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## Original Article

# Hypomagnesaemia among Diabetic Patients with Chronic Kidney Disease: A Cross-sectional Study

Najeeb Mahiyuddin[1], Nazia Nusrat Khan[2], Miftahul Jannat[3], Md Saif Bin Mizan[4], Muhammad Abdur Rahim [5], Khwaja Nazim Uddin[6]

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**Abstract:**

***Background:*** *The clinical role of magnesium, especially of hypomagnesaemia, in diabetic and chronic kidney disease patients has been underestimated for many years.*

***Objective:*** *To determine the frequency of hypomagnesaemia in diabetic patients with chronic kidney disease and to investigate the relationship of magnesium level with glycemic and renal status.*

***Methodology:*** *This was a cross-sectional observational study performed on 100 diabetic patients with chronic kidney disease, admitted to BIRDEM General Hospital, Dhaka, Bangladesh, excluding patients with history of chronic diarrhea, laxative abuse, diuretic use, alcoholism and those on dialysis. Several socio-demographic and biochemical parameters were analyzed and compared with magnesium level.*

***Results:*** *Frequency of hypomagnesaemia among the study population was 37% (p= 0.009) and the frequency was found to be increasing with increasing stage of chronic kidney disease. Frequency of hypomagnesaemia was more in patients with uncontrolled fasting blood glucose, post meal blood glucose and HbA1c level (p<0.05). Serum magnesium levels positively correlated with serum creatinine levels (Pearson's correlation coefficient, r=0.428, p=0.001) and inversely correlated with Glomerular Filtration Rate (GFR) (Pearson's correlation coefficient, r= -0.275, p= 0.006).*

***Conclusion:*** *The frequency of hypomagnesaemia among diabetic patients with chronic kidney disease is common. So it would be prudent to routinely monitor for hypomagnesaemia in diabetic patients with chronic kidney.*

***Key words:*** *Diabetic patients, Chronic kidney disease, Hypomagnesaemia.*

## Introduction:

Magnesium is an essential mineral for bone mineralization, muscular relaxation, neurotransmission and other cell functions.[1] It is absorbed in intestine, stored as bone mineral and excess magnesium is excreted by the urine and faeces. The kidneys play a vital role in the maintenance of magnesium homeostasis.[2] Under physiological conditions, around 95% of filtered magnesium is reabsorbed by the kidneys and only 3–5% is excreted in the urine.[3] However, the kidneys can control the magnesium excretion and re-absorption such that renal excretion of the filtered load of magnesium vary from 0.5 to 70%.[2]

In healthy individuals, serum magnesium concentration is closely maintained within the physiological range.[4] This reference range is 0.7–1.10 mmol/L for total magnesium concentrations in adult blood serum and 0.54–0.67 mmol/L for ionized magnesium.[5]

The ability of the kidneys to maintain the magnesium excretion declines significantly when the renal function deteriorates.[6] Interestingly, in non-diabetic patients with chronic kidney disease the magnesium level significantly increases when creatinine clearance declines but the serum magnesium levels were significantly lower in diabetic patients with chronic kidney disease.[7] The low magnesium in patients with chronic kidney disease may be attributed to use of proton pump inhibitors, malnutrition, co-morbidities such as alcoholism and use of low magnesium containing dialysis fluid, which results in diffusional movements of magnesium from circulation.[8]

Hypomagnesaemia has been strongly associated with type 2 diabetes mellitus (T2DM) and increased risk for diabetes complications.[9] Magnesium is a direct factor in the development of insulin resistance and insulin is an important

1. Registrar, Department of Medicine, Popular Medical College Hospital, Dhaka, Bangladesh.
2. Registrar & Specialist, Cardiac ICU, Ibrahim Cardiac Hospital and Research Institute, Dhaka, Bangladesh.
3. Registrar, Department of Medicine, Ahsania Mission Cancer and General Hospital, Dhaka, Bangladesh.
4. Associate Professor & Head, Department of Nephrology, DrSirajul Islam Medical College and Hospital Limited, Dhaka, Bangladesh.
5. Associate Professor, Department of Nephrology, BIRDEM General Hospital, Dhaka, Bangladesh.
6. Professor, Department of Internal Medicine, BIRDEM General Hospital, Dhaka, Bangladesh

***Corresponding Author**:

Dr. Najeeb Mahiyuddin
Registrar, Department of Medicine
Popular Medical College Hospital, Dhaka, Bangladesh.
Email: najeebmahiyuddin20@gmail.com



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regulator of magnesium homeostasis.[10,11] This leads to a vicious circle, where low magnesium level causes insulin resistance, which in-turn causes hypomagnesaemia.[12] A retrospective study with 550 T2DM patients without known kidney disease revealed, that lower magnesium levels were associated with further deterioration of renal function.[13] Patients with higher serum magnesium levels (between 0.82–1.03 mmol/L) had the slowest progression and the best glycaemic control. Hypomagnesaemia has been observed to increase cardiovascular risk by accelerating atherosclerosis[14-16] and increasing left ventricular mass.[17] Low magnesium level has also been linked to high blood pressure[18] and osteoporosis.[19] Hypomagnesaemia is a predictor of mortality and kidney function decline in CKD patients[20] as well as predictor of mortality in haemodialysis patients.[21] Furthermore, magnesium is a risk factor for non-recovery of renal function in critically ill patients with acute kidney injury.[22]

Considering the deleterious effect of hypomagnesaemia, the magnesium concentrations are not routinely measured in patients, especially in those with diabetes mellitus (DM) and CKD. Therefore the study aims to determine the frequency of hypomagnesaemia in diabetic patients with CKD and the relationship of magnesium level with glycaemic and renal status.

## Methods

This cross sectional observational study was carried out at in-patient nephrology department of BIRDEM General Hospital of Dhaka, Bangladesh after receiving approval from the Institutional Review Board (IRB) of Diabetic Association of Bangladesh (BADAS). The study period was 6 months from 1st May 2017 to 31st October 2017. One hundred hospitalized adult diabetic patients with CKD stage 1-5, not on dialysis, excluding those with history of chronic diarrhea, laxative abuse, diuretic use, alcoholism and those on dialysis, were consecutively selected for the purpose of the study. Informed written consent was obtained from all patients. Data regarding age, gender, religion, education, occupation, monthly income, duration of DM and CKD were collected using a case record form. Capillary blood was used to check the random, fasting and 2 hour after breakfast blood sugar level. Venous blood was drawn from patients by experienced nurses using sterile gloves and sent for biochemical investigations like glycated haemoglobin (HbA1c), serum calcium, serum magnesium and serum creatinine level. Serum calcium, magnesium and creatinine level was analyzed in Beckman Coulter AU480 Clinical Chemistry System, which uses colorimetric method to measure serum calcium and magnesium level and spectrophotometry to analyze serum creatinine level. HbA1c was estimated using high performance liquid chromatography in Premier Hb9210 HbA1c Analyzer. Glomerular filtration rate (GFR) was calculated using the Chronic Kidney Disease Epidemiology Collaboration (CKD-EPI) equation.[23] CKD staging was done according to GFR following the Kidney Disease: Improving Global Outcomes (KDIGO) guidelines.[24] The study population was divided into low magnesium (serum magnesium < 0.7 mmol/L) and normal magnesium group (0.7-1.1 mmol/L) and the socio-demographic and biochemical variables were compared between the two groups. The qualitative observations were indicated by percentages. The quantitative data were expressed as mean ± standard deviation. Paired t-test was used to compare parametric values and chi-square test was used to compare qualitative values. The frequency of hypomagnesaemia was expressed according to stages of CKD and glycaemic status, which was further subdivided into controlled and uncontrolled group, using bar charts. Uncontrolled group of glycaemic status was considered when fasting blood glucose >6 mmol/l, post meal blood glucose >8 mmol/l and HbA1c >7% while fasting blood glucose ≤6 mmol/l, post meal blood glucose ≤8 mmol/l and HbA1c ≤7% were classified into controlled group of glycaemic status. Pearson correlation coefficient (r) was used to see the relationship of serum magnesium with GFR and serum creatinine. A value of p <0.05 was considered statistically significant. Data were analyzed using Statistical Package for Social Sciences (SPSS) software (version 23).

## Results

Frequency of hypomagnesaemia among diabetic patient with chronic kidney disease was 37% (p= 0.009). The socio-demographic and biochemical characteristics of the study population are shown in Table 1. None of the characteristics showed any significant association with low magnesium group. Majority of the subjects were from urban population. The mean duration of DM was 14.2 (±8.2) years and of CKD was 3.2 (±3.0) years in patients with hypomagnesaemia. There was no relationship with duration of diabetes mellitus (Pearson's correlation coefficient, r=0.06, p=0.556) and chronic kidney disease (Pearson's correlation coefficient, r=-0.05, p=0.964) with serum magnesium level. However, frequency of hypomagnesaemia was found to be increasing with increasing stage of chronic kidney disease (Figure 1). In sup group analysis of low magnesium group, it was seen that hypomagnesaemia was significantly seen in uncontrolled group of fasting blood glucose, post meal blood glucose and HbA1c compared to the controlled group (Figure 2). There was a weak negative correlation between serum magnesium and GFR (Pearson correlation test, r= -0.275, p= 0.006) and moderately positive correlation between serum magnesium and serum creatinine (Pearson correlation test, r=0.428, p=0.001) as illustrated in Figure 3 and Figure 4.

**Table I: Sociodemographic and biochemical characteristics of the study population (N=100)**

| Sociodemographic characteristics | Low magnesium Percentage (%) | Normal magnesium Percentage (%) | p-value |
|---|---|---|---|
| **Age (years)** | | | |
| ≤ 50 | 13 | 14 | 0.365* |
| 51-70 | 21 | 42 | |
| > 70 | 3 | 7 | |

| | | | |
|---|---|---|---|
| **Gender** | | | |
| Male | 16 | 36 | 0.179* |
| Female | 21 | 27 | |
| **Residing area** | | | |
| Urban | 30 | 54 | 0.542* |
| Rural | 7 | 9 | |
| **Education level** | | | |
| None | 4 | 9 | 0.629* |
| Primary | 8 | 20 | |
| Secondary | 13 | 21 | |
| Higher secondary | 5 | 3 | |
| Graduate | 4 | 6 | |
| Post-graduate | 3 | 4 | |
| **Monthly family Income (taka)** | | | |
| ≤ 30,000 | 26 | 44 | 0.280* |
| 31,000-60,000 | 9 | 10 | |
| >60,000 | 2 | 9 | |

| **Biochemical characteristics** | **Low magnesium** Mean (±SD) | **Normal magnesium** Mean (±SD) | **p-value** |
|---|---|---|---|
| Average magnesium level (mmol/L) | 0.6 (±0.64) | 0.8 (±0.1) | 0.00** |
| Fasting blood glucose (mmol/L) | 8.8 (±3.0) | 9.9 (±4.7) | 0.380** |
| Post meal blood glucose (mmol/L) | 10.7 (±3.5) | 11.3 (±5.0) | 0.528** |
| Random blood glucose (mmol/L) | 10.8 (±4.1) | 11.9 (±5.4) | 0.291** |
| HbA1c (%) | 9.1 (±3.5) | 8.7 (±3.0) | 0.534** |
| S. creatinine (mg/dl) | 3.5 (±2.1) | 4.8 (±3.1) | 0.18** |
| GFR (ml/min/m$^2$) | 22.8 (±14.4) | 17.6 (±11.7) | 0.05** |
| Calcium (mg/dl) | 8.5 (±1.0) | 8.6 (±1.1) | 0.549*** |

p value calculated by chi-square test
**p value calculated by t test
p value <0.05 is considered significant

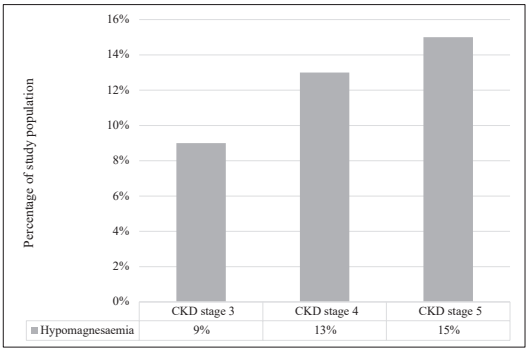


**Figure 1: Frequency of hypomagnesaemia at different stages of chronic kidney disease (100 patients)**

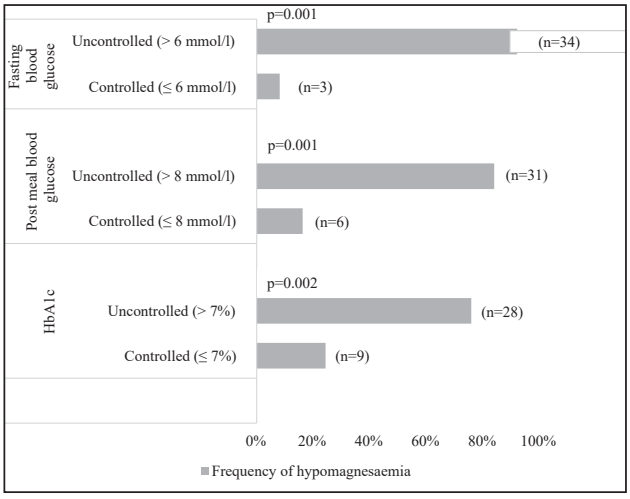


**Figure 2: Frequency of hypomagnesaemia according to glycaemic status (37 patients)**

p value calculated by chi-square test
p value <0.05 is considered significant

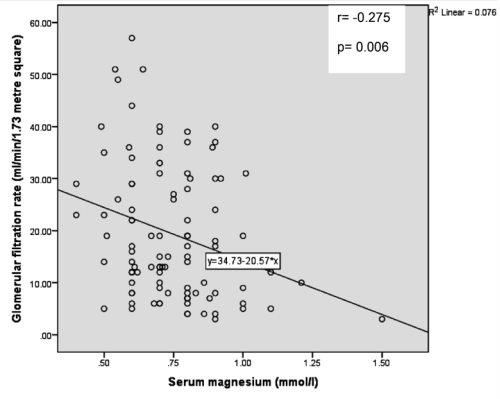


**Figure 3: Linear correlation between serum magnesium (mmol/L) and GFR (ml/min/1.73m$^2$) in the study population**

r = Pearson's correlation coefficient
p value <0.05 is considered significant

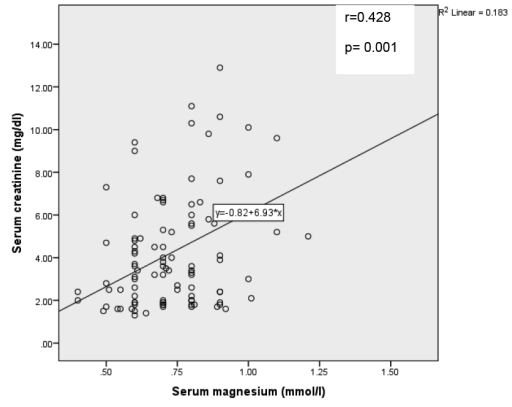


**Figure 4: Linear correlation between serum magnesium (mmol/L) and serum creatinine (mg/dl) in the study population**

r = Pearson's correlation coefficient
p value <0.05 is considered significant

## Discussion

The present study measured serum magnesium levels as well as other parameters in diabetic patients with chronic kidney disease. The aim of the study was to estimate the frequency of hypomagnesaemia in diabetic patients with chronic kidney disease and to see the relationship of serum magnesium with glycaemia and renal status.

Thirty-seven percent of the study population had hypomagnesaemia, effecting females (21 patients) more than males (16 patients). The result is comparable to that of Huang et al., who showed 22.2% frequency of hypomagnesaemia in diabetic patients with chronic kidney disease.[19] However, the sample size (27 diabetic patients with CKD) was smaller than the present study. The incidence of hypomagnesaemia in type 2 diabetes mellitus patient is 13.5 to 47.7%.[13, 25-27] The wide range may be attributed to the difference in definition of hypomagnesaemia, techniques in magnesium measurement and heterogeneity of selected patient cohort.[10] Studies have also reported higher incidence of hypomagnesaemia in women compared with men.[9, 13, 28, 29] The various causes of low serum magnesium in diabetics include diets low in magnesium,[30] osmotic diuresis, insensitivity to insulin affecting intracellular magnesium transport and thereby causing increased loss of extracellular magnesium,[31] extensive use of loop and thiazide diuretics, autonomic neuropathy[10] and reduced tubular reabsorption due to insulin resistance.[32] The frequency of hypomagnesaemia was found to be increasing with increasing stage of chronic kidney disease. Patients with chronic kidney disease maybe hypomagnesaemic due to excessive use of diuretics and proton pump inhibitors, reduced gastrointestinal uptake (due to acidosis, poor nutrition and absorption), low magnesium concentration of dialysate and alcoholism.[8, 33]

The mean duration of diabetes mellitus was 14.2 (±8.2) years and of chronic kidney disease was 3.2 (±3.0) years in patient with hypomagnesaemia. There was no relationship with duration of diabetes mellitus (Pearson's correlation coefficient, r=0.06, p=0.556) and chronic kidney disease (Pearson's correlation coefficient, r=-0.05, p=0.964) with serum magnesium level. Dasgupta et al. revealed mean diabetic duration of 6.8 years in hypomagnesaemic patients[9] while Haque et al. showed mean diabetic duration of 8.85 years and found no direct relationship with serum magnesium and duration of diabetes.[34]

The current study revealed no relationship of serum magnesium with HbA1c and fasting blood glucose level. There are studies showing negative correlation between serum magnesium with HbA1c[35-38] and serum magnesium with fasting blood glucose as well.[35] The non-relationship in the present study can be explained by admission of patients with mostly uncontrolled blood glucose as the people in Bangladesh still mostly consider BIRDEM General Hospital as mainly a diabetic hospital and so get admitted when blood glucose cannot be controlled by home monitoring; hence the results of the correlation with relation to blood glucose level become confounded. However, when the patients with hypomagnesaemia were further subdivided into controlled and uncontrolled group with regards to the blood glucose level then the frequency of hypomagnesaemia was found significantly more in the uncontrolled group of HbA1c (75.7% vs 24.3%, p=0.002), fasting blood glucose (91.9% vs 8.1%, p=0.001) and post meal blood glucose (81.1% vs 18.9%, p=0.001) than those of the controlled group, thus indicating poorer glycaemic control in hypomagnesaemic patients. This result is in agreement with other studies.[9, 36] A meta-analysis of 13 studies concluded that decreased magnesium intake was significantly associated with risk of type 2 diabetes mellitus.[39]

Serum magnesium levels in the present study positively correlated with serum creatinine levels (Pearson's correlation coefficient, r=0.428, p=0.001) and inversely correlated with GFR (Pearson's correlation coefficient, r= -0.275, p= 0.006) in diabetic patients with chronic kidney disease. The finding is similar to another study that involved 56 chronic kidney disease patients, who were further subdivided in diabetic and non-diabetic group.[19] Interestingly, other studies reported negative correlation between serum magnesium and creatinine clearance in chronic kidney disease without diabetes but serum magnesium level were not correlated with serum creatinine levels and GFR in diabetic patients.[7, 40] In moderate chronic kidney disease, the fractional excretion of magnesium increases such that it compensates for the loss of renal function and maintains serum magnesium levels in normal range. When GFR falls to <30 ml/min/$m^2$ the excretion of magnesium tends to decrease and cannot be compensated any longer by increased fractional excretion of magnesium, resulting in hypermagnesaemia.[6] Thus chronic kidney disease patients with diabetes may be similar to those without diabetes when declining renal function was accompanied by increased serum magnesium levels.[19] As such renal failure patients might be vulnerable to changes in magnesium intake.

There are some potential limitations to the study. Firstly, the relatively small sample size may decrease the power of statistical analysis among sub groups. Inadequate statistical power may provide only pilot results for data analysis. Since the study period was only 6 months, so large sample could not be included. Secondly, most of the study population were residents in urban area so the results may not be representative of the general population. Thirdly, the cross sectional design of the study prevented showing any causal relationship between magnesium and glycaemic or renal status. Fourthly, the study used serum magnesium level to evaluate the magnesium status. Measurement of intracellular magnesium content is a more sensitive marker for magnesium balance.[41] However, serum magnesium level correlates fairly well with intracellular magnesium level, even though only 1% of total body magnesium exists extracellularly.[42] Lastly, only one centre (BIRDEM General Hospital) was enrolled in the study. Involvement of multiple centres was not only labourious but expensive as well.

## Conclusion

The frequency of hypomagnesaemia among diabetic patients with chronic kidney disease is common. Glycaemic control is

poor in patients with low magnesium level. Serum magnesium is positively correlated with GFR and negatively correlated with serum creatinine. It would be prudent to routinely monitor for hypomagnesaemia in diabetic patients with chronic kidney disease and the condition be treated whenever possible.

**Recommendation**

A study with larger sample size involving multiple centres could be done for an even better result of the present study. Further study can be done to see the frequency of hypomagnesaemia in non-diabetic patients with chronic kidney disease. Interventional studies are needed to see whether restoration of magnesium level by diet or medication could improve disease control and preclude potential longitudinal complications as well as to see the mechanisms behind it. Further research is required to determine if low serum magnesium is itself nephrotoxic.

**Conflict of interest**

There is no conflict of interest.

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