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INCIDENCE OF DIABETIC NEPHROPATHY IN DIABETIC PATIENTS - A CLINICAL SURVEY

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ABSTRACT

The incidence of Diabetic Nephropathy is rapidly increased in the last few years, therefore it is important to Study the risk factors responsible for the onset of Diabetic Nephropathy. There are 100 Diabetic Patients were chosen for this study from Government Hospital, Virudhunagar. Oral consent was obtained from all the subjects and Blood Samples were collected for the estimation of Glucose, Urea and Creatinine. In our study we have observed that 46 percent of the persons having the symptoms of diabetic nephropathy among 100 samples.

Key words: Diabetes, diabetic nephropathy, risk factors, kidney failure.

INTRODUCTION

Diabetes mellitus comprises a group of common metabolic disorders that result in hyperglycemia. It is caused by a complex interaction of genetics, environmental factors and life style choices. "Once considered a rich man's disease, diabetes today is a common man's problem". Hyperglycemia (increased levels of glucose in the blood) may be due to reduced insulin secretion, decreased glucose usage or increased glucose production. This metabolic dysfunction causes tremendous burden on the diabetic patient and on the Health care system[1].

Three distinct types of diabetes are type I, type II and gestational diabetes in which all acquire similar diagnosis, treatment, symptoms, causes and complications[2].Type I diabetes is caused by autoimmune destruction of the insulin

producing B-cells. These cells are located in clusters in the pancreas called the islets of langerhans. Destruction of the β-cells results in total lack of insulin and chronic hyperglycemia.

Type II diabetes is a chronic condition that affects the body's ability to metabolize sugar or glucose and is characterized by the inability of pancreatic beta cell to keep up with the body's demand for insulin.Gestational diabetes is thought to be caused by insulin-interfering hormones produced during pregnancy. Although gestational diabetes usually disappears after the birth of the baby, women who have had gestational diabetes have 40 % to 60% chance of developing type II diabetes mellitus within 5 to 10 years[3].

The world health organization (WHO) estimated that 90% of the diabetes population has type II diabetes. The international diabetes population (Type I & II) was expected to grow over 592 million in less than 20 years.In 2003, there were 189 million diabetics in the world. The global prevalence of type II diabetes is expected to double in the period of 2000-2005 and may reach a level of almost 324 million people[4].

Damage to the kidney called "Nephropathy" is a common long-term complication of diabetes. Kidney damage arises mainly from diabetes causing damage to the very small blood vessels (called microangiopathy or micro vascular disease) in the kidneys filtration area. Kidney failure can ultimately cause death[5]. Blood urea nitrogen is a toxic waste product (urea) in the blood. Urea is produced from the breakdown of protein already in the body and protein in our diet. A high Blood Urea Nitrogen usually means that kidney function is less than normal, but other factors may also affect the Blood Urea Nitrogen level .Creatinine is another waste product that is made when your body breaks down protein when muscles are injured[6]. A high serum creatinine level means kidney damage and measuring the creatinine level is only the first step to find out the level of kidney failure.

Diabetic nephropathy, diabetic foot ulcers and diabetic neuropathy are the complications of diabetes. Diabetes mellitus is one of the causes of end stage kidney disease, leading to morbidity and mortality in the coming days[7]. Diabetes causes 38.4% of all cases of kidney failure. In 2009 it was the primary diagnosis for 214,909 kidney failure patients. About 40% of people with diabetes will develop Chronic Kidney Failure[8].

Diabetes is a leading cause of Kidney disease and approximately 40% of individuals with diabetes develop diabetic nephropathy. It is the most common cause of chronic kidney disease and end stage renal disease in the world yet there are no specific therapies prevent or halt the progression of the disorder[9].

The main scope of the study is to identify the major risk factor that lead to increase the onset of Diabetic Nephropathy and to identify the relationship between Socio – demographic variables like age, sex, type of diabetes, family history, Smoking and alcoholism habits, duration of diabetes and mode of treatment on the onset of Diabetic Nephropathy.It is important to find the patients at high risk as early as possible and initiate preventive treatment to avoid, or at least postpone, the development of Diabetic nephropathy.

MATERIALS AND METHODS

Diabetic Patients (100) were chosen for this study from Government Hospital, Virudhunagar. The permission for collecting blood samples and oral consent from the subjects has been obtained from the commissioner of Virudhunagar Government Hospital, Virudhunagar. A detailed history including risk factors like age, sex, duration of diabetes, mode of treatment, habits likesmoking and alcoholism were collected from the patients.

Venus Blood was collected, after an overnight fast into heparinsed tubes. The plasma was separated by centrifugation at 3000 rpm for 15 minutes and stored at $4^{o}C$ until analysis which was done in Clinical Laboratory in Virudhunagar Government Hospital, Virudhunagar.

Height and weight of the diabetic patients were measured for calculating the BMI. Body Mass Index of the subjects was calculated by using the following formula[10]:

BMI (kg/m^2)= Body weight (kg)/ Height $(m)^2$

Blood glucose was estimated by enzyme method[11].The Serum Urea was estimated by DiacetylMonoxime method[12]. Serum Creatinine was estimated by Jaffe's method[13]. Statistical significance was calculated by using ANOVA[14] in SPSS software IBM SPSS Statistics 22.0.0.0 version.

RESULTS AND DISCUSSION

The biochemical parameters and anthropometric measurements were analyzed in Diabetic patients with kidney

failure. The results obtained were categorized and the data was analyzed on the basis of sex, age, extent of diabetes, BMI, type of diabetes, habits and their treatment.

Table 1: Changes in biochemical parameters such as Blood Glucose, Urea and Creatinine levels in patients with Diabetic nephropathy in different age groups

BIOCHEMICAL PARAMETERS	Below 41years of age (n=8)	41-50 years of age (n=17)	51-60 years of age (n=24)	Above 60 years of age (n=51)
GLUCOSE	173±82.13	194.23±65.85	202.18±64.21	232.72±107.08
UREA	64.8±33.79	65.3±27.21	66.45±22.82	73.72±28.88
CREATININE	2.78±1.69	2.96±2.17	3.15±2.33	3.22±2.17

Values represent mean ± S.D f>0.05

In this study biochemical parameters like Sugar, Urea, Creatinine levels were significantly higher in diabetic patients with the age group of above 60years (Table 1) when compared with other age groups like 51-60 years, 41-50 years, 31-40 years and below 30years. These results agree with several international studies that were conducted in Russia, London showed that diabetic nephropathy dramatically increases with aging, particularly after the age of 60years[15].

Table 2: Changes in biochemical parameters such as Blood Glucose, Urea and Creatinine levels in patients with Diabetic nephropathy with special reference to the extent of the disease

BIOCHEMICAL PARAMETERS	Below 10 years (n=13)	11-15 years (n=16)	16-20 years (n=25)	Above 20 years (n=46)
SUGAR	205.77±72.89	194.5±73.08	185.69±65.09	231.56±104.15
UREA	60.08±22.83	65.31±24.6	65.54±29.13	74.07±27.52
CREATININE	2.02±0.74	3.72±2.81	2.79±1.91	3.29±2.12

Values represent mean ± S.D f>0.05

Blood glucose, Urea and Creatinine levels were significantly higher in diabetic nephropathy patients those who had diabetes for the past 20 years (Table 2), when compared with other diabetic nephropathy patients who suffered for the past 15-20 years, 10-15 years and below 10years. According to the study of Zimmet PZ, as the duration of diabetes

increases, the incidence of diabetic nephropathy also increases and is statistically significant[16].

Table 3:Changes in biochemical parameters such as Blood Glucose, Urea and Creatinine levels in patients with Diabetic nephropathy with reference to BMI

BIOCHEMICAL PARAMETERS	BMI 19.5-25 (n=19)	BMI 25.5-30 (n=36)	BMI Above 30 (n=45)
GLUCOSE	215.37±82.32	181.19±57.78	231.56±104.15
UREA	64.21±26.36	64.17±26.45	74.07±27.52
CREATININE	2.66±2.13	2.99±2.11	3.29±2.12

Values represent mean ± S.D f>0.05

In our study report showed that a statistically significant increase in levels of glucose, urea and creatinine in patients who had BMI above 30 when compared with other patients had BMI 25-29.5 and BMI 19-24.5. (Table 3) It was already reported that obesity is the main risk factor for the incidence of diabetic nephropathy and is statistically significant[17].Obesity increases the risk of onset of kidney failure in diabetic patients. People at high risk for type 2 DM can prevent or delay the onset of the disease by losing 5 to 7% of their bodyweight[18].

Table 4: Comparison of changes in biochemical parameters in Diabetic nephropathy patients with alcoholism & smoking habits and patients without habits

BIOCHEMICAL PARAMETERS	Diabetic patients without Smoking & Alcoholism (n=30)	Diabetic nephropathy Patients with Smoking (n=20)	Diabetic nephropathy Patients with Alcoholism (n=13)	Diabetic nephropathy Patients with Smoking & Alcoholism (n=33)
SUGAR	104.27±10.08	189.8±74.21	267.08±133.85	208.59±76.92
UREA	26.8±4.98	61.95±18.36	77.38±29.66	73.26±35.72
CREATININE	0.99±0.16	2.49±1.45	3.17±1.90	3.41±2.82

Values represent mean ± S.D f>0.05

Blood glucose urea and creatinine levels were found to be significantly increased in diabetic patients with smoking and alcoholism when compared with normal diabetic patients free from smoking and alcoholism. (Table 4)Alcohol could lead to all sorts of damage to the kidneys. These impacts could vary from cell damage and swelling of the kidneys to alcohols' effect. Alcohol makes an ionic imbalance in the body that could

harmfully have an effect on various metabolic processes[19]. For diabetics who smoke there is a higher risk of getting kidney disease, whether they have Type 1 or Type 2 diabetes. Studies have shown the kidney disease risk in diabetics is about two to three times higher than for those who don't smoke[20]. To avoid the smoking and alcoholism habits can prevent the onset of Diabetic nephropathy[21].

Table 5: Changes in biochemical parameters such as Blood Glucose, Urea and Creatinine levels in patients with Diabetic nephropathy with reference to the nature of treatment

Biochemical Parameters	Patients under dietary management (n=14)	Patients under hypoglycemic drugs (n=26)	Patients under Insulin therapy (n=60)
Glucose	125.64±6.02[a]	151.81±11.20[a]	257.24±86.99[a]
Urea	69.21±36.20[b]	67±25.59[b]	71.89±25.51[b]
Creatinine	2.77±1.65[b]	2.86±1.99[b]	3.66±2.45[b]

Values represent mean ± S.D f>0.05

Urea and creatinine level in diabetic patients were significantly increased intaking the treatment of both tablet and insulin therapy (Table 5) when compared with the subjects have the treatment ofDietary management. These are reports available that the long-term effects of insulin therapy have been more favourable for increasing the onset of diabetic nephropathy, both in type 1 and type 2 diabetic patients[22].

CONCLUSION

In our study we have observed that diabetic patients are prone to kidney failure. The incidence of diabetic nephropathy increases by some risk factors such as aging, obesity, extent of diabetes, smoking andalcoholism. Diabetes mellitus is an 'in curable' syndrome. Strict diet, exercise can delay the onset of type II diabetes. In patients who are diagnosed as diabetes, strict monitoring of renal function tests, Blood sugar control is to be done. Foods which strengthen kidney could be included in the diet regularly.

REFERENCE

1. Mogenson CE (2000). The Kidney and Hypertension in Diabeteb Mellitus(5th edition), *Khuwar Academic Publishers*, pp 13-28.

2. Anderson S. Btrenner BM (1989). Progressive renal disease: A disorder of adaptation, *QJMD***70**: 185.
3. VasuDevan DM, Ashe HA (1995). Text book of Biochemistry for medical student – 10, 120-5.
4. Lewis JB (2007). Kidney Disease Outcome Quality Initiative. Clinical Practice Guidelines and Clinical Practice Recommendation for Diabetes and Chronic Kidney Disease. *National Kidney Foundation AJKD***72(3):**247-259.
5. Mari C and Hall J (2011). Obesity metabolic syndrome and diabetic nephropathy, *National center for Biotechnology Information,***170**:28-35.
6. Zatz R andNucci G (1991). Effects of acute nitric oxide inhibition on rat glomerular microcirculation. *Am J Physiol***261**:360-363**.**
7. VecihiBatuman MD, Colin A and Hutchison (2012). The pathogenesis and diagnosis of acute kidney injury in multiple myeloma. *Nature Reviews Nephrology* **8(1)**: 43-51.
8. Lajer M, Schjoedt KJ and Jacobsen P (2006). Aldosterone synthase (CYP11B2) -344T/C polymorphism is not associated with the initiation and progression of diabetic nephropathy in Caucasian Type 1 diabetic patients. *Diabet Med* **23**:675-680.
9. Borch-Johnsen K, Kreiner and Deckert T (1986). Mortality of Type 1 (insulin- dependent) diabetes mellitus I Denmark: a study of relative mortality in 2930 Danish Type 1 diabetic patients diagnosed from 1933 to 1972. *Diabetologis* **29:** 767-772.
10. Benardot D and Czerwinski C (1991). Selected body composition and growth measures of Junior elite gymnasts. *Journal of the American Dietetic Association***91**: 29-33.
11. Trinder P (1989). Determination of Blood glucose using an oxidase peroxidase system with a non-carcinogenic Chromogen. *Ann Clinical Biochemistry***6**: 24-30.

12. H L Rosenthal (1955). Analytical Chemistry Determination of Urea in Blood and Urine with Diacetylmonoxime method, **27(12)**:1980-1982.

13. Chaston Chapman A (1909). Jaffe's colorimetric method for the estimation of serum creatinine, *Royal Society of Chemistry publishers* **404**:475- 483.

14. Sabine Landau and Brain S Everitt (2004). A Handbook of Statistical Analyses using SPSS. *Chapman CRC press company*, New York.

15. Jungers P,Chauveau P, Descamps-Latscha B and Labrunie M (1996) Age and gender-related incidence of chronic renal failure in a French urban area - a prospective epidemiologic study, Nephrol Dial *Transplant* **11**:1542-1546.

16. Zimmet PZ JL (2010). The economic burden of ESRD in Canada The economic burden of ESRD in Canada. *Kidney International* **72**: 1122- 1129.

17. Vuppuruti S and Sandler D (2003). Lifestyle Rish Factors and Chronic Kidney Disease. *Elsevier Inc* **10(13):** 712-720.

18. Collard CD, Vakeva A and Morrissey MA (2000). Complement activation after oxidative stress: role of the lectin complement pathway. *Am J Pathol* **156:**1549 - 1556.

19. Smith CJ, Fischer TH (2001). Particulate and vapor phase constituents of cigarette mainstream smoke and risk of myocardial infarction. *Atherosclerosis* **158**:257-267.

20. Pryor WA and Stone K (1993). Oxidants in cigarette smoke. Radicals, hydrogen peroxide, peroxynitrate, and peroxynitrite. *Ann N Y Acad Sci* **686**:12-27.

21. Marre M, Chatellier G and Leblanc H *(*1988). Prevention of diabetic nephropathy with enalapril in normotensive diabetics with microalbuminuria. *Bmj* **297**:1092-1095.

22. The Diabetes Control and Complications Trail/ Epidemiology of Diabetes Interventions and Complications Research Group (2000). Retinopathy and Nephropathy in patients with type 1 diabetes four years after a trail of intensive therapy. *N England J Med* **342:** 381-389.