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Original Research Article

A Comparative Study of Serum Creatinine, Urea, Uric Acid, Sodium, and Potassium in Diabetic and Non-Diabetic Patients

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Abstract

Background: Diabetes mellitus (DM) has become a global health problem due to urbanization, increasing prevalence of obesity and physical inactivity. Diabetes is characterized by chronic hyperglycemia that is, high blood glucose level due to complications with carbohydrate, protein and fat metabolism. Diabetes mellitus is associated with absolute or relative deficiencies in insulin secretion, insulin action or both. Diabetic kidney disease or diabetic nephropathy is a major complication that take place in 20% to 40 % of all diabetics. In the western world, diabetic kidney is a primary single cause of end stage renal disease. The aim of this study was to access the value of these parameters and evaluate their correlation in diabetic and non-diabetic subjects.

Material & Method: 100 individuals included in this study in which 50 persons with type 2 diabetes mellitus are taken as case and 50 healthy persons were taken as control in this study using accepted techniques fasting and post prandial glucose, HbA1C, Serum creatinine, urea, uric acid, sodium and Potassium were calculated the increased level of the serum creatinine level and blood glucose were seen in type 2 diabetes mellitus patients while serum urea, uric acid ,sodium and Potassium were found insignificant.

Conclusion: The present study shows positive correlation between serum creatinine and blood glucose levels in comparison to controls.

Keywords: FBS, PP Blood Glucose, HbA1C, Serum Creatinine, Urea, Uric Acid, Sodium and Potassium.
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Introduction

Diabetes mellitus (DM) has become a global health problem due to urbanization, increasing prevalence of obesity and physical inactivity [1]. Diabetes is characterized by chronic hyperglycemia that is, high blood glucose level due to complications with carbohydrate, protein and fat metabolism. Diabetes mellitus is associated with absolute or relative deficiencies in insulin secretion, insulin action or both [2].

When levels of glucose fall to a normal range, production of insulin is stopped until the level of glucose is high again. When the body fails to lower blood glucose level on its own, diabetes mellitus, a chronic health condition develops. Diabetic patients develop symptoms such as: weight loss, polyuria (frequent urination), polydipsia (increased thirst) and polyphagia (increased hunger) as a compensatory way of maintaining osmolality in the body.[3] World health organization (WHO) statistics shows the high prevalence of diabetes mellitus which has affected more than 170 million people worldwide and it is predicted to be raised to 370 million by 2030[4].Until recently, India had more diabetics than any other country in the world, according to the International Diabetes Foundation, although the country has now been surpassed in the top spot by China. Diabetes currently affects more than 62 million Indians, which is more than 7.1% of the adult population. The average age of its onset is 42.5 years. Nearly 1 million Indians die due to diabetes every year.

According to the Indian Heart Association, India is projected to be home to 109 million individuals with diabetes by 2035.A study by the American Diabetes Association reports that India will see the greatest increase in people diagnosed with diabetes by 2030. The high incidence is attributed to a combination of genetic susceptibility plus adoption of a high-calorie, low-activity lifestyle by India's growing middle class.[5] Diabetes mellitus is

classified primarily into Type 1 (Insulin dependent) and Type 2 (non insulin dependent).

Type 1 diabetes mellitus is mainly idiopathic caused by autoimmune disorders. The disease is characterized by an absolute deficiency of insulin caused by an autoimmune attack on islet β -cells of pancreas. The islet of langerhans become infiltrated with activated T lymphocytes, leading to a condition called insulitis. Over a period of years, this autoimmune attack on β-cells leads to gradual depletion of the β-cell population. However, symptoms appear abruptly when 80%-90% of the β-cells have been destroyed. At this point, the pancreas fails to respond adequately to ingestion of glucose, and insulin therapy is required to restore metabolic control and prevent life threatening ketoacidosis.[6]

Type 2 diabetes mellitus arises from insufficient production of the hormone insulin from beta cells of pancreas or in conditions where the peripheral receptors; primarily muscles, liver and fat tissue do not respond adequately to normal insulin levels known as insulin resistance [7].

Instead of the body converting glucose into energy, glucose builds up in the bloodstream causing hyperglycemia. Type 2 diabetes makes up about 90% of cases of diabetes and has a slow and insidious onset. It usually occurs in people who are over forty years of age, are obese, with a sedentary lifestyle, poor diet, hypertension and family history of diabetes.[8]Women with a history of gestational diabetes, which is defined as any degree of glucose intolerance which is first recognized during pregnancy are at an increased risk of developing Type 2 diabetes.[9]

Epidemiological data shows that about 80% of Type 2 diabetic patients are obese, with a body mass index of 30 kg/m more, whereas the other 20% have a body mass index 25-29.9 kg/m which is above expected body mass index.[10] Other data shows there are increasing incidences of Type 2 diabetes mellitus and diabetic patients are at an increased risk of developing complications such as: nephropathy, retinopathy and atherosclerosis.[11]

Diabetes and renal function test-Diabetic kidney disease or diabetic nephropathy is a major complication that take place in 20% to 40 % of all diabetics. In the western world, diabetic kidney is a primary single cause of end stage renal disease [12]. In diabetic nephropathy various serum markers are likely to be deranged which show the indication of disease progression. Such indicators of renal dysfunction are serum creatinine, uric acid, urea, as well as electrolytes such as Sodium, Potassium etc. Measurement of serum creatinine, urea, uric acid, as well as sodium and potassium are different parameters of renal function tests which can assist in detection and prevention of diabetic renal disease at an early stage and can limit the progression to end stage renal disease (ESRD) [13,14].

The aim of this study is to access the value of these parameters and evaluate their correlation in diabetic and non diabetic subjects. The variation in value of serum creatinine, urea, uric acid and sodium/potassium in relation to blood sugar level will also be studied to find out their usefulness for the prognosis and diagnosis of diabetic disease complications.

Material and Methods

The present study was conducted in Department of Biochemistry, Rama Medical College Hospital & Research Centre, Mandhana, Kanpur. A total of 100 persons were taken in this study in which 50 Patients having diabetes mellitus were taken as case group and 50 healthy subjects of same age group were taken as control. A Performa was made and detailed clinical profile, medical history of all the patients was recorded.

Biochemical Analysis: Overnight fasting blood sample of 5ml was collected with dry disposable syringe and needle in a plain vacutainer tube under all aseptic conditions after explaining the procedure to the study subjects for estimation of Fasting Blood glucose, post parindal, and serum creatinine, urea, uric acid, sodium and potassium were taken and sent to laboratory. Auto analyzer was used for evaluation of serum glucose both fasting and post prandial and parameters of kidney function test and sodiun and potassium .all the results were recorded in Microsoft excel sheet and were subjected to statical analysis using SPSS-20 software. Chi-square test and student t-test were used for evaluation of level of significance.

Result

In the present study total hundred subjects of age between 31 to 60 years were enrolled as study subjects out of which fifty healthy non-diabetic subjects were taken as control and fifty diabetic subjects diagnosed of diabetic disease were taken as cases. All the subjects were subjected to detailed history-taking as proforma. Test parameters were tabulated as per the master chart. The results were expressed in terms of mean ± SD. The p value <0.05 was considered as significant. The result of the study was depicted into 9 different tables. Table number 1 and 2 and figure number 1.4 and 1.5 showed age wise distribution of study subjects. The mean±SD in diabetic was 46.65±8.818 and non-diabetic was 40.40±10.420 which was found to be not significant (p=0.144). Table number 3 and figure number 1.6showed fasting blood glucose (FBS) between diabetic and non-diabetic. The mean±SD in diabetic was 167.80±45.424 and non-diabetic was 86.4±10.231, which was found to be

significant (p=0.000). Table number 4 and figure number 1.7 showed the postprandial blood glucose (PPBS) in diabetic and non-diabetic. The mean±SD in diabetic was 230.04±78.446 and non-diabetic was128.68±19.221, which was found to be significant (p=0.000). Table number 5 and figure number 1.8 showed the comparison of serum creatinine in diabetic and non-diabetic. The mean±SD in diabetic was 1.7±0.533 and non-diabetic was 0.91±1.430, which was found to be significant (p=0.001). Table number 6 and figure number 1.9 showed the comparison of serum urea in diabetic and non-diabetic. The mean±SD indiabetic was 46.8±13.768 and in non-diabetic was 27.74±9.57, which was found to be not significant (p=0.420). Table number 7 and figure number 1.10 showed the comparison of serum uric acid in diabetic and non-diabetic. The mean±SD in diabetic was 6.37±1.946 and in non-diabetic was 4.76±2.332, which was not to be significant (p=0.772). Table number 8 and figure number 1.11 showed the comparison of serum sodium in diabetic and non-diabetic. The mean±SD in diabetic was 147.70±4.422 and in non-diabetic was 137.14±0.607, which was found to be not significant (p=0.761). Table number 9 and figure number 1.12 showed the comparison of serum potassium in diabetic and non-diabetic. The mean±SD in diabetic was 6.44±0.607 and in non-diabetic was 4.17±0.464, which was not found to be significant (p=0.117).

Table 1: Age wise distribution of stud subjects

Age in years	Cases	Controls
31-35	8	9
36-40	7	12
41-45	8	6
46-50	11	9
51-55	7	7
56-60	9	7
Total	50	50
Mean±SD	46.56±8.44	44.49±8.12

Samples are age matched with P =0.144

Table 2: Comparison of age between Diabetic and non-Diabetic

	Number	Mean	SD	T	p value
Diabetic	50	46.56	8.818	3.190	0.144
Non-diabetic	50	40.40	10.420	3.190	0.144

The mean±SD in diabetic was 46.65±8.818 and non-diabetic was 40.40±10.420 which was found to be not significant (p=0.144).

Table 3: Comparison of fasting blood glucose (FBS) between diabetic and non-diabetic

	Number	Mean	SD	T	p value
Diabetic	50	167.80	45.424	12.386	0.000
Non-diabetic	50	86.4	10.231	12.386	0.000

The mean±SD in diabetic was 167.80±45.424 and non-diabetic was 86.4±10.231, which was found to be significant (p=0.000)

Table 4: Comparison of postprandial blood glucose (PPBS) in diabetic and non-diabetic

	Number	Mean	SD	T	p value
Diabetic	50	230.04	78.446	8.874	0
Non-diabetic	50	128.68	19.221	8.874	0

The mean±SD in diabetic was 230.04±78.446 and non-diabetic was128.68±19.221, which was found to be significant (p=0.000).

Table 5: Comparison of serum creatinine in diabetic and non-diabetic

	Number	Mean	SD	t	p value
Diabetic	50	1.7	0.533	1.696	0.001
Non-diabetic	50	0.91	1.430	1.696	0.001

The mean±SD in diabetic was 1.7±0.533 and non-diabetic was 1.2±1.430, which was found to be significant (p=0.001).

Table 6: Comparison of serum urea in diabetic and non-diabetic

	Number	Mean	SD	t	p value
Diabetic	50	46.8	13.768	0.447	0.420
Non-diabetic	50	27.74	9.57	0.447	0.420

The mean±SD indiabetic was 46.8±13.768 and in non-diabetic was 27.74±9.57, which was found to be not significant (p=0.420).

Table 7: Comparison of serum uric acid in diabetic and non-diabetic

	Number	Mean	SD	T	p value
Diabetic	50	6.37	1.946	1.429	0.772
Non-diabetic	50	4.76	2.332	1.429	0.772

The mean±SD in diabetic was 6.37±1.946 and in non-diabetic was 4.76±2.332, which was not to be significant (p=0.772).

Table 8: Comparison of sodium in diabetic and non-diabetic

	Number	Mean	SD	T	p value
Diabetic	50	147.70	4.422	0.633	0.761
Non-diabetic	50	137.14	0.607	0.633	0.761

The mean±SD in diabetic was 147.70±4.422 and in non-diabetic was 137.14±0.607, which was found to be not significant (p=0.761).

Table 9: Comparison of potassium in diabetic and non-diabetic

	Number	Mean	SD	T	p value
Diabetic	50	6.44	0.607	2.556	0.117
Non-diabetic	50	4.17	0.464	2.556	0.117

The mean±SD in diabetic was 6.44±0.607 and in non-diabetic was 4.17±0.464, which was not found to be significant (p=0.117).

Table 10: Comparison of serum creatinine, urea, uric acid, sodium and potassium in diabetics and non-diabetics

Parameter		Cases	Controls	P value
FBS(mg/dl)	Mean ± SD	167.80±45.424	86.4±10.23	0.000
PP(mg/dl)	Mean ± SD	230.04±78.446	128.68±19.221	0.000
urea(mg/dl)	Mean ± SD	46.80±13.768	27.74±9.570	0.001
creatinine(mg/dl)	Mean ± SD	1.70±1.553	1.20±1.430	0.420
Uric acid(mg/dl)	Mean ± SD	6.37±1.98	4.76±2.332	0.772
Na^{+} (mmol/l)	Mean ± SD	147.70±5.423	137.14±4.422	0.761
K^{+} (mmol/l)	Mean ± SD	6.98±1.607	4.17±0.464	0.117

Discussion

Different studies showed that a physically active lifestyle is associated with a lower incidence of type-2 diabetes. Akinkughbe et al reported in a study that the prevalence of diabetes is more in people who are engaged in light physical activity work, Nyenwe et al study revealed that, less physical activity was significantly associated with increased risk for DM.

Abebe et al reported in a study physical inactivity were significantly associated with diabetes mellitus. In present study family history of diabetes was significantly higher among diabetics than non-diabetics in the study and it was found to be statistically significant.

In a study done by Amarsingher et al it was found that those individuals with family history of diabetes were diabetic compared to those without family history of diabetes. Similar findings were observed in a study done by Nyenwe et al. Many studies have indicated the association between FBS and PPBS with HbA1c level table 3-4, and fig,1.6-1.7 shows that mean FBS and PPBS level was significantly (p<.001) elevated in patients with poor glycemic control (HbA1c>7%) similar to the study reported by Khattab et al.

As similar our study elevated FBS and PPBS level is similar finding; concluded type 2 diabetic patients was elevated FBS and PPBS level. Table 1 and figure 1.4 Shows that both cases and controls are age matched with p value of 0.144.

Conclusion

The observations made in the study were as follows:

- Serum Creatinine level was significantly (p=0.001) higher among cases (1.7±0.533) as compared with controls (1.2±1.430).
- FBS was significantly (p=0.000) higher among cases (167.80±45.424) than controls (86.4±10.231). As well as Postprandial was

significantly (p=0.000) higher among cases (230.04±78.446) as compared with controls (28.68±19.221).

- But the levels of serum Urea, Uric Acid, sodium and potassium was not to be significant.

A significant association was obtained in the Serum Creatinine level between the diabetic and non-diabetic patients. On the otherside the rest of the parameters like FBS, PPBS, Urea, Uric Acid, Sodium and Potassium, did not show any significant association between diabetic and non-diabetic subjects.

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