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Urinary Nephrin and Serum Magnesium for Early Detection of Nephropathy in Patients with Type 2 Diabetes Mellitus

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KEYWORDS

Magnesium, Nephrin, Type 2 Diabetes Mellitus and Nephropathy

ABSTRACT:

Introduction: Nephropathy is an clinical condition in patients with T2DM. To early detection and progression of nephropathy is necessary to reduce burden for T2DM patients.

Objectives: The role of serum magnesium, and urinary nephrin, for early detection of nephropathy in patients with type 2 diabetes mellitus.

Methods: This was a cross sectional study which compared biochemical, clinical parameters in 40 type 2 diabetes mellitus with normo albuminuria, 40 type 2 diabetes mellitus with micro albuminuria, 40 type 2 diabetes mellitus with macro albuminuria and 40 age, gender and BMI matched healthy controls. Blood sugars, lipid profile, glycated haemoglobin, magnesium, eGFR, urinary ACR and urinary nephrin were analysed.

Results: There were significant differences in serum magnesium, urinary nephrin, urinary ACR, and eGFR among different stages of type 2 diabetes mellitus patients when compared to controls. Additionally, the serum magnesium negatively correlated with BMI, blood sugars, glycated haemoglobin, lipid profile, urinary ACR and positively correlated with eGFR and HDL (P=0.001**). Additionally, the urinary nephrin positively correlated with BMI, blood sugars, glycated haemoglobin, lipid profile, urinary ACR and negatively correlated with eGFR, serum magnesium and HDL (P=0.001**). Furthermore, the receiver operating characteristic curve analysis revealed the serum magnesium and urinary nephrin shown very high significant at area under the curve.

Conclusions: Hypomagnesemia precede beneficial for type 2 diabetes mellitus and urinary nephrin can serve as early predictive and prognostic indices for type 2 diabetic nephropathy.

1. Introduction

Type 2 Diabetes Mellitus (T2DM) is a chronic disease due to accumulation of blood sugars and insulin resistance. Both environmental and genetic variables contribute equally to the development of this prevalent metabolic disease (1). The majority of T2DM patients have hyperglycemia symptoms at first, which, if persistent, can result in long-term issues that eventually malfunction the heart, kidneys, nerves, eyes, and blood vessels (2). According to recent research studies the prevalence of T2DM, there was 366 million diabetics worldwide by 2014, this number estimated to reach 422 million by 2030. According to world health organization India is considered as capital of diabetes because 108 million diabetes were recorded in 2014 and this number expected to reach around 171 million in 2030 (3-6).

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Diabetic Nephropathy (DN) is the most common micro vascular complication in T2DM patients. Although DN predicted to go through distinct stages such as glomerular hyper filtration, microalbuminuria, overt proteinuria, and ultimately develop end-stage kidney disease (7-10). Micro albumin is a marker for nephropathy in patients with T2DM, but research studies are reported this cannot be a sensitive and specific marker since it has lot of flows. It is elevated in other disease conditions like obesity hyper tension, other types of kidney diseases (11). Additionally, many micro albuminuria patients revert to normo albuminuria and the normo albuminuria patients shown advance renal pathological changes. There is need biomarkers for early detection and progression of nephropathy in patients with T2DM (12).

The magnesium is a principle mineral and distributed in the human body is roughly as follows: 55% of it is free, 30% is protein bound (mostly albumin), and 15% is combined with other anions such as phosphate, citrate, etc. The physiological properties of magnesium like, More than 300 distinct enzymes that take part in the metabolism of substrates (carbohydrate, proteins, and lipids) and nucleic acids are known to be activated by magnesium. It is also needed for the processes of insulin production, binding, and action (13-14). Abnormal activity of tyrosine kinase on the insulin receptors, which may be brought on by hypomagnesaemia, can result in post-receptor resistance of insulin as well as impaired or reduced cellular utilization of glucose. Progressive insulin secretory malfunction and insulin resistance are present in 90% of all type 2 DM patients. Therefore, it is clear that magnesium and Type 2 Diabetes mellitus are closely related, and that hypomagnesemia, which is mostly brought on by increased renal excretion, affects roughly one-third of those with Type 2 DM (15-16). Several studies have shown that patients with diabetes who have micro albuminuria are more likely to experience complications that gradually affect the kidney, eyes, heart, and nerves. These complications are also linked to oxidative stress and changed serum magnesium levels (17).

Nephrin is a podocyte-specific protein and it will involve in renal filtration barrier, glomerular basement membrane (GBM), and endothelial cell layer. Any significant pathological changes happen in the podocytes these proteins leak into urine. Additionally, diabetic nephropathy causes podocytes to change phenotypically, results in renal filtration barrier disruption, which causes nephrin to be lost in the urine (18). Some of the recent studies found drastically elevated levels of urinary nephrin in T2DM with micro albuminuria, normo albuminuria when compared to controls. The significantly increased levels of nephrin in urine can act as a marker for nephropathy (19-20). The studies are very limited and also focused on irrespective of micro albumin groups. The present study aimed to determine the urinary nephrin and serum magnesium for early detection of nephropathy in patients with type 2 diabetes mellitus.

2. Objectives

The role of serum magnesium, and urinary nephrin, for early detection of nephropathy in patients with type 2 diabetes mellitus.

3. Methods

This cross-sectional study conducted in department of biochemistry collaborated with nephrology at Raichur Institute of Medical Sciences, Raichur, Karnataka. A total one hundred twenty (120) patients diagnosed with T2DM according to American Diabetic Association Criteria (21) and forty (40) age gender and Body Mass Index (BMI) healthy controls. All the study individuals recruited after approval from Institutional Ethics Committee (IEC) and taken informed consent form. The study groups shown in Table 1.

Study Criteria

The age around 30-70 years and T2DM patients with different stages of nephropathy diagnosed according to kidney disease improving global outcomes (KDIGO) criteria (22) were included in the present study. Subjects has history of type 1 diabetes mellitus, non-diabetic renal disease, thyroid and liver disease, macrovascular complications such as cardiovascular, cerebrovascular and peripheral vascular diseases, active inflammatory disease, urinary tract infections, and were excluded from this study.

Specimen Collection

Five (5) ml of venous blood sample was collected from all the participants in the study after 12 hours of overnight fasting. Among 5 ml blood sample, 1 ml transferred into fluoride tube, 1ml into Ethelin Diamino Tetra Acetic acid tube and remaining 3 ml into a plain

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tube. The blood samples were centrifuged at 3000 rpm for 10 minutes, plasma and serum was separated. Along with the blood sample, a spot urine sample was obtained and centrifuged at 3000 rpm for 10 minutes and immediately urinary albumin and creatinine were analysed. Later separated 1 ml of urine into properly labelled aliquots. The separated materials were placed in adequately labelled aliquots and kept at -800 C biochemical analysis was done.

The blood sugars, total cholesterol, triglycerides, high density lipoprotein, urinary albumin, urinary creatinine, serum magnesium, and HbA1c was estimated by using laboratory standard methods. The urinary nephrin was determined by enzyme linked immunosorbent assay. The low density lipoprotein and very low density lipoprotein was calculated by friedewald's formula. the estimated glomerular filtration rate was calculated by modified diet in renal diseases.

Statistical analysis

The data was expressed as mean ± standard deviation. The comparison between the groups done by using analysis of variance (ANOVA). The correlation between the serum magnesium, urinary nephrin and other parameters of the study was done by using pearson correlation analysis. The receivers operating characteristics (ROC) curve analysis done for early detection and progression marker for nephropathy in T2DM patients with normo albuminuria and controls. The complete analysis was done by using Microsoft Excel Spreadsheets and SPSS version 20. A P value is less than 0.05 consider as significant.

4. Results

The table 2 shows the Age, BMI, Blood sugars and the lipid profile like total cholesterol, TGL, VLDL, LDL HbA1c, significantly elevated in patients with T2DM When compared to controls (P<0.05). The urinary albumin creatinine ratio significantly increased in patients with T2DM when compared to controls (P=0.001**). The eGFR and magnesium significantly decreased in T2DM Patients when compared to controls (P=0.001**). The urinary nephrin significantly very high in T2DM patients when compared to controls (P=0.001**).

The table 3 shows the Age, BMI, Blood sugars and the lipid profile like total cholesterol, TGL, VLDL, LDL, HbA1c, significantly elevated in all the groups of patients with T2DM When compared to controls (P<0.05). The urinary albumin creatinine ratio drastically increased in T2DM patients with micro, macro albuminuria when compared to T2DM patients with normo albuminuria and controls (P=0.001**). The eGFR and magnesium significantly decreased in T2DM Patients with micro, and macro albuminuria when compared to T2DM patients with normo albuminuria and controls (P=0.001**). The urinary nephrin significantly very high in T2DM patients with normo, micro, macro albuminuria when compared to controls (P=0.001**).

The table 4 shows the serum magnesium significantly and positively correlated with eGFR and HDL (P=0.001**). Along with that serum magnesium negatively correlated with FBS, total Cholesterol, TGL, VLDL, LDL, HbA1c, Albumin creatinine ratio and urinary nephrin (P=0.001**). The urinary nephrin significantly and negatively correlated with eGFR, serum magnesium and HDL (P=0.001**). Along with that urinary nephrin positively correlated with FBS, total cholesterol, TGL, VLDL, LDL, HbA1c, Albumin creatinine ratio (P=0.001**).

The table 5 shows the ROC Curve analysis revelled the albumin creatinine ratio and eGFR not shown significant at area under curve with sensitivity (67, 85) and specificity (55, 37) respectively P value is greater than 0.05. The serum magnesium shown very high significant at area under curve with sensitivity (100) and specificity (97) respectively P value is less than 0.05. additionally, the urinary nephrin shown very high significant at area under curve with sensitivity (92) and specificity (87) respectively P value is less than 0.05.

The figure 1 illustrates the urinary albumin creatinine ratio drastically increased in T2DM patients with micro and macro albuminuria when compared to T2DM patients with normo albuminuria and controls.

The figure 2 illustrates the serum magnesium drastically decreased in T2DM patients with normo, micro and macro albuminuria when compared to controls.

The figure 3 illustrates the urinary nephrin drastically increased in T2DM patients with normo, micro and macro albuminuria when compared to controls.

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5. Discussion

Diabetic nephropathy is a common micro vascular consequence in people with diabetes mellitus. 30–40% of patients develop diabetic nephropathy. It develops over ten to twenty-five years into end-stage renal disease (ESRD). Because diabetic nephropathy results in hyper filtration glomerular damage, it produces pathologic changes in the kidneys' structural and functional components (23). The most common test used for diagnosing diabetic nephropathy at this time is microalbuminuria. It does not, however, correspond with the advancement of renal failure and can be brought on by a number of other illnesses, including congestive heart failure, exercise, and urinary tract infections (24). Therefore, more recent biomarkers have been suggested to correlate both quantitatively and qualitatively with the development of diabetic nephropathy.

Diabetes is a major contributor to end-stage renal disease (ESRD). For the diagnosis of diabetic nephropathy, urinary albumin measurement is the gold standard. When type 2 diabetics are diagnosed, their urine albumin levels should be measured in order to screen for nephropathy. It is widely established that hypomagnesemia is linked to Type 2 diabetes and has been frequently linked to a number of diabetic complications. Hypomagnesemia may be linked to a quicker deterioration in kidney function in DM2 patients, according to more recent reports (25-26). However, it is unknown how serum magnesium levels and kidney function relate to one another in DM2.

Additionally, it plays a part in the movement of electrolytes across cell membranes. The sodium and potassium electrolyte concentration gradient is maintained by the Na+/K+-ATPase, which also helps with glucose transport. A prior study included T2DM individuals with micro albuminuria or clinical proteinuria found a substantial decrease in ionized magnesium levels (27). Another study also showed a clear correlation between low magnesium levels and an increased prevalence of MA. Hypomagnesemia has been linked to a significant progression of rapid deterioration of renal function in patients with type 2 diabetes, and low magnesium levels independently predict the progression to end-stage renal disease in patients with advanced type 2 diabetic nephropathy (28).

Nephrin is an essential component of podocytes and forms the glomerular filtration barrier with endothelial cells and the basement membrane. Nephrin is seen in urine when podocytes are injured. Since nephrin is a part of the slit diaphragm that is situated between the podocytes' foot processes, any difference in podocyte size may result in a restriction in the size-selectivity of the slit diaphragm, which would allow certain components to leak. Along with podocytopenia and podocyturia, DN is characterized by decreased nephrin expression (29). In some aberrant circumstances, podocytes may also leak nephrin. It is still unknown if they are produced during the apoptotic process when the podocyte separates and leaks into urine, or if they are shed as by-products of an active vesicular transport process from podocyte foot (30).

Previous research studies, all patients with micro and macro albuminuria and 56% of those with normoalbuminuria had nephrinuria. In another study, patients with type 2 diabetes showed decreased nephrin expression in their renal tubules. This made podocytes more prone to separation and hindered their ability to heal from damage. Patients with micro and macro albuminuria in our study had median urine nephrin levels that were greater than those with micro and normoalbuminuria. However, compared to patients with macro albuminuria, those with microalbuminuria had higher urine nephrin levels (31-33).

Nephrinuria was found to be independently correlated with both e GFR and the logarithmic form of the albumin-creatinine ratio (ACR) in the present study. These characteristics were linked to nephrinuria in people with type 2 diabetes who also had normoalbuminuria. Another study also demonstrated a correlation between normoalbuminuria in patients with type 2 diabetes mellitus and indicators of podocyte destruction, such as urine nephrin and vascular endothelial growth factor (34). Another study indicated that urinary nephrin was a more sensitive and specific diagnostic than micro albumin for identifying early diabetic nephropathy (35). The estimation of serum magnesium is might be beneficial for the patients with type 2 diabetes mellitus and urinary nephrin has capability to serve early prediction for nephropathy in type 2 diabetes mellitus patients.

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Conclusion

Based on study findings, the serum magnesium has significant at area under curve with highest sensitivity and specificity but the measurement of this might be useful for type 2 diabetes mellitus patients. The urinary nephrin might be useful for early detection and progression of nephropathy than urinary albumin creatinine ratio and eGFR in type 2 diabetes mellitus.

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Table 1: Study groups

Group	Subjects	No of Subjects
Group 1	Healthy controls	40
Group 2	T2DM patients with normo albuminuria	40
Group 3	T2DM patients with micro albuminuria	40
Group 4	T2DM patients with macro albuminuria	40

Table 2: Baseline Characteristics of controls and T2DM cases

Parameter	Control	T2DM Cases	P – Value
	Mean ± SD	Mean ± SD	
Age(years)	44.48	± 6.50	49.23 ± 7.25	0.001**
BMI (kg/m2)	22.29	± 1.52	31.47 ± 6.19	0.001**
FBS (mg/dL)	84.15	± 8.47	159.70 ± 27.62	0.001**
eGFR (ml/min)	91.59	± 7.31	41.45 ± 36.30	0.001**
Total Cholesterol (mg/dL)	162.43	± 16.13	250.18 ± 62.58	0.001**
TGL (mg/dL)	111.93	± 21.18	190.30 ± 47.75	0.001**
HDL (mg/dL)	45.45	± 6.68	30.08 ± 5.61	0.001**
VLDL (mg/dL)	24.71	± 11.85	37.68 ± 9.54	0.001**
LDL (mg/dL)	91.69	± 8.73	179.42 ± 60.31	0.001**
HbA1c (%)	4.45	± 0.74	8.17 ± 1.72	0.001**

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Albumin Creatinine Ratio (mg/g creatinine)	17.38	±	4.98	335.18	±	322.58	0.001**
Serum Magnesium	2.19	±	0.37	0.72	±	0.32	0.001**
(mg/dL)							
Urinary Nephrin (ng/mL)	0.82	±	0.28	4.46	±	1.96	0.001**

Table 3: Compression of study variables between three of T2DM patients and controls

Parameter	Controls			T2DM with Normo albuminuria	T2DM with Micro albuminuria					T2DM with Macro albuminuria			P-Value
	Mean ± SD			Mean ± SD	Mean ± SD					Mean ± SD			
Age (years)	44.48	±	6.5	42.25	±	4.67	50.58	±	4.96	54.88	±	5.42	0.001**
BMI (kg/m2)	22.29	±	1.52	24.02	±	2.08	34.97	±	3.87	35.42	±	3.48	0.001**
FBS (mg/dL)	84.15	±	8.47	136.05	±	13.38	153.33	±	14.42	186.73	±	20.01	0.001**
eGFR (ml/min)	91.59	±	7.31	91.64	±	6.58	22.38	±	4.46	10.33	±	2.57	0.001**
Total Cholesterol (mg/dL)	162.43	±	16.13	173.13	±	14.18	273.25	±	21.35	304.15	±	40.86	0.001**
TGL (mg/dL)	111.93	±	21.18	135.68	±	18.66	199	±	20.3	236.23	±	29.91	0.001**
HDL (mg/dL)	45.45	±	6.68	35.18	±	4.37	29.44	±	3.36	25.63	±	4.28	0.001**
VLDL (mg/dL)	24.71	±	11.85	27.21	±	3.38	38.95	±	4.5	46.9	±	6.69	0.001**
LDL (mg/dL)	91.69	±	8.73	105.21	±	12.95	201.6	±	23.53	231.44	±	38.06	0.001**
HbA1c (%)	4.45	±	0.74	7.02	±	0.84	7.59	±	1.23	9.91	±	1.4	0.001**
Albumin Creatinine Ratio (mg/g creatinine)	17.38	±	4.98	18.82	±	4.04	231.58	±	37.49	755.12	±	146.83	0.001**
Serum Magnesium (mg/dL)	2.19	±	0.37	0.84	±	0.3	0.85	±	0.33	0.47	±	0.14	0.001**
Urinary Nephrin (ng/mL)	0.82	±	0.28	3.1	±	1.39	3.97	±	1.36	6.31	±	1.49	0.001**

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Tables 4: correlation of serum magnesium, urinary nephrin, and other study variables

Parameter	Serum Magnesium	Urinary nephrin	P
	r	r	
BMI (kg/m2)	-0.66	0.32	0.001**
FBS (mg/dL)	-0.77	0.91	0.001**
eGFR (ml/min)	0.75	-0.45	0.001**
Total Cholesterol (mg/dL)	-0.67	0.69	0.001**
TGL (mg/dL)	-0.76	0.54	0.001**
HDL (mg/dL)	0.69	-0.73	0.001**
VLDL (mg/dL)	-0.64	0.86	0.001**
LDL (mg/dL)	-0.67	0.79	0.001**
HbA1c (%)	-0.74	0.26	0.001**
Albumin Creatinine Ratio (mg/g creatinine)	-0.76	0.49	0.001**
Serum Magnesium (mg/dL)	1	-0.72	0.001**
Urinary Nephrin (ng/mL)	-0.72	1	0.001**

Table 5: ROC Curve analysis for early detection of nephropathy in T2DM patients with normo albuminuria and controls

Parameter	AUC	95% CI FOR VALUE	CUT FOR VALUE	SENSISTIVITY	SPECIFICITY	P-VALUE
Albumin Creatinine Ratio (mg/g creatinine)	0.585	0.469 to 0.694	0.2250	67.50	55.00	0.1924
eGFR (ml/min)	0.515	0.400 to 0.628	0.2250	85.00	37.50	0.8262
Serum Magnesium	0.999	0.952 to	0.9750	100.00	97.50	<0.0001

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(mg/dL)		1.000				
Urinary Nephrin (ng/mL)	0.957	0.886 to 0.989	0.80000	92.50	87.50	<0.0001

Figure 1: comparison of albumin creatinine ratio between controls and T2DM patients with normo, micro, macro albuminuria

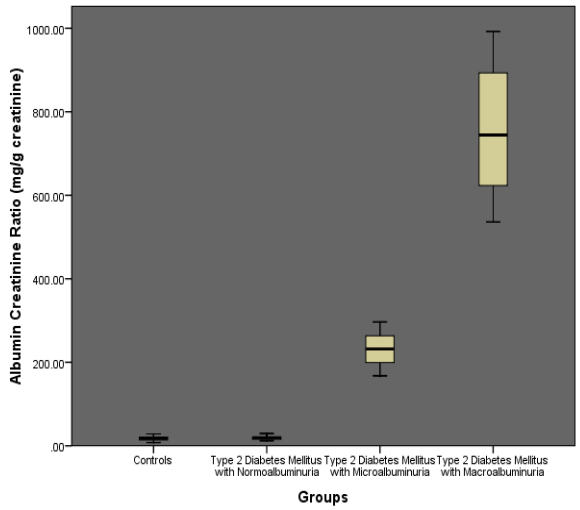

Figure 2: comparison of albumin creatinine ratio between controls and T2DM patients with normo, micro, macro albuminuria

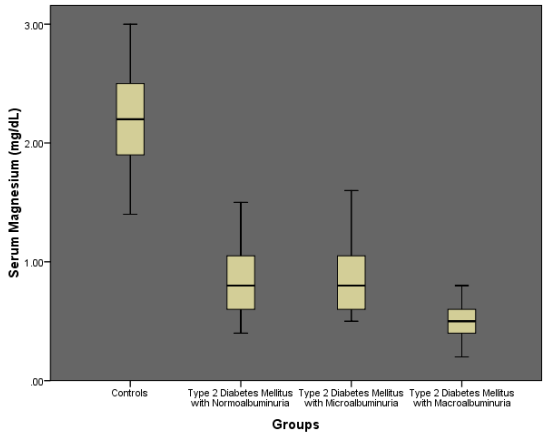

Figure 3 : comparison of urinary nephrin between controls and T2DM patients with normo, micro, macro albuminuria