Correlation of Serum Magnesium Level with Microalbuminuria in Type 2 Diabetes Mellitus

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ABSTRACT

BACKGROUND

Magnesium is an essential cofactor for many enzymes involved in various metabolic processes in the body. Magnesium is present in the human body primarily as free magnesium or protein bound (primarily albumin), in which the free or ionized magnesium is the physiologically active form. Magnesium deficit has been frequently identified in type 2 Diabetes Mellitus patients. The mechanism which eventually leads to the deficiency of magnesium in Type 2 Diabetes Mellitus patients may be because of low dietary intake of magnesium and / or also due to an increased renal excretion of magnesium in urine as a result of osmotic diuresis in such patients. Its deficiency has been implicated in the progression and development of complications of DM. Role of magnesium in the development of microalbuminuria has not been established fully with various authors having differing opinions on its role. In our study, we estimated the serum magnesium levels in type 2 DM and studied its correlation with microalbuminuria.

METHODS

50 patients of Type 2 DM and 50 controls attending Medicine OPD were included in the study. The case and control groups were age and sex matched. History of smoking, alcohol use, chronic diseases history, and use of medications which modify the metabolism of magnesium was obtained by a standard interview questionnaire. Blood samples were collected for serum magnesium estimation in sterile empty vials. Random sample of urine was collected for microalbumin estimation. The samples were processed and analysed by automated chemistry. Data was then analysed using the Statistical Package for Social Sciences (SPSS).

RESULTS

S. Mg in male cases was 1.61 ± 0.16 mg/dL and female cases was 1.67 ± 0.11 mg/dL compared to 1.9 ± 0.16 mg/dL in male controls and 1.93 ± 0.16 mg/dL in female controls. Urine MA in cases was 22.04 ± 7.72 mg/L and 9.34 ± 2.57 mg/L in controls. Urine microalbumin was very significantly increased in cases than in controls ($p < 0.001$). S. Mg was decreased in cases with microalbuminuria (1.63 ± 0.14 mg/dL) but was not significant statistically ($p > 0.05$). Serum magnesium and microalbumin were found to be negatively correlated among cases but was not significant ($p>0.05$).

CONCLUSIONS

Serum Magnesium was found to be negatively correlated with Urine Microalbumin but no statistical significance was observed.

KEY WORDS

S. Mg. (Serum Magnesium), MA (Microalbumin), DM (Diabetes Mellitus)

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BACKGROUND

Diabetes mellitus can be seen in almost every population in the world. Without an effective prevention and control strategy, it is likely to influence the population globally.[1] Genetics and environmental factors are equally responsible for causation of this common metabolic disorder. Mostly patients present with symptoms of hyperglycaemia, which if chronic, leads to the development of long-term complications eventually causing dysfunction with involvement of organs the eyes, kidneys, nerves, heart, and blood vessels.[2]

Magnesium distribution in the human body is approximately as follows-- free magnesium-55%, protein bound (primarily albumin)-30% and about 15% is complexed with other anions like phosphate, citrate etc.[3] Magnesium is a known activator of more than 300 different enzymes, involved in the metabolism of fuels-(carbohydrate, proteins, lipids) and nucleic acids. It also has a role in electrolyte transport across cell membranes.[4] The Na+/K+-ATPase maintains the electrolyte concentration gradient (sodium and potassium) and also assists in transport of glucose. Its activity can be changed or modified in a hypomagnesaemic state.[5] The process of insulin secretion, binding, and activity also requires the involvement of magnesium. Post receptor resistance of Insulin as well as defective or decreased cellular utilisation of glucose can occur as a result of defective tyrosine kinase activity on the insulin receptors which may be caused due to hypomagnesaemia.[6] 90% of all type 2 DM cases show progressive insulin secretory defect along with insulin resistance.[7] The Magnesium deficit when chronic may result, in insulin resistance as well as a reduced utilization of glucose, leading to further aggravation of the already decreased insulin sensitivity seen in patients of type 2 DM.[8]

The total serum magnesium can still be in the normal range even when the ionized magnesium or intra-cellular magnesium is depleted. But when hypomagnesemia is established, it usually indicates systemic Mg deficit in the body. Usually the total serum Mg can be normal even though the ionised magnesium may be deficient.[9]

Type 2 Diabetes Mellitus frequently exists in association with alteration of Mg status. Hypomagnesemia appears to negatively influence insulin sensitivity as well as glucose homeostasis in patients with Type 2 diabetes.[10] It is therefore evident that a close association exists between Mg and Type 2 Diabetes mellitus and approximately about a third of those with Type 2 DM have hypomagnesemia mainly caused by enhanced renal excretion.[11]

Microalbuminuria was first reported in diabetic patients by Viberti et al. in 1982.[12] Numerous studies have demonstrated that presence of microalbuminuria in diabetic patients increases their risk of developing complications, gradually involving the cardiovascular system, eyes, kidney and nerves, which are also known to be associated with oxidative stress and altered levels of magnesium in serum[13].

A significant lowering of ionized Mg levels was noticed in a previous study involving type 2 diabetic patients with microalbuminuria or clinical proteinuria,[14] and another study also demonstrated that low Mg level strongly associated with increased prevalence of MA.[15] Low levels of magnesium independently predict the progression to End stage renal disease in patients with advanced type 2 diabetic nephropathy[16] and also an association between hypomagnesemia and a significant progression of rapid deterioration of renal function in patients with type 2 diabetes was reported.[17]

However, the correlation between microalbuminuria and hypomagnesemia has not been proved conclusively. One study reported that infusion of high doses of Mg in seriously ill patients with trauma, reduced the excretion of microalbumin in urine.[18] Another study showed no correlation with hypomagnesemia in cases and normal subjects with respect to status of microalbumin.[19]

Hence, this study was done to determine the status of Mg levels in Type 2 DM subjects and also to ascertain if there was any correlation of magnesium levels in serum with microalbuminuria.

METHODS

A total of 50 cases and 50 controls from Medicine OPD were included in the study. The study was done after obtaining approval from the Institutional Ethics Committee. The individuals of the control group were selected in such a way that the total number of controls and cases in each age and sex group were the same. History was taken for smoking, alcohol use, chronic diseases history, and medications for antidiabetics & anti-hypertensives were obtained by a standard interview questionnaire.

Inclusion Criteria

The cases included in the study were known type 2 DM patients attending the outpatient clinic in the department of Medicine.

Exclusion Criteria

Renal failure patients, Type I DM patients, chronic alcohol consumption, acute pancreatitis, Patients on loop/thiazide diuretics or Magnesium supplement/Magnesium containing antacids, were excluded from the study.

Under aseptic conditions, 5 mL blood was collected from ante-cubital vein for Serum Magnesium and random sample of urine was collected in sterile container for urine microalbumin. Magnesium was assayed by calmagite method, its normal range was taken as 1.7–2.4 mg/dL. The normal reference range as provided in the kit has been used for the purpose of estimation. Urine MA analysis was done by TURBILYTE-MA™ turbidimetric immunoassay for the detection of albumin in urine. Normal urinary albumin concentration (UAC) was defined as < 20 mg/L. Microalbuminuria was defined in the range of 20-200 mg/L as defined by various standard literatures. The Statistical

Package for Social Sciences (SPSS) was used for data analysis, and data were presented as mean ± standard deviation. p values were regarded significant when ≤0.05.

RESULTS

Out of a total number of 50 cases, 36 were male and 14 were female and the maximum number of cases were seen in the 46-50 age group. 50 controls age and sex matched with cases were taken.

	S. Mg in Controls	S. Mg in Cases
Males	1.9 ± 0.16 mg/dL	1.61 ± 0.16 mg/dL
Females	1.93 ± 0.16 mg/dL	1.67 ± 0.11 mg/dL

Table 1. Serum Magnesium in Controls and Cases

Table 1 shows that S. Mg in male controls was 1.9 ± 0.16 mg/dL and in female controls 1.93 ± 0.16 mg/dL. S. Mg in male cases was 1.61 ± 0.16 mg/dL and female cases was 1.67 ± 0.11 mg/dL. Serum magnesium was found to be low in all age groups of both male & female cases, from this we can infer that serum magnesium is lower in cases than in controls.

Urine Microalbumin Level (mg/L)	Number of Cases	%
Normoalbuminuria	32	64%
Microalbuminuria	18	36%

Table 2. Distribution of Cases with Normoalbuminuria & Microalbuminuria in Study Group

Table 2 shows distribution of cases with MA within normal limits and microalbuminuria in cases. In the above table we can see that 18 cases (36%) had microalbuminuria and 32 cases (64%) had normoalbuminuria. The mean MA level was 22.04 ± 7.72 mg/L in cases.

Age Group (in Years)	Mean Serum Magnesium in Cases with Normoalbuminuria mg/dL)	Mean Serum Magnesium in Cases with Microalbuminuria (mg/dL)
40–45	1.68 ± 0.16	0.00
46–50	1.61 ± 0.17	1.70 ± 0.14
51–55	1.62 ± 0.16	1.64 ± 0.19
56–60	1.50 ± 0.00	1.62 ± 0.11
> 60	1.40 ± 0.00	1.65 ± 0.10
Mean	1.65 ± 0.17	1.63 ± 0.14
"p"value	p> 0.05	

Table 3. Distribution of Mean S. Mg in Cases with Normoalbuminuria and Microalbuminuria

U. Microalbumin	Number of Observed Cases	Mean ± S.D. (mg/L)	P Value
Control	50	9.34 ± 2.57	P < 0.001
Cases	50	22.04 ± 7.72	

Table 4. Comparison of U. Microalbumin between Cases and Controls

From Table 3 we can infer that the concentration of serum magnesium is low both in cases with normoalbuminuria (1.65 ± 0.17 mg/dL) and patients with microalbuminuria (1.63 ± 0.14 mg/dL). It was observed that patients with microalbuminuria had lower serum magnesium level than patients who had normoalbuminuria but was statistically non-significant (p>0.05.).Table 4 shows that the mean Urine. MA in cases is 22.04 ± 7.72 mg/L and in controls is 9.34 ± 2.57 mg/L. Urine MA was found to be significantly increased in cases than in controls (p< 0.001).

Compared Variable	Number of Observed Cases	Mean ± S.D.	"r" Value	P Value
S. Magnesium	50	1.64 ± 0.15	r = - 0.32	P > 0.05
U. Microalbumin	50	22.04 ± 7.72		

Table 5. Correlation between S. Magnesium (mg/dL) and Urine. Microalbumin (mg/L)

Table 5 shows the correlation between Serum magnesium and MA and it was found to be negatively correlated among cases which means that with the decrease of magnesium, urine MA increases. Even though an inverse correlation was established in our study, yet the correlation was found to be statistically not significant (p>0.05)

DISCUSSION

Long term complications of diabetes mellitus may be a consequence of metabolic derangement's and electrolyte changes which has been reported in various studies. Deficiency of magnesium plays an important role in the progression of diabetic complications as reported in previous studies. Therefore evaluating the serum magnesium status may assist in the prevention and management of different complications observed in Type 2 DM.

Microalbuminuria is seen in diabetics and various methods are available for its estimation like Urine albumin concentration (UAC), Albumin Creatinine ratio (ACR) and Twenty-four-hour urinary albumin excretion (UAE).

Twenty-four-hour urinary albumin excretion is the "gold standard" for quantitative evaluation of albuminuria in diabetic patients; however, due to difficult patient compliance, very often over- and underestimation of albuminuria may be seen. The American Diabetes Association[20] have recommended following guidelines for measurement of albumin-to-creatinine ratio (ACR) in a random spot urine collection for diagnosis of microalbuminuria. Microalbuminuria is diagnosed if ACR ranges between 30 and 300 mg/g creatinine. The gender-specific ACR cut-offs of 17–249 mg/g for men and 25–354 mg/g for women have been suggested by Warram et al[21] and albumin concentration cut-offs of 16–159 mg/Litre by Bakker.[22] Cut-off values for urinary albumin in various studies has been advocated as 20-200 mg/L.[23,24]

The guidelines recommended using a first-morning sample because of the potentially higher correlation with 24-h albumin excretion, but a random sample is considered acceptable if a first-morning specimen is not available.

Bakker AJ[22] in his study recommended ACR to be more accurate than the urinary albumin in screening for microalbuminuria. But unlike UAC the ACR requires sex- and age-specific discriminator values to be more precise.

In a study by Alan R. Dyer et al[25] it was suggested that both albumin concentration (UAC) and ACR appear to be a rational alternative to 24-hour albumin excretion on the basis of their relative positives and negatives. They suggested that UAC is a viable alternative to 24-hour albumin excretion for epidemiologic studies in which body size and weight measurements are not readily available or the additional expense incurred for measurement of creatinine is taken into account.

In the present study evaluation of S. Magnesium status in type 2 DM patients showed a significant decrease in its level in cases as compared to controls. This is consistent with studies of Razeena KC et al[26] and Vijaylakshmi S et al.[27]

In 50 Type 2 DM patients, 32 cases (64%) had urine microalbumin within normal range and 18 cases (36%) had microalbuminuria with a mean U. microalbumin found to be 22.04 ± 7.72 mg/L. All the controls had mean U. microalbumin within normal range and it was 9.34 ± 2.57 mg/L. This is consistent with studies of Nasreen et al[28] who also found that 35% of their cases had microalbuminuria. These results were again corroborated by Varghese et al.[29] who found microalbuminuria in 36.3% of their cases and also found it to be increased with duration of Type 2 Diabetes Mellitus.

Also Xu B et al [15] has reported a prevalence of microalbuminuria in somewhat lower range of 11.37%. Further, a study conducted by Corsonello et al[14] demonstrated that diabetic patients with microalbuminuria or overt Proteinuria showed a significant decrease in serum Mg compared with normoalbuminuric group.

However some studies such as Zargar et al[30] and Sales et al[19] were in discordance with our results and they have reported that no association is present between S.Mg & Microalbuminuria in diabetic patients (Type 1 & 2)

According to F. Guerrero-Romero et al[31] the reasons for the variation in different studies may be due to different geographical locations along with change in dietary habits leading to differences in magnesium intake in the study population

Since magnesium is a mild, natural calcium antagonist, intracellular accumulation of calcium occurs in deficient subjects. This increased intracellular calcium may alter the cells responsiveness to insulin, leading to development of insulin resistance.[8] Therefore, an amplification of insulin resistance may be a possible mechanism that connects Serum magnesium to microalbuminuria. In addition, in another study by Barbagallo M et al,[8] it was found that hypomagnesemia can also be caused due to impaired tubular absorption of magnesium in diabetic patients with insulin deficiency or resistance. Therefore, a hypomagnesaemic and insulin resistant state can increase the risk of Microalbuminuria.[32] Anastasia A. Zheltova et al[33] found a strong link between oxidative stress and hypomagnesemia but no conclusive evidence about the mechanism causing oxidative stress in magnesium deficiency has been ascertained. Also, Mirrahimi B et al[18] found in their study supplementation of magnesium in trauma patients reduced microalbuminuria and Ning Shao et al[34] have found a significant correlation between oxidative stress and microalbuminuria. So, based on the evidence of previous studies it can be presumed that deficiency of magnesium causing increased oxidative stress may have a role in the causation of microalbuminuria and further studies to confirm the above hypothesis is required.

Deficiency of magnesium may cause certain changes in the endothelial cells causing endothelial cell injury due to inflammation and oxidative stress.[16] This injury to the endothelial cells, prominently in the glomerular vessel, may increase capillary permeability leading to the occurrence of transient proteinuria. These changes can be assessed by increased urine microalbumin.

Various studies exist which show associations between hypomagnesemia and complications of Type 2 DM such as neuropathy, retinopathy, foot ulcers, and albuminuria.[35]

In our study, we found an inverse correlation (r=–0.32) between serum Mg levels and urine microalbumin which was consistent with many studies by other authors, but our result ($P > 0.05$) were statistically insignificant. A somewhat similar findings were observed in a study conducted by Rao et al[36] who reported only 6% of cases in their study with hypomagnesemia and the urine microalbumin (SUACR) when compared in microalbuminuria with hypomagnesemia group and microalbuminuria with normomagnesemia group, they didn't find any significance of levels of urine microalbumin in the groups. They surmised that these results may be due to small number of cases and reduced detection of hypomagnesemia in their cases.

Rooney et al[37] in their study measured Total serum magnesium and ionised magnesium in healthy subjects before and after supplementation of oral magnesium and for 10 weeks resulted in increased concentrations of ionised Mg but was not statistically significant.

Martha Rodríguez-Morán et al[38] in a randomized double-blind placebo-controlled trial study on type 2 Diabetes mellitus patients with hypomagnesemia concluded that on receiving oral magnesium supplementation had improved insulin sensitivity and better metabolic control.

Dae Jung Kim et al[39] studied the long-term associations between magnesium intake and its relation to systemic inflammation, insulin resistance and progression to diabetes among young American adults. They observed that increased dietary magnesium lead to decrease in serum inflammatory markers such as high-sensitivity C-reactive protein (hs-CRP), interleukin-6 (IL-6) and the homeostasis model assessment of insulin resistance (HOMA-IR). They also found that magnesium intake was inversely associated with incidence of diabetes.

Our major limitations were small sample size and since the design of study was cross sectional in nature the exact causation between magnesium and microalbumin is not known and also progression of microalbuminuria with changing magnesium status with reduced dietary intake & increased renal excretion cannot be ascertained properly.

Also, the analytical method used to estimate S. Mg was photometric which measures the total magnesium content in serum. Use of better methods which measures the free ionised magnesium using Ion Selective Electrodes (ISE), may be used which corresponds with the actual magnesium status in the body. Altura et al[40] suggested that measuring

total serum or plasma magnesium is not a true indicator of Mg status and magnesium has several forms and the fraction which is physiologically most important and active is the ionized free magnesium and therefore, specific and precise laboratory methods like ion sensitive electrodes are needed for more actual evaluation of magnesium. Therefore, future large scale studies with advanced analytical methods and clinical trials on the effect of magnesium supplementation to prevent diabetic complications can be conducted to come up with an unerring insight into the relationship between hypomagnesemia and microalbuminuria.

CONCLUSIONS

Serum Magnesium was found to be significantly lowered in type 2 diabetes mellitus patients, and it was inversely correlated with urine microalbumin. However, the relation between serum magnesium and urine microalbumin was not statistically significant.

Further large scale studies and clinical trials on the effect of magnesium supplementation on diabetic complications are needed to evaluate the relationship between hypomagnesemia and microalbuminuria.

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