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A Study of Various Biochemical Parameters of Lipid Metabolism in Renal Disorders

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Dyslipidemia contributes to the rate of progression of atherosclerosis and chronic kidney disease. Also chronic kidney disease leads to the development of secondary abnormalities in lipid metabolism that contribute to increased cardiovascular morbidity and mortality. on lipid induced glomerular damage.Present study was conducted to assess cardiac risk among the patient of CKD. The study comprised into two groups 30 morbid and 42 healthy control, all were in age group of 20 to 50 years having nephrotic syndrome with ESRD undergoing haemodialysis attending nephrology clinic, male, female ratio 3:2, blood urea, S. creatinine, lipid profile, apolipoprotein A and B were estimated it was found that patient of NS had significantly decreased level of HDL-Chol, Apo A1, Apo A1/Apo B, HDL-Chol./T.Chol. as compared to control same time significantly increased level of T-Chol., TG, LDL-Chol., VLDL-Chol, APO-B, LDL-Chol/HDL-Chol., Apo B/Apo A1.

Key words: Dyslipidemia, Glomerulosclerosis, Nephrotic syndrome (NS), ESRD.

Dyslipidemia contributes to the rate of progression of atherosclerosis and chronic kidney disease. Also chronic kidney disease leads to the development of secondary abnormalities in lipid metabolism.

There is growing evidence of a pathogenic link between atherosclerosis and progressive kidney disease, which in part accounts for the accelerated cardiovascular mortality in patient with chronic kidney diseases[1-3]. Apolipoprotein have been receiving increasing attention in view of their important role in the structural integrity and functional specificity of lipoprotein particle, the two major lipoprotein i.e. apoipoprotein A and B have been studied along with lipid profile, so as to understand further mechanism underlying the athrogenicity of certain dyslipoproteinemic states in vitro cell culture studies of human[4-7] glomerular cells have provided useful information on lipid induced glomerular damage[8,9].

Moreover understanding the pathogenesis of vessel injury in atherosclerosis has provided insights into mechanism that leads to kidney injury as hyperlipidemia accelerates the rate of glomerulosclerosis and reduction in lipid level reverse this effect[10-13].

In several animal species lipogenic diet lid to glomerulosclerosis often preceded by glomerular enlargement, mesangial expansion and hypercellularity[14,15].

Lowering of serum lipid and cholesterol was associated with a reduction of structural and functional renal damage[16,17] lipid abnormalities in renal disease of different etiology and haemodialysis has not been used extensively in Indian population moreover the result have been variable and controversial[18-21].

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MATERIAL AND METHODS

The study was carried out at the department of Biochemistry at MLB Medical College, Jhansi with the collaboration of associated hospital. Blood was obtained from 30 with NS and 42 healthy control group all were age group of 20 to 50 years, mean age 40 years and weight 55 kg and male female ratio 3:2.

Before collecting the blood sample from patients and control 12 hours overnight fasting and collected 5 ml heparinised blood from each patient in a sterilized vial and kept for half on hour proper coagulation and than serum was aspirated by centrifugation at 3000 rpm for 10 minutes and lipid profile measured by fully automated clinical chemistry analyzer –

1. Plasma total chol. By CE-CO PAP enzymatic end point method[22].
2. Estimation of S. HDL Chol. By precipitation method.
3. VLDL and LDL were estimated by simple calculation by using friedewald's equation[23].
4. Estimation of S. TG by GPO-PAP end point method[24].
5. Estimation of blood glucose by GOD-POD end point method. Value have been expressed as mean+SD the result were analyzed using student 't' Test p <0.05 was considered as significant.
6. APO lipoprotein A1 and B estimation by orion diagnostic as immunochemical assay[25] sample were studied by colorimetrically for blood urea, creatinine, blood sugar described in practical clinical biochemical by Varley.

RESULTS

In our study high TG (P<0.001, 91.7%) T.chol. (IP<0.005, 28.2%), LDL Chol. (P<0.05, 33.25%), VLDL Chol. (P<0.00192, 93%), APOB (P<0.001, 43.28%) significantly increase and HDL Chol. was significantly low (P<0.005, 17%) as compared to controls, hence this group has shown increased risk of atherosclerosis. It is well established that increased rates of synthesis and secretion of VLDL cholesterol by the liver play a major role[22-25] (Table 1A & B).

Table 1.A

	Group V (n=30)	Control (n=42)	P Value
Tch. mg% mean+SD	218.57+50.2	183.56+47.32	<0.05
TG mg% mean+SD	207.74+48.21	100.9+32.0	<0.005
HDLch mg% Mean+SD	32.77+8.31	37.37+11.52	NS
LDLch mg% Mean+SD	144.24+34.12	125.59+31.76	<0.05
VLDLch mg% Mean+SD	41.54+12.27	19.98+4.31	<0.005
APO A1 mg% Mean+SD	102.98+29.71	98.12+31.10	NS
APO B mg% Mean+SD	96.53+28.54	63.41+18.10	<0.005

Table 1.B

	Group V (n=30)	Control (n=42)	P Value
APO A1/B Mean+SD	1.06+0.32	1.55+0.39	<0.05
LDL-ch/HDL-ch	4.5+1.14	3.48+0.99	<0.005
HDL -ch/Tchol Mean+SD	0.146+0.04	202+0.05	<0.05
TG/Tchole Mean+SD	0.949+0.19	0.546+0.10	<0.005
Tchole/HDL -ch Mean+SD	6.81+1.31	4.945+0.98	<0.05
Apo B/A1 Mean+SD	0.946+0.30	0.642+0.27	<0.005
Calculated HDL Mean+SD	35.01+9.20	38.81+12.32	NS

DISCUSSION

The study group compared of 30 patients with 42 control group significantly increased level of TG, LDL. Chol., VLDL Chol., APO B, and T.Chol, APOB/A1, ($P<0.005$), it may because of increased synthesis and decreased catabolism of lipoproteins may accounts for severe hyperlipidemia which frequently occurs in patients with NS, NS is defined is protienuria, hypoalbunemia odema, and hypercholesterolemia, high lipid levels are not only marker of disease, but also contribute to the process of glomerulosclerosis[26] hyperlipidemia is integral post of the NS. It causes increased risk of atherosclerosis[27-30].

Recent experiments have suggested that hyperlipidemia may also play a role in the progression of renal disease.

CONCLUSIONS

There was general trend towards increase in TG level along with increase in total cholesterol and LDL & VLDL accompanied by decrease in HDL level., APO A1, APO A1/ APO B ratio an atherogenic index was evaluated in all patient dialyzed and undialyzed in both groups. This is the scope of dyslipidemia in nephrotic is an addition to the effect of basic disease. Dislipidemia is one of the possible factors contributing to atherosclerosis may be worsened by haemodialysis.

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