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Original article

COMPARATIVE STUDY: CORRELATION OF UREA AND SERUM CREATININE WITH DURATION OF DIABETES AND GLYCEMIC INDEX IN INDIVIDUALS WITH TYPE 1 AND 2 DIABETES MELLITUS.

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ABSTRACT.

Background:
Kidney failure and nephropathy, defined as a dysfunctional kidney with a decline in ultrafiltration and a rise in blood urea and creatinine levels, are frequently caused by diabetes.
Aim: The motive of this research was to evaluate the concentration of blood urea and creatinine in individuals with diabetes, and also to examine the relationship between these parameters and the duration of diabetes as well as glycosylated hemoglobin concentration.

Methods:
Concentrations of urea and creatinine in the blood were evaluated in samples of Juvenile diabetes and Diabetes mellitus patients attending diabetic clinics as well as non-diabetic patients in a tertiary hospital. For the study, 144 male participants between the ages of 35 and 55 were chosen for each group. All trial participants' fasting blood sugar, post-meal blood sugar parameters, and glycosylated hemoglobin were ascertained. The relationship between blood creatinine and urea levels, glycosylated hemoglobin, and length of illness in all diabetes participants was examined.

Results:
In the type 1 study group, blood creatinine and urea levels were correlated with glycosylated hemoglobin levels and the length of diabetes, but not with the Diabetes mellitus study group. Serum creatinine (F-value = 50.96) and urea (F-value = 33.4) levels increased statistically significantly in the diabetes groups relative to the control group.

Conclusion:
Creatinine and urea are straightforward and practical indicators that can be used as predictive assays to evaluate the condition of the kidneys (nephropathy) in individuals with diabetes.

Recommendations:
Intensive treatment can address elevated HbA1c levels in diabetes, but it may not reverse rising serum urea and creatinine caused by permanent kidney damage. Early detection and intervention are crucial to control glomerular injury and prevent further increases in serum urea and creatinine levels.

Keywords: *Serum creatinine, Urea, Type I diabetes, Type II diabetes*
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INTRODUCTION.

In India, diabetes mellitus is potentially becoming an epidemic. Diabetic complications can lead to a significant amount of morbidity and mortality [1]. Chronic hyperglycemia associated with diabetes is linked to organ damage and failure, particularly in the kidneys, eyes, nerves, heart, and vascular system. According to certain estimates [1, 2], Nephropathy caused by diabetes can occur in 25–45% of diabetic people and it is clinically noticeable throughout their lives. In those with Juvenile diabetes, the peak onset of nephropathy occurs 10 to 15 years after the disease first appears. After 25 years, there is a 1% annual risk of overt renal disease development for persons without proteinuria [2].

Hyperglycemia in diabetes results from too much glucose combined with unbound amino acids on tissue or circulatory proteins. Through an Amadori rearrangement, this non-

enzymatic mechanism first produces early glycosylation compounds that are reversible, which are subsequently end products of irreversible progressive glycosylation. By cross-linking with collagen, the tissue build up of AGEs may be a factor in the related renal and microvascular problems [2]. The occurrence of diabetic nephropathy can be decreased with proper glycemic management. Microalbuminuria is the first discernible anomaly of nephropathy, and it is followed by a rise in the levels of serum creatinine and a glomerular filtration rate falling (GFR) [3].

Uncontrolled diabetes may cause hyperglycemia, which is linked to an abnormal rise in serum creatinine and blood urea. Thus, the two key indicators for identifying any kidney problem are urea and creatinine. While the generation of urea depends on various factors such as liver function, protein consumption, and rate of protein degradation, changes in serum creatinine will more consistently reflect changes in GFR. Thus, measuring serum creatinine and blood urea helps stop the advancement of end-stage renal disease and aids in the early detection and prevention of diabetic kidney disorders. We set out to assess blood urea and serum creatinine levels in diabetes patients and compare these parameters in non-diabetic patients because renal problems are more common in diabetic patients [3].

Objectives.

- Assess and contrast the creatinine and urea levels between individuals with Juvenile diabetes and Diabetes mellitus and those without the condition.
- Establish a relationship between the length of diabetes and the glycosylated hemoglobin levels in individuals with diabetes and the concentrations of these chemicals.

MATERIALS AND METHODS.

Study design.

With institutional ethical review committee approval, this investigation was designed to be carried out to evaluate the concentration of blood urea and creatinine in individuals with diabetes, also to examine the relationship between these parameters and the duration of diabetes as well as glycosylated hemoglobin concentration, in Darbhanga Medical College and Hospital, Laheriasarai, Bihar, India from September 2022 to till August 2023. Males between the ages of 30 and 55 made up all the subjects. The study included 144 patients with Juvenile diabetes and Diabetes mellitus who attended the diabetic clinic, as well as 144 non-diabetic participants who were chosen as a control group from the general community.

Study setting.

The study was carried out on 288 subjects divided into three groups

1. Group 1 - Juvenile diabetes with insulin-dependent diabetes mellitus (IDDM) n = 72
2. Group 2 - Diabetes mellitus with non-insulin-dependent diabetes mellitus (NIDDM) n = 72
3. Group 3 - non-diabetic patients (control group) n = 144

The anthropometric characteristics listed below were examined:

- Age: Using a calendar, the age was calculated from birth to the nearest month (<6 and >6 months).
- Height: Using a height measuring stadiometer, the measurement was made in centimetres.
- Weight in body: It was calculated using a portable human scale, expressed in kilograms
- Body mass index (BMI)

Data collection.

Using commercially available kits that were modified for auto analyzers, the parameters of the biochemistry were evaluated in the clinical laboratory. All the diabetes patients had blood samples taken in an ethylenediamine tetra acetic acid bulb to evaluate the relationship between post-meal plasma glucose and fasting. Using the glucose oxidase and peroxidase technique, serum glucose was estimated [4]. The alkaline Jaffe's Picrate method [5] was used to quantify creatinine, while Berthelot's method [6] was used to determine serum urea. A clinical chemistry analyzer that is automated was utilized to ascertain these biochemical parameters. For creatinine, the normal range was 0.8–1.4 mg/dL, while for urea, it was 10-45 mg/dL [7]. Using the diagnostic glycosylated hemoglobin kits from Asritha Diatech, the HbA1c of each study participant was determined using the ion exchange resin method by the guidelines [8].

Statistical analysis.

The results were analyzed using the Statistical Package for Social Sciences (SPSS) version 21. The significance of the difference of the mean was calculated using Analysis of Variance (ANOVA).

Ethical considerations.

The ethical aspects of the research were carefully thought out to preserve patient privacy and confidentiality. An institutional research committee ethics clearance letter was obtained before patient data was accessed.

Bias.

There was a chance that bias would arise when the study first started, but we avoided it by giving all participants identical information and hiding the group allocation from the nurses who collected the data.

RESULTS.

Juvenile diabetes with insulin-dependent diabetes mellitus (IDDM, N=72), Diabetes mellitus with non-insulin-dependent diabetes mellitus (NIDDM, N=72), and non-diabetic patients (control group, N=144) all had anthropometric measurements that were listed in mean ± SD in Table 1. The subjects' age, height, weight, and BSA did not significantly differ across the three groups.

Table 1: Human-metric parameters.

Specifications	Groups	N	Mean ± SD	Median	IQR	ANOVA test	
						F value	P value
Age (yrs.)	IDDM	144	47.01±5.89	45	10	1.495	0.227
	NIDDM	144	48.5±4.78	48	8		
	Control	144	48.1±5.62	48	10		
Height (cm)	IDDM	144	161.21±7.37	162	9.75	0.795	0.453
	NIDDM	144	162.11±5.94	162	9		
	Control	144	162.57±6.38	163	7		
Weight (kg)	IDDM	144	54.86±5.44	55	6.75	1.603	0.204
	NIDDM	144	56.07±6.99	56	8		
	Control	144	56.54±4.77	56	6.75		
BMI	IDDM	144	20.74±2.78	20.52	3.54	2.500	0.084
	NIDDM	144	21.65±3.3	21.66	4.48		
	Control	144	21.64±2.27	21.19	3.56		

Age, weight, height, and BSA did not substantially differ among those participating among the three groups. Table 2 displays glycosylated hemoglobin, fasting blood sugar, and post-meal blood sugar levels, and the groups with control, Juvenile diabetes, and Diabetes mellitus. Juvenile diabetes and Diabetes mellitus had considerably higher glycosylated hemoglobin levels when compared to the control group.

Table 2: Comparison among groups for Fasting blood sugar, HbA1c, and Post meal blood sugar.

Parameters	Groups	Mean ± SD	Median	IQR	ANOVA test	
					F value	P value
FBS (mg/dL)	IDDM	87.58±7.8	88	7.5	46.295	0.0
	NIDDM	97.5±11.5	96	12		
	Control	83.8±6.5	86	6		
PBS (mg/dL)	IDDM	162.9±24.5	170	49	126.198	0.0
	NIDDM	171.42±14.60	172	25		
	Control	127.93±9.85	128	9		
HbA1c (%)	IDDM	5.98±0.32	5.9	0.58	3.407	0.035
	NIDDM	6.62±0.03	5.8	0.6		
	Control	5.11±0.30	5.1	0.5		

Creatinine and urea levels in healthy individuals (control), Juvenile diabetes, and Diabetes mellitus are displayed in Table 3 as mean ± SD. Serum creatinine and urea levels increased statistically significantly in the diabetes groups relative to the control group.

Table 3: Differences among groups for serum urea and creatinine.

Parameters	Groups	Mean ±SD	Median	IQR	ANOVA test	
					F value	P value
Creatinine (mg/dL)	IDDM	1.31±0.19	1.3	0.1	50.96	0.0
	NIDDM	1.28±0.16	1.3	0.2		
	Control	1.03±0.19	1.1	0.28		
Urea (mg/dL)	IDDM	31±3.67	32	6	33.4	0.0
	NIDDM	31.13±3.1	31	5		
	Control	27.2±2.9	27	2		

Table 4 displays the Pearson's coefficient association between serum creatinine and urea, glycosylated hemoglobin, and the length of diabetes in the groups with insulin-dependent diabetes/Juvenile diabetes and non-insulin-dependent diabetes /Diabetes mellitus. Serum urea and creatinine in the Juvenile diabetes group were related to the length of diabetes and the glycosylated hemoglobin level, but not the Diabetes mellitus group.

Table 4: relation of factors including duration of diabetes and the glycosylated hemoglobin.

NIDDM	*Duration*			*HbA1c*		
	Pearson correlation	*P value*	*Correlation*	*Pearson correlation*	*P value*	*Correlation*
Urea	0.096	0.420	Not much	0.118	0.325	Not much
Creatinine	-0.033	0.783	Not much	0.093	0.438	Not much
HbA1c	-0.122	0.307	Not much			
Duration				-0.122	0.307	Not much
IDDM	Duration			HbA1c		
	Pearson correlation	P value	Correlation	Pearson correlation	P value	Correlation
Urea	0.197	0.096	Not much	0.28	0.016	Vital
Creatinine	0.196	0.099	Not much	0.275	0.019	Vital
HbA1c	0.716	0.0	Vital			
Duration				0.716	0.0	Vital

DISCUSSION.

A significant contributor to morbidity and death is diabetes mellitus. The kidney disease that develops because of diabetes is called diabetic nephropathy. According to an international investigation [9] there are many complications, but neuropathy was the most frequent complication, followed by issues with the heart, kidneys, retina, and foot ulcers. Diabetes control deteriorated with a prolonged illness duration. Juvenile diabetes and Diabetes mellitus can result in diabetic kidney failure [9]. Both forms of diabetes have different kidney pathologies and incidences [10]. Even though type 1 diabetes was thought to have a significantly greater overall incidence of nephropathy [11], more recent research indicates that the risk to the kidneys is the same in both types of diabetes [12].

Despite research on the concentration of creatinine, urea, and microalbuminuria in Diabetes mellitus, as well as comparison studies between Juvenile diabetes and Diabetes mellitus for identical variables have rarely been reported. Few research [12-14] has examined the relationship between blood creatinine and urea levels, which are indicators of GFR, and the length of diabetes and the glycemic index (glycosylated hemoglobin levels). Nephropathy is a consequence of long-term diabetes that has been linked to microalbuminuria and hypertension in numerous investigations [12-14]. Although the precise origin of diabetic nephropathy is uncertain, advanced glycation products, hyperglycemia, and cytokine activation are some of the suggested reasons. Many writers [12-14] have emphasized that low glycemic status is a major contributor to diabetic nephropathy, yet little is known about the connection between blood creatinine and urea levels and the glycemic state (HbA1c levels) of diabetic individuals.

Many people acknowledge that insufficient control of glucose, or hyperglycemia, is among the main characteristics of diabetes mellitus [15]. In this study, Serum creatinine and urea levels in the study group of Juvenile diabetes and Diabetes mellitus were statistically substantially higher than in the group of healthy subjects, with higher in people with Diabetes mellitus than in the Juvenile diabetes population. When compared to healthy subjects, Patients with Juvenile diabetes and Diabetes mellitus had higher post-meal, fasting blood glucose, and glycosylated hemoglobin values; the type 1 diabetic study group had greater levels than the type 2 diabetic study group.

For Juvenile diabetes and Diabetes mellitus, the length of diabetes was 7.32 ± 1.52 and 2.47 ± 1.82 years, respectively. Compared to Diabetes mellitus, Juvenile diabetes individuals had longer diabetes durations and higher HbA1c levels. Prior research conducted by multiple investigators has documented elevated concentrations of blood creatinine and urea in individuals with diabetes [16-18].

CONCLUSION.

In Type 1 diabetic participants, a linear association between blood creatinine and urea level was seen in response to elevated HbA1c levels. It is strongly advised to estimate blood urea and creatinine levels in addition to HbA1c levels to monitor diabetic patients. Maintaining appropriate blood pressure and blood glucose levels lowers the risk of developing renal impairment, which is typically linked to both diabetes and hypertension. The co-morbidity of hypertension and diabetes increases the risk of kidney disease development. Since serum urea and serum creatinine are significant indicators of renal impairment, individuals with diabetes and hypertension, as well as those with hypertension and diabetes, should have their levels regularly examined.

LIMITATION.

The limitations of the study are that we need more study population at different intervals to validate our findings and ascertain if these modifications are transient or permanent. It is necessary to investigate the effects of additional potential causes.

RECOMMENDATION.

Although an intensive treatment plan can lower the elevated HbA1c levels, it will be difficult to reverse the elevated levels of serum urea and creatinine that are on the rise due to permanent kidney damage, which is a permanent phenomenon in diabetes mellitus. Early detection and intervention would be the sole method to control this increasing glomerular injury and the resulting high levels of serum and creatinine web.

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CONFLICT OF INTERESTS.

No conflicts of interest are disclosed by the authors.

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