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Original article:

Study of lipid profile parameters in Chronic Kidney Disease at tertiary care hospital

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Abstract:

Introduction: Dyslipidemia is a common complication of chronic kidney disease and contributes to the high cardiovascular morbidity and mortality in such patients.

Material and methods: The present study entitled "Study of Lipid profile, Lipoprotein (a), Apoprotein AI and B in chronic kidney disease" is case-control study and has been carried out in our institute during the period of February 2011 - August 2012. All the study subjects were examined & investigated according to predesigned proforma .The study protocol was approved by the Ethical Committee of the Institute. Informed written consent was obtained from all the study subjects enrolled in the study 50 diagnosed chronic kidney disease patients (Cases) attending medicine outpatient department (OPD) and / or admitted in ward/kidney unit in this institute and who were willing to participate in the study were selected for the present study.

The mean age of distribution in group A (Cases) is 47 ± 11.6 years and the mean age of distribution in group B (Controls) is 47.32 ± 11.76 years. (Fig- 5.1). Most of subjects of control and cases of chronic kidney disease are between 51- 60 years.

Results and Conclusion: Serum triglyceride, VLDL cholesterol, Total Cholesterol, Lipoprotein (a), Apo B levels were elevated significantly and decrease in serum HDL-cholesterol, Apo AI levels were recorded in chronic kidney disease cases than that of controls.

Keywords : Chronic kidney disease , lipid profile

Introduction:

Dyslipidemia is a common complication of chronic kidney disease and contributes to the high cardiovascular morbidity and mortality in such patients. The 1998 Report of the NKF Task Force on Cardiovascular Disease in Chronic Renal Disease, drew attention to cardiovascular disease as an outcome of chronic kidney disease. [1]The Task Force recommended that patients with chronic kidney disease be considered in the -highest risk group‖ for subsequent cardiovascular disease (CVD) events. The excess risk of cardiovascular disease to some extent, is due, to a higher prevalence of conditions that are recognized as risk factors for cardiovascular disease in the general population (–traditional‖ CVD risk factors) and to hemodynamic and metabolic factors

characteristic of chronic kidney disease (–CKD-related| CVD risk factors). In addition, the Task Force emphasized the high mortality from cardiovascular disease. Cardiovascular disease is the leading cause of death in patients with kidney failure. After adjusting for age, gender, race, and diagnosis of diabetes; mortality from cardiovascular disease is far higher in patients with kidney failure compared to the general population.[2]

Material and methods:

The present study entitled "Study of Lipid profile, Lipoprotein (a), Apoprotein AI and B in chronic kidney disease" is case-control study and has been carried out in our institute during the period of February 2011 - August 2012. All the study subjects were examined & investigated according to predesigned proforma .The study protocol was approved by the Ethical Committee of the Institute. Informed written consent was obtained from all the study subjects enrolled in the study

50 diagnosed chronic kidney disease patients (Cases) attending medicine outpatient department (OPD) and / or admitted in ward/kidney unit in this institute and who were willing to participate in the study were selected for the present study.

50 age and sex matched healthy and apparently normal controls were also selected for study. The cases and controls were in the age group of 21-65 years of either sex.

Inclusion criteria:

Criteria for chronic kidney disease

a) Diagnosed cases of chronic kidney disease.

b) Patient of either sex between 21 to 65 years of age.

c) Those who gave consent.

Criteria for controls: Age & Sex matched healthy and apparently normal individuals without family history of kidney disease.

EXCLUSION CRITERIA:

1) Patients with diagnosed chronic kidney disease treated with renal transplantation or dialysis.

2) Patients with diagnosed cases of acute renal failure like prerenal, renal and postrenal acute renal failure or azotemia.

3) Patients with diagnosed chronic kidney disease with abnormal cardiac function secondary to myocardial ischemic disease and/or left ventricular dysfunction.

4) Patients of nephrotic syndrome, Liver diseases.

Results:

TABLE-1: SERUM LIPID PROFILE, LIPOPROTEIN (a), APO AI AND APO B IN GROUP A-I (CASES) AND GROUP B (CONTROLS)

SERUM LIPIDS	GROUP A-I n=25	GROUP B n=50	p VALUE	INFERENCE
Total Cholesterol	185.7± 17.6	178.5 ± 17.1	0.021	S
Triglyceride	211.3 ± 25	119.6 ± 23.4	<0.0001	HS
VLDL Cholesterol	42.3 ± 5	23.9 ± 4.7	<0.0001	HS
HDL Cholesterol	39.2 ± 3.9	48 ± 5.9	<0.0001	HS
LDL Cholesterol	104.3 ± 20	104.8 ± 15	0.914	NS
LDL-C/HDL-C	2.7± 0.7	2.2 ± 0.4	0.001	HS
TC/HDL-C	4.8 ± 0.7	3.7 ± 0.5	<0.0001	HS
Lipoprotein(a)	36.8 ± 8.1	7.0 ± 3.9	<0.0001	HS
Apo AI	76.3 ± 15.4	123.3 ± 19.2	<0.0001	HS
Apo B	164.3 ± 24.5	105.2 ± 19.1	<0.0001	HS
Apo B/Apo AI	2.2 ± 0.4	0.9 ± 0.1	<0.0001	HS

n=number of subject; S – significant; NS – Not significant; HS - Highly significant. It shows serum lipid profile in Group A-I (Cases) and Group B (Controls).

The mean value of total cholesterol of Group A-I (Cases) and Group B (Control) is found to be 185.7 ± 17.6 mg/dl and 178.5 ± 17.1 mg/dl respectively. The mean total cholesterol level in Group A-I is increased as compared to that of Group B and the difference is significant. p=0.02. p<0.05

Group A-I (Cases) has mean serum triglyceride levels of 211.3 ± 25 mg/dl which is significantly higher as compared to that of Group B (Controls) i.e. 119.6 ± 23.4 mg/dl. The increase in mean value of triglyceride of Group A-I (Cases) as compared to Group B (Controls) is highly significant statistically with p- value <0.0001

The mean serum VLDL cholesterol level of Group A-I (Cases) is 42.3 ± 5.0 mg/dl while that of group B (Controls) is 23.9 ± 4.7 mg/dl. The increase in mean serum VLDL cholesterol in Group A-I as compared to Group B is statistically highly significant ($p < 0.0001$).

The mean serum HDL cholesterol level of Group A-I (Cases) i.e. 39.2 ± 3.9 mg/dl is found to be significantly lower than that of Group B (Controls) i.e. 48 ± 5.9 mg/dl. Statistical comparison of the mean serum HDL cholesterol levels of Group AI and Group B show, highly significant difference with p value < 0.0001.

The levels of mean serum LDL cholesterol in Group A-I and in Group B (Controls) are 104.3 ± 20 mg/dl and 104.8 ± 15.0 mg/dl respectively. The difference is not statistically significant ($p = 0.914$ i.e. $p > 0.05$).

The mean values of atherogenic ratio (LDL-C/HDL-C) in Group A-I (Cases) and group B (Controls) are found to be 2.7 ± 0.7 and 2.2 ± 0.4 respectively.

There is statistically highly significant increase in atherogenic ratio (LDL- C/HDL-C) in group A-I as compared to group B ($p=0.001$).

TABLE-2: SERUM LIPID PROFILE, LIPOPROTEIN (a), APO AI AND APO B IN GROUP A-II (CASES) AND GROUP B (CONTROLS)

SERUM LIPID	GROUP A-II n=25	GROUP Bn=50	p VALUE	INFERENCE
Total Cholesterol	182.8 ± 17.07	178.5 ± 17.1	0.098	NS
Triglyceride	276.4 ± 46.4	119.6 ± 23.4	< 0.0001	HS
VLDL Cholesterol	55.3 ± 9.3	23.9 ± 4.7	< 0.0001	HS
HDL Cholesterol	27.7 ± 6.61	48 ± 5.9	< 0.0001	HS
LDL Cholesterol	99.85 ± 16.5	104.8 ± 15	0.236	NS
LDL-C/HDL-C	3.72 ± 0.74	2.2 ± 0.4	< 0.0001	HS
TC/HDL-C	6.87 ± 1.26	3.7 ± 0.5	< 0.0001	HS
Lipoprotein(a)	52.2 ± 13.7	7.0 ± 3.9	< 0.0001	HS
Apo AI	50.0 ± 12.2	123.3 ± 19.2	< 0.0001	HS
Apo B	180.5 ± 25.5	105.2 ± 19.1	< 0.0001	HS
Apo B/Apo AI	3.7 ± 1.0	0.9 ± 0.1	< 0.0001	HS

n= number of subjects; NS – Not significant; HS - Highly significant. It shows serum lipid profile in Group A-II (Cases) and Group B (Controls).

Discussion:

The relationship between the blood urea and the plasma creatinine has been well established in the chronic renal failure. Evaluation of the blood urea is considered as an essential marker for monitoring renal failure. This measurement pertains primarily to glomerular function and as filtration slows or ceases, the blood urea rises. Paralleling this rise is an increase in the plasma creatinine level.[3]

Along with kidney function test, levels of serum total cholesterol, triglyceride, VLDL cholesterol, HDL cholesterol, LDL cholesterol, Lipoprotein (a), Apo AI and Apo B were estimated in all the participants of both the study groups and the atherogenic ratio i.e. LDL-C/HDL-C, TC/HDL-C and Apo B/Apo AI were calculated. Of the total lipid profile parameters which were evaluated, serum triglyceride, VLDL cholesterol, HDL-cholesterol, Total

cholesterol, Lipoprotein (a), Apo A-I, Apo B and the atherogenic ratios i.e. LDL- C/HDL-C, TC/HDL-C and Apo B/Apo A-I ratio were noticed to be significantly altered in chronic kidney disease. Whereas, **LDL cholesterol** exhibited **non-significant** alteration.

During evaluation, Group A-I, Group A-II (Cases) and Group B (Controls) had mean serum triglyceride levels of 211.3 ± 25 mg/dl, 276.4 ± 46.4 mg/dl and119.6 ± 23.4 mg/dl respectively .Group A had statistically higher triglyceride level as compared with Group B control ($p<0.0001$).

Alam and Bhatt (1991)[4] reported a similar finding of significantly higher levels of serum triglyceride and decreased levels of serum HDL cholesterol in uremic patients treated conservatively and by maintenance haemodialysis.

Shoji et al (1992)[5] found that VLDL cholesterol increases with marginal significance and there is significant reduction in HDL cholesterol especially in HDL_2 sub-fraction and increase in HDL triglyceride with decreased HDL_2/HDL_3 cholesterol ratio in patients of chronic kidney disease.

Hypertriglyceridemia is one of the most common quantitative lipid abnormalities in patients with CKD. The concentrations of triglyceride-rich lipoproteins [very-low density lipoprotein (VLDL), chylomicrons, and their remnants] start to increase in early stages of CKD. Several studies have shown that patients with impaired renal function exhibit increased concentrations of triglycerides even though serum creatinine levels are within normal limits. The predominant mechanism responsible for increased concentration of triglyceride rich lipoproteins in predialysis patients is delayed catabolism. The reduced catabolic rate may be due to diminished lipoprotein lipase activity as a consequence of the down-regulation of the enzyme gene and the presence of lipase inhibitors .[6,7]

Conclusion:

Serum triglyceride, VLDL cholesterol, Total Cholesterol, Lipoprotein (a), Apo B levels were elevated significantly and decrease in serum HDL-cholesterol, Apo AI levels were recorded in chronic kidney disease cases than that of controls.

References:

1. Tsimihodimos V, Zoi M, Moses E .Dyslipidemia Associated with Chronic Kidney Disease. The Open Cardiovascular Medicine Journal. 2011; 5: 41-48.
2. Bargman JM, Skorecki K. Chronic Kidney Disease. In: Fauci AS, Braunwald E, Kasper DL, Hauser SL, Longo DL, Jameson JL, et al, editors. Harrison‘s Principles of Internal Medicine.17th ed. New Delhi: Mc-Graw Hill; 2008. p.1761-71.
3. Vasudevan DM, S Sreekumari, Vaidyanathan K, editors. Cardiovascular Diseases and Hyperlipidemias. Textbook of Biochemistry for medical students. 6th edition. New Delhi: Jaypee Brothers; 2011. p. 292-300.
4. Alam SM, Bhatt AK. Abnormal lipoprotein in uremic patients treated conservatively and by maintenance haemodialysis. J Assoc Physicians India. 1991 Feb; 39(2):170-1.

5. Haffner SM, Katherine KG, Gilbert A, Philip AM, Michael PS, Katherine RT. Increased Lipoprotein (a) concentrations in Chronic Renal Failure.J.Am.Soc.Nephrol.1992; 3:1156-1162.
6. Shoji T, Nishizawa Y, Nishitani H, Yamakawa M, Morii H. Impaired metabolism of high density lipoprotein in uremic patients. Kidney International.1992 Jun;41(6):1653-61.

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