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Original Research Article

Profile of Fasting Lipids in Chronic Kidney Disease Patients: An inpatient case control study

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Abstract

Background: Dyslipidemia has a major role in cardiovascular disease (CKD), which is the primary cause of death for those with chronic kidney disease (CKD). Therefore, it is essential to look into the lipid profile of CKD patients in order to lower morbidity and mortality.

Methods: Each of the 50 subjects is divided into three groups: CKD patients without hemodialysis (group 2), CKD patients with hemodialysis (group 3), and healthy controls (group 1). In all cases, the lipid profile is evaluated following a 12-hour fast.

Results: Total cholesterol (TC), low density lipoprotein (LDL), very low density lipoprotein (VLDL), and triglycerides (TG) are all higher in CKD patients than in healthy controls, but high density lipoprotein (HDL) is lower (p-value for each parameter <0.001). Diabetic CKD patients have higher levels of TC, TG, and VLDL than non-diabetic CKD patients, and the p-value for each of these parameters is less than 0.05. There was no discernible difference in TC and LDL between groups 3 and 2, however there was a statistically significant rise in TG and VLDL and a drop in HDL when compared to group 2 (p-value for each <0.05).

Conclusion: According to the current study, dyslipidemia occurs in CKD patients regardless of their management approach; however, the hemodialysis group had a much greater level of dyslipidemia and, as a result, a higher risk of cardiovascular disease. Since it slows the growth of the disease and dyslipidemia, it is better to start using lipid-lowering medicine.

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

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Introduction

The morbidity of chronic kidney disease (CKD) is better understood than its mortality. CRD is a final consequence of chronic renal parenchymal disease that can arise from a variety of causes. Since the advent of dialysis, the severity of the effects of chronic renal disease has changed dramatically.

One of the leading causes of death for people with mild to severe endstage renal disease (ESRD) and chronic kidney disease (CKD) is cardiovascular disease (CVD).[1] Regardless of its agents, it eventually causes the remaining nephrons to enlarge both structurally and functionally. Clinically, the patients are asymptomatic; nevertheless, as the disease process advances and nephron losses increase, end-stage renal disease (ESRD) develops, exhibiting persistent uremia symptoms. Additionally, it is advised to use angiotensin converting enzyme inhibitors (ACEIs) and angiotensin receptor blockers (ARBs) to control hypertension in order to decrease the course of renal disease.[2]

One independent risk factor for the development of renal disease is dyslipidemia. There are several lines of evidence supporting the detrimental impact of hyperlipidemia on the development of renal disease. It is well established that hyperlipidemia hastens the course of renal disease. Numerous mechanisms seem to be involved in the processes

that lead to lipid-induced kidney injury, which is well supported by research. Reduced HDL concentration and hypertriglyceridemia are the most common lipid abnormalities found in chronic renal disease. Typically, the LDL values are either elevated or normal.[3]

Virchow first identified a link between lipids and kidney illness in 1860 when he reported fatty degeneration of the renal epithelium in Bright's disease. Due to the introduction of hemodialysis, patients' life expectancy has increased in recent years, making the scope of the issue more obvious. Twenty-six percent of dialysis patients have coronary artery disease.[4]

Material and Methods

In the Department of Medicine at Sri Krishna Medical College and Hospital in Muzaffarpur, Bihar, chronic renal patients between the ages of 18 and 80 participated in this hospital-based case control study between March 2025 and August 2025. Three groups of 150 patients each are formed. 50 healthy control patients made up group 1, 50 CKD patients without hemodialysis made up group 2, and 50 CKD patients receiving hemodialysis made up group 3. Patients who have established chronic kidney disease, regardless of the cause, as demonstrated by radiological evidence (bilateral shrunken kidney/loss of corticomedullary differentiation) or biochemical evidence (elevated blood urea, serum creatinine for more than three months) are included in the study. Patients who have received a renal transplant, acute renal failure, nephrotic syndrome, are taking medications that affect lipid metabolism, such as beta blockers, statins, or oral contraceptive pills, and pregnant women are not included. 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

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.

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 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

For individuals with mild to severe chronic kidney disease (CKD) and end-stage renal disease (ESRD), cardiovascular disease (CVD) is a leading cause of death. According to Hallan SI et al., cardiovascular mortality is higher in ESRD patients aged 25–34 than in people of the same age and race who are members of the general population.[7,8] In one retrospective cohort analysis, only 0.5% to 1% of patients with mild to moderate chronic kidney disease (CKD) acquired end-stage renal disease (ESRD) during a 5-year follow-up. At the same time, 19 and 24% of patients with mild and moderate CKD, respectively, passed away from cardiovascular problems.[9]

These decreases in HDL cholesterol levels, which are often a sign of compromised reverse cholesterol transport, could be caused by a number of factors. 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. In CKD, LACT activity is continuously reduced, which results in a drop in HDL levels.[10] The current study shows that hypertriglyceridemia, a lipid disorder, frequently coexists with chronic kidney disease (CKD). This is consistent with findings from recent Indian research by Gupta DK, Das BS, and Bagdae J.[11,12] as well as Western studies. The most significant pathophysiological mechanisms behind the development of hypertriglyceridemia in renal failure are the decreased activity of lipoprotein lipase (LPL) and the direct inhibitory effect of different uremic "toxins" on the enzymes involved in lipid metabolism.[13] In their research, Chan MK et al. also discovered that the main anomaly was hypertriglyceridemia.[14] One of the first signs of renal failure is hypertriglyceridemia.

Because the hemodialysis drug heparin inhibits the enzyme lipoprotein lipase (LPL), which hydrolyzes triglycerides, hemodialysis patients have higher triglyceride levels than non-hemodialysis patients. delayed VLDL catabolism, which results in elevated VLDL cholesterol levels in chronic renal illness. Both the HDL cholesterol content and the apo C-II concentration are low in uremia. Normally, HDL in plasma transfers this apo C-II to VLDL. Reduced VLDL metabolism and triacylglycerol catabolism result from a reduction

in apo C-II. Thus, the concentration of VLDL rises.[15]

Conclusion

Blood triglyceride and low-density lipoprotein levels rise statistically significantly with increasing stage in hemodialysis patients with chronic kidney disease. It has been found that in CKD patients without hemodialysis, VLDL increases statistically significantly with increasing stage. TC, TG, and VLDL increase statistically significantly ($p < 0.05$) in diabetic CKD patients compared to non-diabetic CKD patients. Hemodialysis patients have lower HDL and higher TG and VLDL than CKD patients without hemodialysis. The p-value for each parameter is less than 0.05. The most common lipid abnormalities in CKD patients were raised TGL and VLDL levels and decreased HDL-C values.

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