

**Original Research Paper** 

Biochemistry

# Analysis of biochemical parameters variations in diabetic nephropathy

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ABSTRACT	This study was carried out in and 50 age matched healt profile, sr. protein, sr. urea a Plasma fasting glucose, sr. female age 30-50 year and group. Extremely significan nephropathy patients as co From the study we conclud lower plasma lipid concent	n department of biochemistry & department of Medicine S.S.M.C. Rewa. Study comprised 59 patients hy individuals. We have compared various biochemical parameters like plasma blood glucose, lipid and sr. creatinine in normal healthy control groups and diabetic patients. creatinine, sr. urea and lipid profile were increased and were extremely significant in both male & d 51-70 years in diabetic nephropathy patients as compared to same age male and female control tly decreased sr. total protein was found in both male and female 30-50 year and 51-70 years diabetic mpared to same age male and female control group. de that patients with diabetic nephropathy should improve glycemic control and in addition should ration and other biochemical parameters.	

## **KEYWORDS**

### Introduction:

Diabetic nephropathy is characterized by persistent albuminuria, elevated blood pressure and relentless decline in glomerular filtration rate and high risk of mortality and morbidity. (1,2) Diabetes mellitus is a metabolic disorder of carbohydrate metabolism in which glucose is unutilized by the body tissues producing hyperglycemia.

Over hydration is frequently present in diabetic nephropathy patients which causes dilution of serum albumin concentration on one hand and onset of congestive cardiac failure on the other hand(3). Many symptoms of uremia result from accumulation of urea, creatinine and other nitrogenous end products of amino acid metabolism(4).

The Diabetes Control and Complications Trial (DCCT) demonstrated that high glycemic control in Type I diabetics significantly prevents the onset of development of retinopathy, nephropathy and neuropathy. Evidence also supports the benefit of tight control in Type II diabetics.

In this study we have estimated and compared various biochemical parameters like plasma blood glucose, lipid profile, sr. protein, sr. urea and sr. creatinine in normal healthy control groups and diabetic patients.

#### Material and methods:

This prospective consecutive study was ethically approved by the ethical committee of S.G.M.H. Rewa. Well informed written consent was taken from every study subject. All patients with diabetic nephropathy attending the OPD and admitted in the wards of department of Medicine of S.G.M.H. Rewa were included in the study.

Study comprised 59 patients and 50 age matched healthy individuals. Patients with episodes congestive cardiac disease, atherosclerosis and stroke were excluded from the study. Fasting blood samples were collected from the study subjects under all aseptic precautions. Sample for fasting blood glucose was collected in Sodium Fluoride bulb and the plain bulb was used for the rest parameters.

Following parameters were estimated,

a. Plasma blood glucose by GOD-POD method

b. Serum Protein by Biuret method

c. Serum Creatinine by Jaffes method

d. Serum Urea by DAM method

e. Serum Cholesterol by CHOD-POP method

f.Serum HDL Cholesterol by Phospho tungstic method

g. Serum Triglycerides by GPO- PAP method

h. LDL Cholesterol was calculated by Friedewald's equation Observations:

## Table 1 : Distribution of study population

Sr.no	Age range	Normal healthy control groups				Diab patie	etic nts	nepl	hrop	athy	
		Total N.C.	Male	%	F e m ale	%	Total N.C.	M a l e	%	Fem ale	%
1	30-50	26	14	53.8 5	12	46.1 5	30	16	53. 33	14	46. 67
2	51-70	24	13	54.1 6	11	45.8 3	29	14	48. 27	15	51. 72
Total	50	27	54.00	23	46.0 0	59	30	50.8 5	29	49. 15	

Table 2 : Biochemical parameters values (Mean ± S.D.) for
normal male healthy controls & male diabetic nephropathy
patients ( age : 30-50 year)

Sr. no.	Biochemical parameter	Normal male healthy control	Male diabetic nephropathy patients	p value
		(Mean±S.D.)	(Mean±S.D.)	
1	Fasting blood glucose (mg/dl)	85.36±10.06	211.47±29.2 5	p <0.0001
2	Total protein (gm/dl)	7.10±0.39	4.93±0.54	p <0.0001

3	Creatinine (mg/dl)	0.91±0.16	2.05±0.77	p <0.0001
4	Urea (mg/dl)	25.30±6.71	47.33±1.00	p <0.0001
5	Cholesterol (mg/dl)	181.36±21.3 8	303.93±20.4 0	p <0.0001
6	HDL- Cholesterol (mg/dl)	45.71±5.64	25.67±0.62	p <0.0001
7	LDL- Cholesterol (mg/dl)	93.71±25.35	168.13±4.39	p <0.0001
8	Triglyceride (mg/dl)	91.57±19.44	170.67±4.50	p <0.0001

p < 0.0001 = Extremely significant.

Table 3 : Biochemical parameters values (Mean  $\pm$  S.D.) for normal male healthy controls & male diabetic nephropathy patients ( age : 51-70 year)

Sr	Biochemical	Normal male	Male diabetic	
no.	parameter	healthy control	nephropathy patients	pvalue
		(Mean±S.D.)	(Mean±S.D.)	
1	Fasting blood glucose (mg/dl)	76.31±7.19	215.15±31.92	p <0.0001
2	Total protein (gm/dl)	7.32±0.38	4.58±0.36	p <0.0001
3	Creatinine (mg/dl)	0.94±0.12	3.24±0.53	p <0.0001
4	Urea (mg/dl)	27.62±8.24	56.46±4.77	p <0.0001
5	Cholesterol (mg/dl)	181.54±20.3 5	304.64±18.42	p <0.0001
6	HDL- Cholesterol (mg/dl)	52.69±3.40	24.38±1.19	p <0.0001
7	LDL- Cholesterol (mg/dl)	103.38±22.6 3	171.38±6.01	p <0.0001
8	Triglyceride (mg/dl)	100.62±29.9 9	172.23±4.68	p <0.0001

p < 0.0001 = Extremely significant.

Table 4 : Biochemical parameters values (Mean ± S.D.) for normal female healthy controls & female diabetic nephropathy patients (age: 30-50 year)

Sr. no.	Biochemical parameter	Normal female healthy control	Female diabetic nephropathy patients	p value
		(Mean±S.D.)	(Mean±S.D.)	
1	Fasting blood glucose (mg/dl)	79.67±8.50	218.23±28.12	p <0.0001
2	Total protein (gm/dl)	6.69±0.38	4.61±0.30	p <0.0001
3	Creatinine (mg/dl)	0.72±0.11	2.40±0.83	p <0.0001
4	Urea (mg/dl)	25.58±5.18	45.63±1.05	p <0.0001
5	Cholesterol (mg/dl)	168.58±14.6 9	312.38±23.41	p <0.0001

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6	HDL- Cholesterol (mg/dl)	43.50±5.85	24.69±0.63	p <0.0001
7	LDL- Cholesterol (mg/dl)	104.83±22.5 7	167.92±4.44	p <0.0001
8	Triglyceride (mg/dl)	99.50±28.13	168.77±5.09	p <0.0001

p < 0.0001 = Extremely significant.

## Table 5 : Biochemical parameters values (Mean $\pm$ S.D.) for normal female healthy controls & female diabetic nephropathy patients (age: 51-70 year)

Sr. no.	Biochemical parameter	Normal female healthy control	Female diabetic nephropathy patients	p value
		(Mean±S.D.)	(Mean±S.D.)	
1	Fasting blood glucose (mg/dl)	83.27±9.67	213.29±29.26	p <0.0001
2	Total protein (gm/dl)	6.68±0.39	4.07±0.29	p <0.0001
3	Creatinine (mg/dl)	0.83±0.15	2.89±0.61	p <0.0001
4	Urea (mg/dl)	27.36±7.45	55.22±4.68	p <0.0001
5	Cholesterol (mg/dl)	181.18±19.81	306.21±20.86	p <0.0001
6	HDL- Cholesterol (mg/dl)	46.91±6.38	26.14±0.95	p <0.0001
7	LDL- Cholesterol (mg/dl)	95.55±23.14	165.69±4.37	p <0.0001
8	Triglyceride (mg/dl)	87.36±20.54	168.43±5.60	p <0.0001

p < 0.0001 = Extremely significant.

## Discussion:

In our study plasma fasting glucose, sr. creatinine, sr. urea and lipid profile were increased and were extremely significant in both male & female age 30-50 year and 51-70 years in diabetic nephropathy patients as compared to same age male and female control group. Extremely significantly decreased serum total protein was found in both male and female 30-50 year and 51-70 years diabetic nephropathy patients as compared to same age male and female and female control group.

This pattern of abnormalities is due to several pathogenic mechanisms. First, urinary protein loss stimulates LDL synthesis by the liver. It is likely that proteinuria with the resultant hypoalbunemia leads to up regulation of 3 hydroxy 3 methyl glutaryl CoA reductase with a consequent hyper-cholesteremia(5). Conversely, low HDL with a poor maturation of HDL-3 to cholesterol rich HDL-2 is due to acquired lecithin-cholesterol acyltransferase deficiency secondary to abnormal urinary losses of this enzyme(6). Progressive renal failure which is associated with proteinuria is also accompanied by lipoprotein transport. Concentration of total cholesterol, VLDL, LDL cholesterol and triglyceride rises with increasing albumin excretion rate in patients with Type I diabetes mellitus. The prevalence of hyper triglyceridemia levels seen in our study is similar to the reports of other workers Koenig R J et al(7) and Taskinen MR et al(8). In those patients with total cholesterol level more than 7 milimoles /L rate of decline in GFR was at least three times higher than those with a level less than 7 milimoles/L. The power of serum cholesterol levels in predicting the progression of diabetic nephropathy in Type I diabetes was confirmed by a Danish group in a study of 301 patients who had diabetes and overt nephropathy and were

#### followed up for 7 yrs(9).

Strict control of hyperglycemia lower the risk of nephropathy and other diabetic complications in Type I diabetes mellitus(10). The decline in renal function over time has been associated between initial glomerular filtration rate, initial urinary albumin excretion rate, hyperglycemia and age(11). Thus all patients with chronic renal failure experience a secondary form of dyslipidemia that mimics the atherogenic dyslipidemia of insulin resistant patients(12). This observation raises the possibility that the diabetic nephropathy may underlie or mediate the association between lipids and a loss of renal function. Insulin resistance characterizes Type I diabetes in patients with albuminuria and their first degree relative without diabetes(13, 14) and underlies many of alterations of diabetic nephropathy including high blood pressure, lipid abnormalities and a family history of hypertension and cardiovascular disease(12). These observations and recent study by Orchard et al(15) suggest that insulin resistance is likely to proceed and play a role in the vascular damage of diabetic nephropathy.

#### Conclusion:

At the end we conclude that patients with diabetic nephropathy should improve glycemic control and in addition should lower plasma lipid concentration and other biochemical parameters.

#### References:

- Mogesen C E. Microalbuminuria predicts clinical proteinuria and early mortality in onset diabetes. N Engl J Med. 1984; 310:356-60.
- 2. Pugh J A, Medina R, Ramirez M. Comparison of the course to end stage renal disease of Type-I & Type-II diabetic nephropathy. Diabetologica. 1993; 36: 1094-8. 3. Braunwald E, Fauci A S, Kasper D L, Hauser S L, Longo D L and Jameson J.
- Harrison's principle of internal medicine". Chronic renal failure. 2001, 15th ed., p 1551
- 4. H Sh. Ahmed, et al. Biochemical study on diabetic Nephropathy patients. I B N A I -Haitham J for Pure & Sci. 2010: 23 (3).
- Vaziri ND, Sato T, Liang K: Molecular mechanism of altered cholesterol metabolism 5 6.
- in focal glomerulosclerosis. Kidney Int. 63: 1756-1763, 2003. Vazari ND, Liang K, Park JS: Acquired lecithin: Cholesterol acyltransferase (LCAT) deficiency in nephritic syndrome. Am J Physio149: F823-F829, 2001.
- 7 Koenig R J, Peterson C M, Jones R L, Saudek C, Lehrman M, Cerami A, Correlation of glucose regulation and haemoglobin A1C in Diabetes Mellitus. N Engl J Med. 1976;295:417-20
- Taskinen M R. diabetic dyslipidemia in NIDDM. International Diabetes Monitor. 8. 1996:8:1-7
- Hovind P, Rossing P, Tarnow L, Smidt UM, Parving HH: Remission and regression in 9. the nephropathy of type I diabetes when blood pressure is controlled aggressively. Kidney Int. 60:277-283, 2001.
- 10. The Diabetes Control and Complication Trial Research Group. The effect of intensive treatment of diabetes on the development and progression of long-term complication in insulin dependent diabetes mellitus. N Engl J Med. 1993; 329: 977-86
- 11. Pinho-Silveiro S, et al. Glomerular hyperfiltration in NIDDM patients without overt proteinuria. Diabetes Care. 1993; 16: 115-9.
- 12, Reavan GM: Role of insulin resistance in human disease. Diabetes37: 1595-1607. 1988
- Yip J, Mattock MB, Morocutti A, Sethi M, Trevisan R, Viberti G: Insulin resistance in 13. insulin-dependent diabetic patients with microalbuminuria. Lancet342: 883-887, 1993
- 14. De Cosmo S, Bacci S, Piras GP, Cignarelli M, Placentino G, Margaglione M, Colaizzo D, Di Minno G, Giorgino R, Liuzzi A, Viberti GC: High prevalence of risk factor for cardiovascular disease in parents of IDDM patients with albuminuria. Diabetologia. 40: 1191-1196, 1997.
- Orchard TJ, Chang YF, Ferrell RE, Petro N, Ellis DE: Nephropathy in type I diabetes: A manifestation of insulin resistance and multiple genetic susceptibilities? Further evidence from the Pittsburgh Epidemiology of Diabetes Complication Study. Kidney Int62: 963-970, 2002