

Estimation of Biochemical Parameters of Lipid Metabolism In Diabetics

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ABSTRACT

In this present study included 40 morbid and 50 healthy control all were in age group of 20 to 60 years having renal failure of various etiology but in this study group patients of diabetes with ESRD studied, male, female ratio 3:2, lipid profile, blood sugar, apolipoprotein A and B were estimated by fully automated clinical chemistry analyzer, orion diagnostic immuno chemical assay and blood sugar by GOD, POD method. It was found that patient of ESRD with diabetes had HDL-chol, APO A1, APO A1/APO B, HDL Chol./T.Chol. was significantly decreased ($p < 0.05$) as compared to control. Same time T-Chol., TG, LDL-Chol., VLDL-Chol, APO-B, LDL-Chol/HDL-Chol., APO B/A1, significantly increased ($p < 0.05$) as compared to control. It can be concluded from the present study that patient of ESRD with diabetes had significantly decreased level of HDL-Chol, APO A1, APO A1/ APO B, and significantly increased level of TG, VLDL, APO B/APO A1, ratio on atherogenic index was evaluated in all patient dialyzed and undialyzed in both groups. Indicated that atherogenic changes is more advanced in diabetic than in non diabetics with ESRD, this is the scope of dyslipidemia in diabetics with nephropathy is an addition to the effect of basic disease.

Key words: Hyperlipidemia, Hyperglycemia, Lipid metabolism, Lipids, ESRD.

INTRODUCTION

Diabetes mellitus is characterized by absolute or relative deficiencies in insulin secretion and/or insulin action associated with chronic hyperglycemia and disturbances of carbohydrate, lipid and protein metabolism. Long-term vascular complications represent a major cause of morbidity and mortality in patients with diabetes mellitus. In addition, various biochemical disorders associated with vascular complications, such as hyperlipidemia and oxidative stress which frequently co-exist with diabetes mellitus¹ appear inadequate to explain the increased risk of vascular diseases. The observations suggest that additional factors may be involved in the acceleration of diabetic vascular disease.

Patients with diabetes are at increased risk of coronary cerebral and peripheral vascular disease and frequently have abnormal plasma lipid levels. Several recent findings have indicated the possible presence of structural and functional abnormalities that may impair the lipid metabolism transport system in diabetics. In diabetic patients with renal impairment were exposed to a uremic state and chronic haemodialysis which may provide further modification to lipid metabolism.²

MATERIALS AND METHODS

The study was carried out at the department of Biochemistry at MLB Medical College with the collaboration of associated hospital. Blood was obtained from 40 diabetes and 50 healthy

controls all were age group of 20 to 60 years, mean age 35 years and weight 47 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 an hour proper coagulation and then 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 method³.
2. Estimation of S. HDL Chol. By precipitation method.
3. VLDL and LDL were estimated by simple calculation by using friedewald's equation.³
4. Estimation of S. TG by GPO-PAP end point method.⁴
5. Estimation of blood glucose by GOD-POD end point method. Value have been expressed as mean \pm 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 assay⁵ sample were studied by colorimetrically for blood urea, creatinine, blood sugar

described in practical clinical biochemical by Varley.

Diabetes patient with ESRD subdivided into 2 groups

- (a) Patient undergoing chronic Haemodialysis – 18n
- (b) Patient on conservative management without HD 22 n.

RESULTS

In our study if has found that patients of diabetes with ESRD had HDL chol. APO A1, APO A1/APB, HDL Chol., T. Chol. Was significantly decreased ($p < 0.05$) as compared to control table 1 same time T chol, TG, LDL Chol, VLDL Chol, APO B, LDL Chol, / HDL Chol, T Chol / HDL, APO B1, APO A1 significantly increased.

Diabetes with ESRD showed marked high levels of TG in both dialyzed 62.6% and undialyzed population (71%). Also significant lower value observed for HDL-Chol. (26.48%) and LDL Chol. (18.7%) in undialyzed and dialyzed group, HDL Chol. (34.77%), LDL (20.7%), VLDL increase was significant in both undialyzed (72.98%) and dialyzed (63.92%) population. APO B / APO A1 ratio an atherogenic index was elevated in all patients

Table 1: Comparison of lipoprotein levels in end stage renal disease with diabetes

S. No.	Parameters (Mean \pm SD)	Diabetic Group	Control (n=50)	P value
1	T.Chol.(mg%)	203.81+9.53	175.00+45.3	<0.05
2.	Triglyceride	220.30+60.31	125.00+40.12	<0.005
3.	HDL. Chol.	24.74+6.73	35.33+8.51	<0.05
4.	LDL Chol.	169.22+21.38	125.12+33.81	<0.05
5.	VLDL Chol.	126.52+43.02	25.23+5.16	<0.005
6.	APO A1	127.00+45.00	142.00+37.48	<0.05
7.	APO B	139.00+71	98.56+25.14	<0.005
8.	APO A1/B	0.913+0.19	1.448+0.23	<0.05
9.	LDL Chol. + HDL Chol.	7.04+1.26	3.571+1.13	<0.005
10.	HDL Chol. / T.Chol.	0.102+0.04	0.2+0.07	<0.0005
11.	TG/T/Chol.	1.018+0.32	0.714+0.94	<0.005
12.	T.Chol / HDL	8.238+2.11	5.0+1.04	<0.005
13.	APO B/A1	1.094+0.31	0.69+0.23	<0.005
14.	Calculated HDL	27.21+4.91	35.53+7.33	<0.05

in both groups, prominent increase was demonstrated in diabetic dialyzed kidney. The result indicated that the atherogenic change is more advanced in diabetic than in non diabetic ESRD.

DISCUSSION

The study group compared of 40 patients of diabetes with ESRD shown significantly increased level of TG ($p < 0.001$), T.Chol ($p < 0.05$) APO lipoprotein B ($p < 0.05$), LDL-Chol. / HDL Chol. ($p < 0.005$), TG / T.Chol, T.Chol / HDL-Chol. ($p < 0.005$), APO B, APO A1 ($p < 0.005$).

Also significant lower values observed for HDL chol, ($p < 0.05$) APO lipoprotein A ($p < 0.05$) APO A1 / APO B ($p < 0.05$), HDL Chol. / T. Chol. ($p < 0.005$) atherogenic changes more advanced in diabetic than in non diabetic ESRD.

Our study suggests that the scope of dyslipidemia in diabetics with nephropathy is in addition to the effect of the basic disease this is also in agreement with observation made by Picard *et al.*⁶

Hyper TG is most common abnormality seen in diabetes with renal failure out a decreased cholesterol content of HDL-Chol.^{7,8,9} there is high risk of coronary, cerebral and peripheral vascular disease by various workers^{10,11,12}.

CONCLUSION

Several recent findings have indicated the possible presence of structural and functional abnormalities that may impair the lipid metabolism transport system in diabetes.¹³ That the scope of dyslipidemia in diabetes with nephropathy is an addition to the effect of basic disease.

REFERENCES

1. Kannel WB, McGee D. Diabetes and cardiovascular risk factors : the *Framingham study*. *Circulation* **59**: 8-12 (1979).
2. Shohat J, Bonez G., Role of lipids in the progression of renal disease in chronic renal failure : evidence from animal studies and pathogenesis. *Israel J. of Med. Sci.* **29**: 228-239 (1993).
3. Tietz Textbook of clinical chemistry. 3rd ed. Philadelphia : WB Saunders Company; p. 809-61 (1990).
4. Friendwald WT, Levy RI, Fredrickson DS., Estimation of concentration of low density lipoprotein cholesterol in plasma without use of the ultracentrifuge. *Clin. Chem.*, **18**: 449-502 (1972).
5. Nader R., Paul B., John A., Lipids, Lipoproteins and Apollipoproteins, In Tietz Textbook of Clinical Chemistry, 3rd ed., Burtis C.A. and Ashwood E. R., Eds. W.B. Saunders, Philadelphia, 809-852 (1994).
6. Riepponen, P, Mrneimi J, Rautoja T., Immunoturbidimetric determination of apolipoproteins A1 and B in serum, *Scand J. Clin. Lab. Invest.*, **47** : 739-744 (1987).
7. Picard S., Borson-Chazat F. Bertnezene F. Is the plasma lipoprotein pattern of importance for treatment with cyclosporine? *Transplant Proc.* **18**(Suppl.5): 50-51 (1991).
8. Norbeck HE, Oro L, Carlson LA., Serum lipoprotein concentration in chronic uremia *Am J. Clin Nutr.*, **31**: 1881-1885 (1978).
9. Brunzell JD, Albers JJ, Hass JB, Goldberg AP, Agada L, Sherrard DJ., Prevalence of serum lipid abnormalities in chronic haemodialysis. *Metabolism* **26**: 903-910 (1977).
10. Risen WF, Mordasini R., Hyperlipidemia in renal failure. Phenotypes and Pathogenic mechanisms. *Contrib. Nephrol.* **41**: 312-320 (1984).
11. Attman PO, Alaupovic P, Gustafson A., Serum apolipoprotein profile of patients with chronic renal failure *Kid. Int.*, **32**: 368-375 (1987).
12. Chan MK, Varghese Z, Moorhead JF., Lipid abnormalities in uremia, dialysis and transplantation *Kid. Int.* **19**: 625-637 (1981).
13. Ash SR, Rainier JB, Sopp WE, Truitt RB, Janle EM, Kissinger PT, Paulos JT., A subcutaneous capillary filtrate collector for measurement of blood chemistries. *ASAIO J.*, **39**: 699-705 (1993).