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Is Hypomagnesemia a Risk Factor for Atherogenic Dyslipidemia in Patients with Chronic Kidney Disease?

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What is known on this subject?

Atherosclerosis is an important cause for increased morbidity and mortality in chronic renal failure. Magnesium may have possible positive effects on the cardiovascular system due to endothelial-mediated vasodilatation, improved lipid metabolism, reduced inflammation, and inhibition of the platelet functions.

What this study adds?

This study demonstrated that there was no relationship between magnesium levels and atherogenic dyslipidemia in patients with chronic kidney disease.

ABSTRACT

Objective: Atherosclerosis, which starts from early stages of chronic kidney disease (CKD), is an important cause for increased morbidity and mortality. We aimed to investigate whether hypomagnesemia is a marker of increased atherogenic dyslipidemia in patients with CKD with a glomerular filtration rate (GFR) <60 mL/min/1.73 sq m.

Material and Methods: In our study, a total of 80 patients who did not receive renal replacement therapy with GFR <60 mL/min/1.73 sq m, who were diagnosed with CKD and abided by the study entry criteria were retrospectively studied. Patients' gender, age, presence of comorbid disease(d), medications being used, and laboratory findings were recorded. Urea, creatinine, serum electrolytes [calcium, phosphorus, magnesium (Mg)], uric acid, fasting blood glucose, glycosylated hemoglobin, albuminuria/creatinine in spot urine, creatinine clearance, and lipid profile levels were examined.

Results: A total of 36 (45%) male and 44 (55%) female patients were included in the study. The average age was 62.79±14.08 years. Diabetes mellitus was present in 32 (40%) patients, hypertension in 53 (66.25%) patients, and hyperlipidemia in 14 (17.50%) patients. The mean Mg value of our patients was 1.83±0.35. Average for lipid levels were total cholesterol (174.59±57), triglycerides (TG) (173.59±86.85), low-density lipoprotein-C (102.42±43.61), high-density lipoprotein cholesterol (HDL-C) (38.68±12.28), non-HDL-C (134.75±49.72), TG/HDL-C (4.93±3.09), and atherogenic index in plasma (0.61±0.28). Patients were divided into two groups according to their Mg levels. Patients whose Mg levels were <1.7 mg/dL were in group A, and patients whose Mg levels were ≥1.7 mg/dL in group B. When the parameters were compared between the groups, the difference between the two groups was not statistically significant (p>0.05).

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ABSTRACT

Conclusion: In this study conducted in patients with CKD, there was no relationship between Mg levels and lipid parameters. There is a need for larger, more comprehensive, prospective studies on this issue.

Keywords: Chronic renal disease, magnesium, total cholesterol, triglyceride, HDL-C, LDL-C

Introduction

Chronic kidney disease (CKD) is characterized by irreversible and progressive nephron loss and is an important public health problem with increasing prevalence and high morbidity-mortality (1). Atherosclerosis beginning in the early stage of CKD is an important cause for increased morbidity and mortality (2). Increased oxidative stress in CKD accelerates atherosclerosis. Oxidative stress causes peroxidation of atherogenic lipids that play an important role in cardiovascular disease (3).

Abnormalities in lipid metabolism are known to be very important in the development of atherosclerosis. A variety of lipid abnormalities like increased serum total cholesterol (TC), low-density lipoprotein cholesterol (LDL-C) levels, and reduced high-density lipoprotein cholesterol (HDL-C), clearly increase the risk of coronary heart disease (CHD). Experimental and clinical studies revealed that one of the major changeable causes of atherosclerosis is hyperlipidemia (HL). Antihyperlipidemic medication and medical nutrition treatments are recommended to correct lipid metabolism disorders in patients to prevent complications linked to atherosclerosis like CHD, cerebrovascular events, peripheral artery disease, and hypertension (HTN) (4).

Kidneys play a very important role in serum magnesium (Mg) concentration and homeostasis (5). There has been focus on possible positive effects of Mg on the cardiovascular system due to endothelial-mediated vasodilatation, improved lipid metabolism, reduced inflammation, and inhibition of platelet functions by Mg. Low Mg levels in circulation are associated with increased blood pressure, atherogenic dyslipidemia, clotting disorders, inflammatory burden, oxidative stress, carotid wall thickness, and increased CHD (6,7).

In this study, the aim was to investigate the correlation between serum Mg levels and serum lipid parameters among patients with CKD.

Material and Methods

Patients attending the internal medicine clinic as outpatients were investigated. The study retrospectively assessed 80 patients who were not receiving renal replacement therapy with glomerular filtration rate (GFR) <60 mL/min/1.73 m², a diagnosis of CKD, and who abided by the study criteria. Exclusion criteria for the study included those receiving renal replacement therapy like hemodialysis, peritoneal dialysis, or kidney transplant; those using Mg; and those with a history of CHD, malignancy, acute-chronic infection or inflammatory diseases, cirrhosis, pregnancy, or the presence of any hematologic disease. The patients' gender, age, comorbid diseases, medications used, and laboratory results were recorded.

Urea, creatinine, serum electrolyte levels (calcium, phosphorus, Mg), uric acid, fasting blood glucose, glycosylated hemoglobin (HbA1c), albuminuria/creatinine in spot urine, creatinine clearance, and lipid profile levels were investigated. All biochemical tests were studied with a Beckman Coulter Chemistry Analyzer AU680 (AC 208/220/230/240 V, SN: 2017025450). When calculating the serum TC, HDL-C, and triglycerides (TG) levels, an autoanalyzer with a spectrophotometric measurement method was used. LDL-C was calculated using the Fridewald formula as a routine. In the presence of TG >400 mg/DL, the spectrophotometric measurement method with an autoanalyzer was used. The Fridewald formula was LDL-C=[TC - (HDL-C + TG/5)]. Non-HDL-C=TC - HDL-C and atherogenic index of plasma (AIP)=Log (TG/HDL-K) were also calculated. The GFR was calculated based on the Modification of Diet in Renal Disease Study Group (MDRD) formula. The MDRD study group formula is [186 × (serum creatinine) - 1.154 × (age) - 0.203 × (0.742 if female) × (1.21 if African descent)]. All patients had spot albumin/creatinine measured in the first morning urine.

This study was performed according to the guidelines of the Declaration of Helsinki, and it was approved by the Ethics Review Committee of İstanbul Taksim Training and Research Hospital (date: 07.02.2018, number: 66).

Statistical Analysis

Descriptive statistics were used for continuous variables (mean, standard deviation, minimum, median, maximum). Comparison of more than two independent variables abiding by the normal distribution used the Kruskal-Wallis test. For

comparison of two independent variables without normal distribution, the Mann-Whitney U test was used. To analyze the relationship between two continuous variables without normal distribution, Spearman's rho correlation was used. Statistical significance was set at $p<0.05$. Analyses were completed using MedCalc Statistical Software version 12.7.7 (MedCalc Software bvba, Ostend, Belgium; http://www.medcalc.org; 2013) program.

Results

Out of all the patients included in our study, total of about 36 were male (45%) and 44 were female (55%). The patients were aged from 25 to 85 years with a mean age of 62.79±14.08 (median: 66). When assessed in terms of chronic diseases, 32 patients had diabetes mellitus (DM) (40%), 53 had HTN (66.25%), and 14 had HL (17.5%). When the medication use of patients is investigated, 15 patients used antihyperlipidemics (18.75%), 33 patients used diuretics (41.25%), five patients used oral antidiabetic medication (OAD) (6.25%), 45 patients used antihypertensives (57.5%), and 30 patients used insulin (37.5%).

The mean Mg levels of patients were 1.83±0.35 mg/dL (minimum: 1.24 mg/dL, maximum: 3.2 mg/dL) (Table 1). There were 39 patients (48.75%) with Mg level <1.7 mg/dL and 41 patients (51.25%) with ≥1.7 mg/dL, and these were named group A and group B, respectively. Comparisons were performed between the two groups in terms of age, gender, epidermal GFR (eGFR) level, urea, creatinine, uric acid, albuminuria/creatinine in spot urine, LDL-C, TG, TC, HDL-C, non-HDL-C AIP, diagnoses (DM, HTN, HL), and medications used (Table 2).

There were no significant differences between groups A and B when compared in terms of age, gender, diabetes and HTN, diuretic medication and antidiabetic medication (OAD and insulin) use ($p>0.05$). There were three patients in group B (7.32%) and 11 patients in group A (28.21%) with HL diagnosis, and this difference was statistically significant ($p=0.014$). There were four patients in group B (9.76%) and 11 patients in group A (28.21%) using antilipidemic medication, and this difference was statistically significant ($p=0.035$). Antihypertensive medication use was present among 18 patients in group B (43.90%) and 27 patients in group A (71.79%). The difference was identified to be statistically significant ($p=0.012$) (Table 3).

When examined in terms of biochemical parameters, the LDL-C levels measured in groups A and B had mean values of 99.05±51.01 g/dL and 105.63±35.55 g/dL, and the difference was not found to be statistically significant ($p=0.513$). The mean values for non-HDL-C levels measured in groups B and A were 140.59±41.63 g/dL and 128.62±56.92 g/dL, and the difference was not significant ($p=0.289$). When examined in terms of plasminogen activator inhibitor (Log TG/HDL), the mean values in groups B and A were 0.62±0.24 and 0.6±0.32, and the difference was not statistically significant ($p=0.789$) (Table 2).

Table 1. Laboratory characteristics of the patients

Parameters	Mean ± standard deviation
Glucose	125.24±56.99 107 (75-401)
HbA1c	7±1.73 6.4 (4-11.9)
Urea	103.43±42.61 96.5 (40-261)
Creatinine	2.84±1.01 2.65 (1.14-6.2)
Uric acid	7.5±3.05 7.02 (2.9-25.7)
eGFR	29.81±13.36 26.28 (9.09-59)
Total cholesterol	174.59±57 173.5 (64-324)
TG	173.59±86.85 151 (32-462)
LDL-C	102.42±43.61 106 (20-229)
HDL-C	38.68±12.28 36.5 (15-74)
Non-HDL-C	134.75±49.72 131.5 (41-267)
AIP	4.93±3.09 4.05 (0.76-15.9)
Magnesium	1.83±0.35 1.8 (1.24-3.2)
Calcium	8.66±0.87 8.9 (5.8-11.2)
Phosphorus	4.29±1.24 4.11 (1.83-8.3)
Calcium x phosphorus	36.98±11.08 34.6 (15.9-82.1)
Albuminuria/creatinine	972.8±1658.43 518.3 (5-7809)
AIP	0.61±0.28 0.61 (-0.12-1.2)

Student's t-test p, Mann-Whitney U test p. HbA1c: Glycosylated hemoglobin, eGFR: Epidermal glomerular filtration rate, TG: Triglycerides, LDL-C: Low-density lipoprotein cholesterol, HDL-C: High-density lipoprotein cholesterol, AIP: Atherogenic index of plasma

Groups B and A were compared in terms of eGFR in our study. In groups B and A, the mean eGFR values were identified as 29.35±13.27 mL/min/1.73 m² and 30.3±13.6 mL/dk/1.73 m². There was no difference in statistical terms between these values (p=0.825). When albumin/creatinine in spot urine were examined, the mean values in groups A and B were 1001.44±1665.39 and 951.03±1687.12, and the difference was not accepted as statistically significant (p=0.804) (Table 2).

There was no statistically significant correlation between Mg and any parameter (Spearman's rho p>0.05) (Table 4).

Discussion

Much evidence obtained from *in vitro* studies, animal models, and observational studies show that low Mg levels are associated with endothelial dysfunction, atherosclerosis, and vascular calcification (8). Hypomagnesemia may be associated with increased cardiovascular mortality in CKD and more rapid reduction in kidney functions (9). There are many studies showing inadequate Mg intake and/or hypomagnesemia increase inflammation, oxidative stress, insulin resistance, and HL (8).

Van Laecke et al. (9) researched whether there was a correlation with the prognostic significance between serum Mg level, and mortality linked to all causes in patients with CKD diagnosis who were not receiving renal replacement treatment. In this study including 1.650 patients, at the end of mean 5.1 years follow-up duration, a total of 284 deaths were observed. Mean serum Mg level was found to be 2.09±0.27 mg/dL. They compared two groups with serum Mg level <1.8 mg/dL and >2.2 mg/dL. In the hypomagnesemia group, the mortality risk linked to all causes was observed to increase by 61% (9).

A study by Lacson et al. (10) investigated whether there was a relationship between serum Mg level and mortality linked to all causes in 27,554 patients receiving hemodialysis treatment. Patients included in the study were divided into seven groups according to the serum Mg level, and the mean serum Mg level for all patients was identified as 1.86±0.32 mg/dL. At the end of 2 year follow-up, a total of 4.531 deaths were observed. Mortality linked to all causes was identified to be highest in the group with the lowest serum Mg level (Mg <1.30 mg/dL). Moving from the group with the lowest Mg level to the next highest group (Mg >2.50 mg/dL), mortality linked to all causes appeared to reduce (10). As stated above, in studies by Van Laecke et al. (9) and Lacson (10), mean serum Mg levels were 2.09±0.27 mg/dL and 1.86±0.32 mg/dL, respectively. In our study, serum Mg levels were identified to be close to the

Table 2. Comparison of patient groups according to the parameters

	A group	B group	p
Total cholesterol	169.23±65.22 164 (64-324)	179.68±48.18 182 (66-285)	0.420
Triglyceride	177.03±102.08 146 (32-462)	170.32±70.53 159 (54-407)	0.679
LDL-C	99.05±51.01 95.5 (20-229)	105.63±35.55 107.5 (34-197)	0.513
HDL-C	38.23±12.69 37 (15-72)	39.1±12.02 36 (17-74)	0.773
Non-HDL-C	128.62±56.92 112 (41-267)	140.59±41.63 138 (45-242)	0.289
TG/HDL	5.05±3.37 4.03 (0.76-15.6)	4.81±2.83 4.1 (1.24-15.9)	0.795
eGFR	30.3±13.6 26.5 (15.5-59)	29.35±13.27 25.89 (9.09-58)	0.825
Age	63.44±12.41 65 (30-85)	62.17±15.63 68 (25-84)	0.765
Albuminuria/creatinine	1001.44±1665.39 477.5 (5-7010)	951.03±1687.17 541 (15.8-7809)	0.804
AIP	0.6±0.32 0.61 (-0.12-1.19)	0.62±0.24 0.61 (0.09-1.2)	0.789

Student's t-test p, Mann-Whitney U test p. LDL-C: Low-density lipoprotein cholesterol, HDL-C: High-density lipoprotein cholesterol, TG: Triglycerides, eGFR: Epidermal glomerular filtration rate, AIP: Atherogenic index of plasma

Table 3. Comparison of patient groups according to various parameters

		A group		B group		p
		N	%	N	%	
Gender	Male	18	46.15	18	43.90	0.840
	Female	21	53.85	23	56.10	
HTN	No	11	28.21	16	39.02	0.306
	Yes	28	71.79	25	60.98	
DM	No	21	53.85	27	65.85	0.273
	Yes	18	46.15	14	34.15	
HL	No	28	71.79	38	92.68	0.014$^{\beta}$
	Yes	11	28.21	3	7.32	
Antilipidemic	No	28	71.79	37	90.24	0.035$^{\beta}$
	Yes	11	28.21	4	9.76	
Diuretic	No	22	56.41	25	60.98	0.678
	Yes	17	43.59	16	39.02	
Antihypertensive	No	11	28.21	23	56.10	0.012$^{\beta}$
	Yes	28	71.79	18	43.90	
OAD	No	36	92.31	39	95.12	0.671
	Yes	3	7.69	2	4.88	
Insulin	No	21	53.85	29	70.73	0.119
	Yes	18	46.15	12	29.27	

Pearson chi-square, $^{\beta}p<0.05$, HTN: Hypertension, DM: Diabetes mellitus, HL: Hyperlipidemia, OAD: Oral antidiabetic medication

Table 4. Correlation between magnesium with other laboratory parameters

Parameters		Magnesium correlation
Total cholesterol	R	0.181
	P	0.108
TG	R	0.097
	P	0.394
LDL-C	R	0.119
	P	0.300
HDL-C	R	0.079
	P	0.486
Non-HDL-C	R	0.201
	P	0.073
TG/HDL	R	0.080
	P	0.483
eGFR	R	0.012
	P	0.918
Age	R	0.047
	P	0.681

Spearman's rho β p<0.05. TG: Triglycerides, LDL-C: Low-density lipoprotein cholesterol, HDL-C: High-density lipoprotein cholesterol, eGFR: Epidermal glomerular filtration rate

lower limit of 1.83±0.35 mg/dL. When examined from this aspect, mean serum Mg levels in our study were like those of the study by Lacson et al (10).

A study by Dey et al. (11) investigated the correlation between hypomagnesemia and atherogenic dyslipidemia. In this study, they compared 90 patients with grade 2-5 CKD, and hypomagnesemia with 90 people from the healthy population. In the group with hypomagnesemia, TC (p<0.001), LDL-C (p<0.001), and non-HDL-C (p<0.001) values were identified to be higher, and this difference was found to be statistically significant. However, they did not identify any statistical difference for very low-density lipoprotein cholesterol (VLDL-C), HDL-C, and TG levels. At the same time, all these parameters were correlated with the severity of CKD (11). In this study by Dey et al. (11), the patient group with CKD and hypomagnesemia were compared with a healthy population. The significant difference in the results of their study might have been due to the difference in the demographic characteristics between the two groups included in the study. In our study, CKD patients with hypomagnesemia were compared to CKD patients without hypomagnesemia.

Robles et al. (12) performed a study to investigate whether there was a positive correlation between Mg levels and serum

lipid parameters in patients receiving hemodialysis treatment. In the study including 25 non-diabetic CKD patients receiving hemodialysis treatment, there were positive correlations identified between Mg with TC ($p<0.001$), LDL-C ($p<0.01$), VLDL-C ($p<0.001$), and apolipoprotein E ($p<0.01$) (12).

Ansari et al. (13) performed a study to investigate whether there was a positive correlation between serum Mg levels with dyslipidemia in patients with end-stage renal failure receiving hemodialysis treatment. In this study comprising 50 patients, there were clear positive correlations identified between serum Mg level with lipoprotein a ($p<0.007$), serum HDL ($p<0.01$), and serum TG ($p<0.005$) (13).

Baradaran and Nasri (14) performed a study to investigate whether there was a correlation between serum Mg level with dyslipidemia among hemodialysis patients. In this study including 36 patients, clear positive correlations were identified between serum Mg with lipoprotein a ($p<0.05$) and serum TG ($p<0.05$). There were no correlations identified between serum Mg with TC, HDL-C, and LDL-C ($p>0.05$) (14).

We completed our study in patients with CKD who were not receiving renal replacement treatment. As stated in detail above, though these three studies identified correlations between serum Mg level with a variety of lipid parameters, in our study, despite the lack of a significant correlation between hypomagnesemia and lipid parameters, there were higher rates of HL diagnosis, antihyperlipidemic, and antihypertensive medication use rates in the hypomagnesemia group. Based on these findings, we think hypomagnesemia may be associated with dyslipidemia and HTN development.

A study divided 144 patients with type-2 diabetic nephropathy and 311 patients with non-diabetic CKD into two classes according to serum Mg levels (≤1.8 and >1.8 mg/dL). Among diabetic nephropathy patients, the group with low serum Mg was found to have a 2.12-fold higher risk of end-stage renal disease compared to the group with high serum Mg levels. In this study, it was proposed that Mg supplementation may have a renoprotective effect in type-2 diabetic nephropathy patients (15).

Many studies found that hypomagnesemia was associated with a reduction in kidney functions. In our study, there was no correlation between serum Mg level with eGFR in the correlation study. We connect the lack of identification of a significant correlation between these parameters to the lack of prospective examination in our study, and the lack of follow-up for progression in the patients.

Study Limitations

The most important limitation of our study is that patients had chronic diseases like HTN, DM, and HL in addition to CKD diagnosis, and for this reason used, medications that may affect Mg and lipid levels. This may have affected the results of the study.

Conclusion

In our study on patients with CKD, there was no correlation between Mg levels with lipid parameters. However, those with hypomagnesemia had higher antihyperlipidemic and antihypertensive medication use rates, which led to the consideration of a correlation between hypomagnesemia with dyslipidemia and HTN development. There is a need for large scale, broad scope, prospective studies investigating this topic.

Ethics

Ethics Committee Approval: This study was performed according to the guidelines of the Declaration of Helsinki, and it was approved by the Ethics Review Committee of İstanbul Taksim Training and Research Hospital (date: 07.02.2018, number: 66).

Informed Consent: Retrospective study.

Peer-review: Internally peer-reviewed.

Authorship Contributions

Concept: B.B., Design: B.B., Data Collection or Processing: S.K., Analysis or Interpretation: O.M., B.B., Literature Search: S.K., O.M., B.B., Writing: S.K., O.M., B.B.

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