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AN ASSESSMENT OF FASTING LIPID PROFILE IN PATIENTS OF CHRONIC KIDNEY DISEASE PRESENTING AT TERTIARY CARE HOSPITAL

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Abstract:

Objective: *To assess the fasting lipid profile in patients of chronic kidney disease presenting at tertiary care hospital.*

Material and methods: *This case control study was conducted at Department of Department of Pathology, Sahiwal Medical College, Sahiwal from February 2019 to August 2019 over the period of 6 months. Total 50 health control, 50 CKD patients with out hemodialysis and 50 CKD patients on hemodialysis were selected. Age range in this study was 18-80 years. Mean lipid parameters were studied in all groups.*

Results: *Mean age of the patients in group A (controls), group B (CKD patients with out hemodialysis) and group C (CKD patients on hemodialysis) was 39.92±16.59 years, 42.02±14.30 years and 48.08±13.15 years. In group A male/female were 22 (44%) / 28 (56%), in group B was 36 (72%) / 14 (28%) and in group C was 28 (56%) /22 (44%). Mean BMI in group A was 22.43±2.14 followed by group B 22.99±1.90 and in group C was 22.86±2.11. Total cholesterol, HDL, LDL, TG and VLDL in controls vs CKD patients was 130.59±16.12 vs 195.21±24.64, 54.21±3.94 vs 38.35±4.01, 94.96±18.83 vs 153.07±23.84, 94.02±19.92 vs 205.75±53.40 and 13.96±3.78 vs 29.14±16.33 respectively. Difference of mean lipid parameters between controls and CKD patients was statistically significant.*

Conclusions: *Results of present study showed there is a significant difference of mean lipid parameters between controls and CKD patients. Difference of mean HDL, mean TG and mean VLDL between CKD patients without hemodialysis and CKD patients on hemodialysis was statistically significant.*

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

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INTRODUCTION:

Chronic kidney disease (CKD) is one of today's leading public health problems, with increasing frequency and prevalence. According to the Kidney Disease Outcomes Quality Initiative (K/DOQI), CKD is defined as kidney damage or a decreased kidney glomerular filtration rate (GFR) of <60mL/min/1.73m^2 for at least 3 months. [1] Cardiovascular disease (CVD) is a major cause of mortality in patients with mild to moderate chronic kidney disease (CKD) and end stage renal disease (ESRD). [2]

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. [3]

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 are meagre. [4]

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. [5]

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. [6]

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. [7]

MATERIAL AND METHODS:

This case control study was conducted at Department of Department of Pathology, Sahiwal Medical College, Sahiwal from February 2019 to August 2019 over the period of 6 months. Total 50 health control, 50 CKD patients without hemodialysis and 50 CKD patients on hemodialysis were selected. Age range in this study was 18-80 years. An approval was taken from ethical committee and written informed consent was taken from every patient. 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. Five ml blood sample was taken for lipid profile and renal function test. Laboratory findings were noted on pre-designed proforma along demographic profile of the patients.

All the collected was entered in SPSS version 20 and analyzed. Mean and SD was calculated for numerical data and frequencies were calculated for categorical data. Student t test was used to detect difference of mean lipid profile between the groups. P value 0.05 was considered as significant.

RESULTS:

Mean age of the patients in group A (controls), group B (CKD patients with out hemodialysis) and group C (CKD patients on hemodialysis) was 39.92±16.59 years, 42.02±14.30 years and 48.08±13.15 years. In group A male/female were 22 (44%) / 28 (56%), in group B was 36 (72%) / 14 (28%) and in group C was 28 (56%) /22 (44%). Mean BMI in group A was 22.43±2.14 followed by group B 22.99±1.90 and in group C was 22.86±2.11. (Table 1)

Total cholesterol, HDL, LDL, TG and VLDL in controls vs CKD patients was 130.59±16.12 vs 195.21±24.64, 54.21±3.94 vs 38.35±4.01, 94.96±18.83 vs 153.07±23.84, 94.02±19.92 vs 205.75±53.40 and 13.96±3.78 vs 29.14±16.33 respectively. Difference of mean lipid parameters between controls and CKD patients was statistically significant. (Table 2)

Mena total cholesterol of group B and C was 195.63±16.76 vs 194.80±30.75 and the difference was statistically insignificant with p value 0.8673. Mean

HD of group B and C was 39.50±4.39 vs 37.20±3.24 and difference was statistically significant with p value 0.003. Mean LDL of group A was 153.66±26.80 and men LDL of group C was 149.99±23.53 but the difference of mean LDL between the both groups was statistically insignificant with p value 0.4686. Mena TG was 187.67±27.88 and 223.91±65.69 respectively in group B and C and the difference was statistically significant with p value 0.000. Mean VLDL of group B and C was 23.71±9.94 and 34.57±19.49 and the difference was statistically significant with p value 0.000. (Table 3)

Table 1: Baseline characteristics

Characteristics	Group A	Group B	Group C
Age (years) (mean±SD)	39.92±16.59	42.02±14.30	48.08±13.15
Sex (male/Female)	22 (44%) / 28 (56%)	36 (72%) / 14 (28%)	28 (56%) /22 (44%)
BMI (kg/m2)	22.43±2.14	22.99±1.90	22.86±2.11

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

Lipid Parameters (mg/dl)	Healthy controls (mean±sd mg/dl)	CKD patients (mean±sd mg/dl)	P value
Total cholesterol	130.59±16.12	195.21±24.64	0.001
HDL-cholesterol	54.21±3.94	38.35±4.01	0.001
LDL-cholesterol	94.96±18.83	153.07±23.84	0.001
Triglycerides	94.02±19.92	205.75±53.40	0.001
VLDL	13.96±3.78	29.14±16.33	0.001

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

Lipid Parameters (mg/dl)	Group B (mean±sd mg/dl)	Group C (mean±sd mg/dl)	P value
Total cholesterol	195.63±16.76	194.80±30.75	0.8673
HDL-cholesterol	39.50±4.39	37.20±3.24	0.003
LDL-cholesterol	153.66±26.80	149.99±23.53	0.4686
Triglycerides	187.67±27.88	223.91±65.69	0.000
VLDL	23.71±9.94	34.57±19.49	0.000

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 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:

Results of present study showed there is a significant difference of mean lipid parameters between controls and CKD patients. Difference of mean HDL mean TG and mean VLDL between CKD patients without hemodialysis and CKD patients on hemodialysis was statistically significant.

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