kidney disease irrespective of the etiology and as evidenced radiologically (bilateral shrunken kidney/loss of corticomedullary differentiation) or biochemically (elevated blood urea, serum creatinine for more than 3 months) and those with renal transplant patients, patients with acute renal failure and nephrotic syndrome, who are on drugs affecting lipid metabolism like beta blockers, statins and oral contraceptive pills and female patients who are pregnant are excluded from study.

After overnight fasting of 12 hours, venous blood is collected for lipid profile and renal function tests. Along with them complete blood count, Liver function tests, urine examination, USG abdomen and pelvis were collected. The serum total cholesterol (TC), high density lipoprotein cholesterol (HDL-C), triglycerides (TGs) and very low-density lipoprotein (VLDL) are measured using commercially available Randox autoanalyzer and low-density lipoprotein cholesterol (LDL-C) calculated from Friedewald`s Formula (LDL=TC-HDL-TG\5). [5]

Non numerical entries were coded numerically into nominal/ordinal distribution before analysis. Continuous variables were analyzed using Mean±standard deviation. Mean difference between two independent groups was analyzed using student t-test. This was analyzed using SPSS for windows version 20.0 (Trial version).Calculation of eGFR [6] is done by equation from the modification of diet in renal disease study (MDRD).

Estimated GFR (ml/min per 1.73 m^2) = $1.86\times(SCr)^{-1.154} \times (age)^{0.203}$.Multiply by 0.742 for women, multiply by 1.21 for black-African ancestry, 0.763 for Japanese, 1.233 for Chinese and patients are grouped in to stages according Kidney Disease Improving Global Outcome (KDIGO) Classification. [7] GFR (ml/min/1.73 m^2) categories, Stage-1:>90, Stage-2: 60-89, Stage-3A: 45-59, Stage-3B: 30-44, stage-4: 15-29, Stage-5: <15.

Results

The basic characteristic features of candidates are shown in Table 1.

Table 1: Baseline characteristics of study group

Characteristics	Group 1	Group 2	Group 3
No. of patients	50	50	50
Age (yrs.) (Mean±SD)	39.92±16.59	42.02±14.30	48.08±13.15
Sex (Male/Female)	22/28	36/14	28/22
BMI (kg/m^2)	22.43±2.14	22.99±1.90	22.86±2.11
No. of diabetes patients	00	20	28

The fasting lipid profile pattern between healthy controls and CKD patients, CKD patients without hemodialysis and with hemodialysis are shown in Table 2 and 3 respectively. In this study in group 2: 6, 15, 29 candidates were in stages 3b, 4 and 5 respectively. In group 3: 2 and 48 candidates were in stage 4 and 5 respectively. There was increase in TC, LDL, TG and VLDL and decrease in HDL with increase in stages in groups 2 and 3 but significance could not be assessed as candidate number was small for comparison. The fasting lipid profile in healthy controls and CKD patients as shown in Table 2 reveals that, there in increase in TC, LDL, TG and VLDL in CKD patients compare to healthy controls and was significant for each parameters (<0.005) and decease in HDL in CKD patients compare to healthy patients and it was also statistically significant (p <0.05).

Table 2: Fasting lipid profile of healthy controls and CKD patients

Parameter (mg/dl)	Healthy controls (Mean±SD)	CKD patients (Mean±SD)
Total cholesterol	130.59±16.12	195.21±24.64
HDL-cholesterol	54.21±3.94	38.35±4.01
LDL-cholesterol	94.96±18.83	153.07±23.84
Triglycerides	94.02±19.92	205.75±53.40
VLDL	13.96±3.78	29.14±16.33

Table 3: Fasting lipid profile of CKD patients without hemodialysis and with hemodialysis

Parameter (mg/dl)	Group 2 (Mean±SD)	Group 3 (Mean±SD)
Total cholesterol	195.63±16.76	194.80±30.75
HDL-cholesterol	39.50±4.39	37.20±3.24
LDL-cholesterol	153.6±26.80	149.99±65.69
Triglycerides	187.67±27.88	223.91±65.69
VLDL	23.71±9.94	34.57±19.49

Table 4: Fasting lipid profile of diabetic and non-diabetic CKD patients

Parameter (mg/dl)	CKD patients with Diabetes Mellitus (Mean±SD)	CKD patients without Diabetes Mellitus (Mean±SD)
Total cholesterol	198.69±30.87	192.0±16.70
HDL-cholesterol	38.19±4.09	38.49±3.97
LDL-cholesterol	151.05±25.44	154.92±22.34
Triglycerides	228.56±49.55	184.78±48.33
VLDL	33.97±16.93	24.68±14.53

The fasting lipid profile between CKD patients without hemodialysis and with hemodialysis as shown in Table 3 reveals there is increase TG, VLDL and decrease in HDL group 3 compare to group 2 and the changes were statistically significant ($p < 0.05$). The difference in values of TC and LDL were not statistically significant in either group.

The fasting lipid profile in diabetes and non-diabetes CKD patients is shown in Table 4. The increase in total cholesterol and triglycerides and very low-density lipoprotein in diabetic CKD patients is statistically significant compare to non-diabetic CKD patients (p.0.05).

Discussion

Cardiovascular disease (CVD) is major cause of mortality in patients with mild to moderate chronic kidney disease (CKD) and end stage renal disease (ESRD). In Hallan SI et al, it is found that cardiovascular mortality is higher in 25-34-year-old ESRD patients compare to individuals from the general population of the same age and race. [8] In a retrospective cohort study very few patients (0.5-1%) with mild to moderate CKD developed ESRD over a 5-year follow up, while 19 and 24% of these patients with mild and moderate CKD patients respectively, died because of cardiovascular complications in that same period. [9]

Several mechanisms may underlie these reductions in HDL cholesterol levels, which is usually an indication of impaired reverse cholesterol transport. Apo AI, which is the activator of lecithin cholesterol acyltransferase (LCAT), is reduced in CKD due to down regulation of hepatic Apo AI genes leads to decline in the activity of LACT, which causes reduced cholesterol esterification and impairment of HDL maturation. The activity of LACT is consistently diminished in CKD, so there is decrease in HDL levels. [10]The present study demonstrates that CKD is commonly accompanied by lipid abnormality in the form of hypertriglyceridemia. This is similar to the observations made in Western studies and recent Indian studies by Gupta DK, Das BS and Bagdae J. [11,12] 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 represent the most important patho-physiological mechanisms underlying the development of hypertriglyceridemia in renal failure. [13] Chan MK et al, also found hypertriglyceridemia was the major abnormality in their studies. [14] Hypertriglyceridemia represents an early feature of renal failure.

The increase in triglycerides in hemodialysis patients is more compare to non-hemodialysis patients due to, heparin which is used in hemodialysis inhibits lipoprotein lipase (LPL), which is responsible for hydrolysis of triglycerides. The increased VLDL cholesterol concentration in chronic kidney disease because of delayed catabolism of VLDL. In uremia the cholesterol content of HDL is low and the apo C-II concentration is also low. Normally this apo C-II is transferred from HDL in plasma to VLDL. The decreased in apo C-II leads to decreased triacylglycerol catabolism and VLDL metabolism. So, VLDL concentration increases. [15]

Conclusion

Patients with CKD receiving hemodialysis have statistically significant increases in their blood triglyceride and low-density lipoprotein levels as their stage increases. It has been discovered that there is a statistically significant rise in VLDL with increasing stage in CKD patients who do not receive hemodialysis.

When comparing diabetic CKD patients to non-diabetic CKD patients, there is a statistically significant rise in TC, TG, and VLDL ($p < 0.05$). When compared to CKD patients without hemodialysis, TG and VLDL are elevated while

HDL decreases in hemodialysis patients. Every parameter has a p-value of less than 0.05. In CKD patients, decreased HDL-C levels and elevated TGL and VLDL levels were the predominant lipid abnormalities.

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