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Correlation of Urinary 8-Hydroxy-Deoxyguanosine with Clinical Markers of Nephropathy in Patients with Type 2 Diabetes Mellitus

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KEYWORDS

Type 2 Diabetes Mellitus, Nephropathy, Urinary 8-Hydroxy-Deoxyguanosine, and Serum Magnesium

ABSTRACT:

Introduction: Kidney problems is most common in patients with Type 2 Diabetes Mellitus. For detection of kidney problems need a sensitive and specific marker.

Objectives: Present study aimed to Correlate urinary 8-hydroxy-deoxyguanosine with clinical markers of nephropathy in patients with Type 2 Diabetes Mellitus.

Methods: This cross-sectional analytical study included 180 Type 2 Diabetes Mellitus patients and 60 controls. For all the study subjects we analysed fasting blood sugar, HbA1c, lipid profile, urinary albumin creatinine ratio, eGFR, serum magnesium, and urinary 8OHdG.

Results: There was significantly decreased serum magnesium levels in all the groups of Type 2 Diabetes Mellitus patients when compared to controls. There was significantly increased urinary 8-hydroxy-deoxyguanosine levels in all the groups of Type 2 Diabetes Mellitus patients when compared to controls. Additionally, the serum magnesium negatively corelated with urinary albumin creatinine ratio and 8-hydroxy-deoxyguanosine and positively corelated with eGFR. Similarly, 8-hydroxy-deoxyguanosine positively corelated with urinary albumin creatinine ratio and serum magnesium and negatively corelated with eGFR.

Conclusions: Based on study results determination of magnesium beneficial for type 2 diabetes mellitus patients and urinary 8-hydroxydeoxyguanosine might be used as early predictive and prognostic marker for nephropathy in type 2 diabetes mellitus patients.

1. Introduction

Type 2 Diabetes Mellitus (T2DM) is a long-term illness marked by elevated blood sugar levels. It can lead to a number of consequences, the most serious of which is chronic kidney disease. An imbalance between the body's antioxidants and free radicals leads to oxidative stress, which is a major factor in the etiology of T2DM and the emergence of its consequences (1-2). A surplus of free radical's harms cells by interfering with the structure and functionality of proteins, lipids, and DNA (3).

Magnesium is a macro element act as a cofactor for many enzymatic reactions and involved in range of physiological processes include energy metabolism, glycolysis, and protein synthesis. Furthermore, extracellular magnesium ions cause an increase in intracellular calcium and iron ions, which in turn causes a rise in peroxide formation and a decrease in their clearance as well as the activation of inflammatory pathways (4-5). Consequently, the alteration of magnesium ions is linked to chronic inflammatory disorders like atherosclerosis and chronic kidney disease (CKD) and can cause pathological alterations like insulin resistance and endothelial cell death in patients with T2DM (6).

The recent studies reported 8 hydroxy-20-deoxyguanosine (8-OHdG) is directly linked to DNA

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damage; it is a more specific sign of oxidative stress-induced harm than the many other oxidative stress biomarkers. The 8-OHdG specifically evaluates the degree of oxidative stress to the DNA level, which is important in T2DM and associated consequences (7-8). This is in contrast to other metabolites that represent overall oxidative stress levels in the body. Increased oxidative stress and possible genetic damage are caused by elevated 8 OHdG levels in T2DM individuals, which may have long-term effects on cellular activity and general health. This special property of 8 OHdG offers important insights into the mechanisms underlying diabetic complications such renal disease that are linked to oxidative stress (9-10).

The oxidative DNA damage occurs when the base excision and repair mechanism eliminates oxidized guanosine from nuclear and mitochondrial DNA, producing 8-hydroxy-2'-deoxyguanosine (8-OHdG) as a by-product. Oxidative stress has been identified as a common cause of T2DM complications, including nephropathy (11-12). Thus, 8-OHdG is a marker for diabetic nephropathy and oxidative DNA damage.

2. Objectives

Present study aimed to Correlate urinary 8-hydroxy-deoxyguanosine with clinical markers of nephropathy in patients with Type 2 Diabetes Mellitus.

3. Methods

This cross-sectional study was carried out department of biochemistry and medicine at the Raichur Institute of Medical Sciences in Raichur, Karnataka. A total of 180 patients were diagnosed with type 2 diabetes based on the American Diabetic Association Criteria (13) and 60 healthy controls who were matched by age, gender, and Body Mass Index (BMI). All study participants were hired following Institutional Ethics Committee (IEC) clearance and completion of an informed consent form. The study groups that are displayed in Table 1.

Criteria of the study

The current analysis included patients with type 2 diabetes who were between the ages of 30 and 70 and had varying degrees of nephropathy as determined by the kidney disease improving global outcomes (KDIGO) criteria (14). Participants were excluded from this study if they had a history of type 1 diabetes mellitus, non-diabetic renal illness, thyroid and liver disease, macrovascular complications like peripheral vascular, cardiovascular, and cerebrovascular diseases, active inflammatory disease, or urinary tract infections (15-16).

Collection of samples

Six millilitres of fasting venous blood sample were drawn from each study participant. Four millilitres of the blood sample were transferred in a plain tube, one millilitre was transferred in a fluoride tube, and one millilitre was transferred in an Ethelin Diamino Tetra Acetic acid tube. Additionally, a spot urine sample was collected. All collected samples were separated by centrifuging them for ten minutes at 3000 rpm. All the separated samples were transferred into appropriately labelled aliquots. The samples stored at -800 C until analysis was performed.

Methods

The routine biochemical parameters like blood sugar, total cholesterol, triglycerides, high density lipoprotein, urine albumin, urine creatinine, serum magnesium, and HbA1c was determined by laboratory techniques. The enzyme-linked immunosorbent assay was used to measure the amount of 8-OHdG in the urine. The friedewald's formula was used to determine the low density lipoprotein and very low density lipoprotein. The estimated glomerular filtration rate was determined using a modification of diet in renal diseases.

Statistical analysis

The mean ± standard deviation was used to express the data. The analysis of variance (ANOVA) was used to compare the groups. Pearson correlation analysis was used to determine the relationship between the study's other measures, including serum magnesium and urine 8-OHdG. The receivers operating characteristics (ROC) curve study was performed on T2DM patients with normo albuminuria and controls in order to diagnose and track the evolution of nephropathy early. SPSS version 20 and Microsoft Excel Spreadsheets were used throughout the entire analysis. A P value is deemed significant if it is less than 0.05.

4. Results

Table 2 illustrates age, BMI, blood sugar levels, and lipid profiles such as total cholesterol, TGL, VLDL, and LDL HbA1c, which are all markedly higher in patients

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with type 2 diabetes mellitus patients when compared to the controls (P<0.05). When compared to controls, T2DM patients, eGFR and magnesium levels dramatically dropped (P=0.001**). The T2DM patients had significantly higher urine albumin creatinine ratio and urinary 8-OHdG (P=0.001**).

Table 3 shows age, BMI, blood sugar levels and lipid profiles such as total cholesterol, TGL, VLDL, LDL, and HbA1c, all of which are markedly higher in all patient groups with type 2 diabetes mellitus patients, in contrast to the controls (P<0.05). The T2DM patients with micro and macro albuminuria show significantly increased levels of urine albumin creatinine ratio when compared T2DM with normo albuminuria and controls (P=0.001**). The T2DM patients with micro and macro albuminuria show substantial decrease in both eGFR and magnesium when compared to T2DM with normo albuminuria and controls (P=0.001**). The T2DM patients with normo, micro, and macro albuminuria had significantly higher urinary 8-OHdG levels when compared to controls (P=0.001**).

Table 4 demonstrates a substantial and positive correlation (P=0.001**) between serum magnesium, HDL and eGFR. Additionally, there was a negative correlation (P=0.001**) between serum magnesium and FBS, total cholesterol, TGL, VLDL, LDL, HbA1c, albumin creatinine ratio, and urinary 8-OHdG. There was a substantial and negative correlation between the urinary 8-OHdG, HDL, serum magnesium, and eGFR (P=0.001**). Additionally, there was a positive correlation between urinary 8-OHdG, FBS, total cholesterol, TGL, VLDL, LDL, HbA1c, and albumin creatinine ratio (P=0.001**).

The albumin creatinine ratio and eGFR were not found to be significant at the area under the curve with sensitivity (32, 92) and specificity (87, 22), respectively, where the P value was greater than 0.05. The serum magnesium showed very high significance at the area under the curve, with sensitivity (95) and specificity (97), respectively, the P value was less than 0.05. furthermore, the urinary 8-OHdG has a very high significant area under the curve with sensitivity (72) and specificity (97) respectively P value of less than 0.05 (Table 5).

The T2DM patients with micro and macro albuminuria patients shown drastically significant decrease levels of eGFR when compared those with T2DM patients with normo albuminuria and controls (**figure 1**).

The T2DM patients with micro and macro albuminuria patients shown drastically significant increased levels of albumin creatinine ratio when compared those with T2DM patients with normo albuminuria and controls (**figure 2**).

The T2DM patients with normo, micro and macro albuminuria patients shown drastically significant decrease levels of serum magnesium when compared to controls (**figure 3**).

The T2DM patients with normo, micro and macro albuminuria patients shown drastically significant increased levels of urinary 8-OHdG when compared to controls (**figure 4**).

5. Discussion

The present study observed significantly reduced serum magnesium levels were significantly inversely correlated with obesity in T2DM patients. The recent studies have also shown that magnesium has anti-oxidative stress and anti-inflammatory properties by functioning as a physiological inhibitor of nicotinamide adenine dinucleotide phosphate oxidase and xanthine oxidase, as well as a cofactor of adenosine triphosphate to prevent the generation of reactive oxygen species. Thus, we hypothesized that serum magnesium, primarily through its anti-inflammatory function, protects T2DM patients against the development and progression of obesity and abdominal obesity (17-19). The significant decreased serum magnesium results oxidative stress and tissue damage particularly in patients with T2DM.

Additionally, this study highlight the importance of 8 hydroxy-20-deoxyguanosine (8-OHdG) as a biomarker for DNA damage and oxidative stress in T2DM patients, as well as the fact that the same biomarker has a respectable degree of discriminatory power for identifying kidney disease in T2DM patients (20-21). Similarly, the previous studies also reported type 2 diabetic nephropathy patients to have higher levels of oxidative stress. Increased oxidative DNA damage in DKD is suggested by the higher 8-OHdG levels in these patients as compared to individuals without kidney dysfunction, linking this change to kidney failure in this population (22).

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The present study 8-OHdG with HbA1c, we found positive correlation between them. The novel studies reported the severity of T2DM complications, such as nephropathy, cardiovascular illnesses, and the risk of death in people with T2DM, have also been linked to elevated 8-OHdG levels (23-24). Similarly previous studies also reported consistent with these findings discovered that the risk of DKD in the diabetic population is influenced by glycated hemoglobin levels. Through a number of processes, such as the polyol pathway, the generation of advanced glycation end products (AGEs), and mitochondrial dysfunction, hyperglycemia per se is known to enhance the production of reactive oxygen species (ROS). These ROS have the ability to seriously harm DNA and other biological constituents (25-26).

Hyperglycemia-induced ROS in the kidneys can cause tubular and glomerular damage, which advances chronic kidney disease. The kidneys' high oxygen consumption and metabolic activity make them especially vulnerable to oxidative stress. Diabetes patients' kidneys are more susceptible to oxidative stress, which can worsen fibrosis, apoptosis, and inflammation, further compromising renal function. Patients with CKD who have type-2 diabetes may have high 8-OHdG levels for a number of reasons (27-28). Hypomagnesemia might cause type 2 diabetes mellitus and its complications particularly in kidney. The significant excretion of urinary 8OHdG levels indicate degree of kidney damage in patients with Type 2 diabetes mellitus.

Conclusion

Based on study results determination of magnesium beneficial for type 2 diabetes mellitus patients and urinary 8-hydroxydeoxyguanosine might be used as early predictive and prognostic marker for nephropathy in type 2 diabetes mellitus patients.

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Table 1: Study groups

Group	Subjects	No of subjects
Group 1	Healthy Controls	60
Group 2	T2DM Patients with Normo Albuminuria	60

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Group 3	T2DM Patients with Micro Albuminuria	60
Group 4	T2DM Patients with Macro Albuminuria	60

Table 2: Baseline characteristics of controls and T2DM cases

Parameter	Control			T2DM Cases			P-Value
	Mean ± SD			Mean ± SD			
Age (years)	41.30	±	5.79	49.34	±	7.51	0.001**
BMI (kg/m2)	22.87	±	1.04	30.59	±	6.59	0.001**
FBS (mg/dL)	80.88	±	7.25	165.06	±	27.56	0.001**
HbA1c (%)	4.19	±	0.65	8.85	±	2.20	0.001**
Total Cholesterol (mg/dL)	155.98	±	14.67	264.64	±	78.40	0.001**
TGL (mg/dL)	115.03	±	15.01	195.26	±	61.24	0.001**
HDL (mg/dL)	44.38	±	6.61	32.12	±	5.90	0.001**
VLDL (mg/dL)	23.01	±	3.00	39.05	±	12.25	0.001**
LDL (mg/dL)	88.60	±	15.42	193.47	±	71.88	0.001**
eGFR (ml/min)	89.63	±	7.34	41.32	±	34.51	0.001**
Albumin Creatinine Ratio (mg/g creatinine)	17.74	±	4.73	349.16	±	339.03	0.001**
Serum Magnesium (mg/dL)	2.13	±	0.30	0.76	±	0.37	0.001**
Urinary 8-Hydroxydeoxyguanosine (ng/mL)	1.55	±	0.69	5.41	±	2.47	0.001**

Table 3: Comparison of study variables between subgroups of T2DM patients and controls

Parameter	Control			T2DM with Normo albuminuria			T2DM with Micro albuminuria			T2DM with Macro albuminuria			P-Value
	Mean ± SD			Mean ± SD			Mean ± SD			Mean ± SD			
Age (years)	41.30	±	5.79	42.40	±	4.46	49.03	±	4.74	56.60	±	5.12	0.001**
BMI (kg/m2)	22.87	±	1.04	22.87	±	1.94	31.49	±	2.46	37.42	±	3.61	0.001**
FBS (mg/dL)	80.88	±	7.25	142.00	±	8.01	158.30	±	12.78	194.88	±	24.22	0.001**
HbA1c (%)	4.19	±	0.65	6.94	±	0.64	8.38	±	1.01	11.24	±	1.89	0.001**
Total cholesterol (mg/dL)	155.98	±	14.67	168.00	±	15.17	279.55	±	13.25	346.38	±	41.12	0.001**
TGL (mg/dL)	115.03	±	15.01	126.38	±	19.51	193.40	±	19.56	266.00	±	26.03	0.001**
HDL (mg/dL)	44.38	±	6.61	37.68	±	4.33	31.20	±	3.77	27.48	±	4.33	0.001**
VLDL (mg/dL)	23.01	±	3.00	25.28	±	3.90	38.68	±	3.91	53.20	±	5.21	0.001**
LDL (mg/dL)	88.60	±	15.42	105.05	±	15.47	209.67	±	13.96	265.70	±	41.16	0.001**
eGFR (ml/min)	89.63	±	7.34	88.77	±	7.25	23.89	±	4.96	11.31	±	3.45	0.001**
Albumin Creatinine Ratio (mg/g creatinine)	17.74	±	4.73	16.96	±	4.39	229.02	±	34.67	801.52	±	107.83	0.001**
Serum Magnesium (mg/dL)	2.13	±	0.30	1.13	±	0.32	0.68	±	0.20	0.48	±	0.18	0.001**
Urinary 8-Hydroxydeoxyguanosine (ng/mL)	1.55	±	0.69	3.74	±	1.72	5.00	±	2.19	7.49	±	1.85	0.001**

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Table 4: Correlation of serum magnesium, urinary 8-hydroxydeoxyguanosine, and other study variables

Parameter	Serum Magnesium		Urinary 8-Hydroxydeoxyguanosine	
	r	P-value	r	P-value
BMI (kg/m2)	-0.66	0.001**	0.53	0.001**
FBS (mg/dL)	-0.72	0.001**	0.67	0.001**
HbA1c (%)	-0.74	0.001**	0.70	0.001**
Total cholesterol (mg/dL)	-0.70	0.001**	0.68	0.001**
TGL (mg/dl)	-0.68	0.001**	0.82	0.001**
HDL (mg/dl)	0.61	0.001**	-0.54	0.001**
VLDL (mg/dl)	-0.68	0.001**	0.76	0.001**
LDL (mg/dl)	-0.70	0.001**	0.72	0.001**
eGFR (ml/min)	0.68	0.001**	-0.74	0.001**
Albumin Creatinine Ratio (mg/g creatinine)	-0.69	0.001**	0.65	0.001**
Serum Magnesium (mg/dL)	1	0.001**	-0.63	0.001**
Urinary 8-Hydroxydeoxyguanosine (ng/mL)	-0.63	0.001**	1	0.001**

Table 5: ROC curve analysis for prediction of nephropathy in T2DM Patients with normo albuminuria and controls

Parameter	AUC	95% CI for Value	Cut of Value	Sensitivity	Specificity	P-Value
Albumin Creatinine Ratio (mg/g creatinine)	0.547	0.432 to 0.659	0.2000	32.50	87.50	0.4762
eGFR (ml/min)	0.502	0.388 to 0.616	0.1500	92.50	22.50	0.9698
Serum Magnesium (mg/dL)	0.988	0.934 to 1.000	0.9250	95.00	97.50	<0.0001
Urinary 8-Hydroxydeoxyguanosine (ng/mL)	0.923	0.842 to 0.971	0.7000	72.50	97.50	<0.0001

Figure 1: The eGFR levels between controls and all the groups of T2DM patients

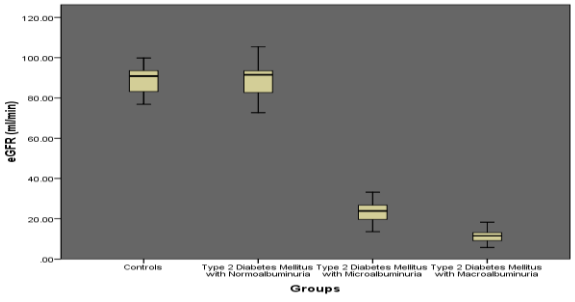

Figure 2: The albumin creatinine ratio between controls and all the groups of T2DM patients.

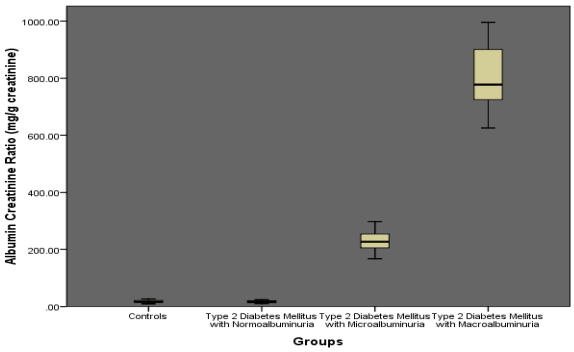

Figure 3: The serum magnesium levels between controls and all the groups of T2DM patients

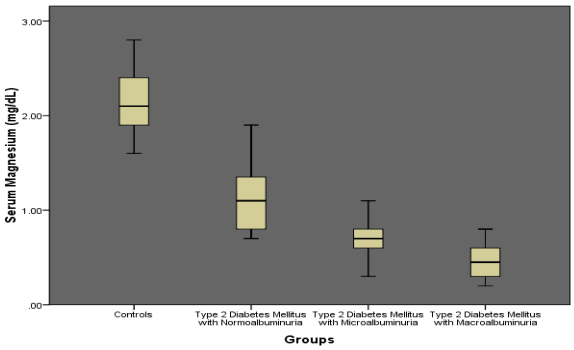

Figure 4: The urinary 8-OHdG levels between controls and all the groups of T2DM patients