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The Relationship Between Serum Magnesium Level, Plasma Atherogenic Index and Glomerular Filtration Rate

Serum Magnezyum Düzeyi, Plazma Aterojenik İndeksi ve Glomerüler Filtrasyon Hızı Arasındaki İlişki

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ABSTRACT

Aim: Magnesium is associated with many chronic diseases, especially cardiovascular diseases. The purpose of this study is to evaluate the relationship between serum magnesium level, glomerular filtration rate (GFR) and atherogenic index of plasma (AIP).

Materials and Methods: The data of 214 patients included in the study were analyzed retrospectively. Those with an estimated GFR of 60 mL/minute/1.73 m^2 and above were grouped as the "high GFR group", and those with an estimated GFR of less than 60 mL/minute/1.73 m^2 were grouped as the "low GFR group". AIP was calculated by taking the logarithm of the ratio of serum triglyceride level to serum high-density lipoprotein level.

Results: There were 72 patients in the high GFR group and 142 patients in the low GFR group. There was no difference between the groups in terms of serum magnesium level and AIP. The factors related to serum magnesium level and AIP were investigated in the whole study population. A negative correlation was found between serum magnesium level and AIP [Odds ratio (OR): -0.212]. When the factors associated with AIP were examined, AIP was found to be negatively correlated with serum magnesium level (OR: -0.189) whereas positively correlated with body mass index, systolic blood pressure and serum uric acid level (OR: 0.154; 0.276; 0.165, respectively). There was no relationship between AIP and magnesium levels and GFR.

Conclusion: The inverse relationship between serum magnesium level and AIP is consistent with the literature on the relationship between hypomagnesemia and atherogenicity. The lack of a relationship between AIP and serum magnesium level and GFR may be attributed to the small number of patients.

Keywords: Glomerular filtration rate, magnesium, atherogenic index of plasma

ÖZ

Amaç: Magnezyum, kardiyovasküler hastalıklar başta olmak üzere birçok kronik hastalıkla ilişkilendirilmektedir. Bu çalışmanın amacı, serum magnezyum düzeyi, plazma aterojenik indeksi (PAİ) ve glomerüler filtrasyon hızı (GFH) arasındaki ilişkinin değerlendirilmesidir.

Gereç ve Yöntem: Çalışmaya dahil edilen 214 hastanın verileri retrospektif olarak incelendi. Tahmini GFH 60 mL/dakika/1,73 m^2 ve üzerinde olanlar "yüksek GFH grubu", 60 mL/dakika/1,73 m^2'den düşük olanlar "düşük GFH grubu" olarak gruplandırıldı. Serum trigliserit düzeyinin serum yüksek yoğunluklu lipoprotein düzeyine oranının logaritması alınarak PAİ hesaplandı.

Bulgular: yüksek GFH grubunda 72, düşük GFH grubunda 142 hasta yer aldı. Serum magnezyum düzeyi ve PAİ açısından gruplar arasında farklılık saptanmadı. Tüm popülasyonda serum magnezyum düzeyi ile ilişkili faktörler incelendiğinde, sadece PAİ ile magnezyum arasında negatif yönde bir ilişki saptandı [Odds ratio (OR): -0,212]. Yine tüm popülasyonda PAİ ile ilişkili faktörler incelendiğinde, PAİ ile serum magnezyum düzeyi arasında negatif (OR: -0,189); vücut kitle indeksi, sistolik kan basıncı ve serum ürik asit düzeyi arasında pozitif yönde bir ilişki saptandı (sırasıyla; OR: 0,154; 0,276; 0,165). Gerek PAİ, gerekse magnezyum düzeyleri ile tahmini glomerüler filtrasyon hızı arasında ilişki saptanmadı.

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Sonuç: Serum magnezyum düzeyi ile PAİ arasındaki ters yönlü ilişki, hipomagnezeminin aterojenite ile ilişkisine dair genel literatür bilgisi ile uyumludur. PAİ ve serum magnezyum düzeyi ile GFH arasında bir ilişkinin saptanmamış olması, hasta sayısının azlığına bağlanabilir.

Anahtar Kelimeler: Glomerüler filtrasyon hızı, magnezyum, plazma aterojenik indeksi

INTRODUCTION

Magnesium is the major intracellular cation in the body after potassium and is a cofactor of more than 300 enzymes that play role in many vital processes, including blood pressure regulation, glucose metabolism and lipid peroxidation[1]. The total body magnesium amount in an adult is approximately 22-26 g, and 50-60% of this amount is found in the bones. Serum levels of magnesium reflect approximately 1% of the intracellular component (1.2-2.5 mg/dL). From the perspective of the chronic kidney disease (CKD) course, magnesium retention is expected to increase, especially at values below 30 mL/minute/1.73 m^2, together with the decrease in glomerular filtration rate (GFR)[2]. Western-style diet with low vegetable-fruit content, malabsorption, use of diuretics and laxatives are the main causes of hypomagnesemia[3]. It has been suggested that hypomagnesemia causes an increase in inflammation, oxidative stress and lipid peroxidation and thus plays a role in the pathogenesis of diabetes, metabolic syndrome, cardiovascular diseases and endothelial damage[4-10]. Information on magnesium metabolism in the CKD process is limited. In recent years, the number of studies showing that hypomagnesemia may play a role in the pathogenesis, progression and complications of CKD has been increasing[11-16].

Dyslipidemia is a major risk factor for cardiovascular diseases and related complications, affecting approximately half of the population in developed countries. Despite being so common, awareness and effective treatment rates are quite low[17]. CKD is a process in which the frequency of hyperlipidemia increases with decreasing GFR. On the other hand, hyperlipidemia has also been reported to accelerate CKD progression[18,19]. Hypomagnesemia is one of the lesser known and partially newly defined risk factors that may be associated with hyperlipidemia[20,21].

In order to determine the cardiovascular risk profile associated with hyperlipidemia, inexpensive and easy-to-use indices including serum lipid profile and some hematological/biochemical parameters have been defined in clinical practice. Atherogenic index of plasma (AIP), being one of them, is the logarithm of the ratio obtained by dividing the plasma triglyceride level by the plasma high-density lipoprotein (HDL) level, and it has been reported to be well correlated with cardiovascular risk[22-24].

The aim of this study is to compare the high GFR and low GFR groups in terms of serum magnesium level and AIP, and to evaluate the relationship between serum magnesium level, GFR and AIP.

MATERIALS AND METHODS

Electronic medical records of 1412 adult patients attending nephrology outpatient clinic at the Tekirdağ Namık Kemal University Hospital between June 2021 and January 2022 were reviewed retrospectively. The following exclusion criteria were used: taking magnesium replacement and lipid-lowering agent, being on dialysis treatment; having acute/chronic diarrhea, reduced oral intake, diabetes mellitus, hereditary renal tubular disorders, acute kidney injury (in last 6 weeks) and active systemic infectious, inflammatory or malignant disease. Two hundred and fourteen patients were included in the evaluation. Estimated glomerular filtration rate (tGFR) value was calculated with the CKD-EPI formula[25]. Those with a tGFR value of 60 mL/minute/1.73 m^2 and above were grouped as the "high-GFR group", and those below 60 mL/minute/1.73 m^2 were grouped as the ""low-GFR group". AIP value is calculated by taking the logarithm of the ratio of serum triglyceride level to serum HDL level and body mass index (BMI) was calculated by dividing the patient's body weight (kg) by the square of the patient's height (m^2).

Statistical Analysis

Statistical analysis was performed using Statistical Package of Social Science (SPSS) version 25 software. Compatibility with normal distribution of parametric variables was evaluated using Shapiro-Wilk test. The Student's t-test was applied in the comparison of normally distributed data, and the Mann–Whitney U test for non-normally distributed data. The chi-square test was used to compare categorical variables. Multivariate linear regression analysis was performed for the parameters found to be associated with serum magnesium level and AIP as a result of Pearson' correlation analysis.

P value < 0.05 were regarded as statistically significant.

RESULTS

General Characteristics of the Study Group

Of the 214 patients included in the study, 48.6% were male and 51.4% were female. The mean age of the patients was 59.7±14.1 years, and BMI was 29.4±5.4 kg/m^2. The rate of patients with a diagnosis of hypertension was 83.6%, and the rate of patients with coronary artery disease was 33.6%. The rates of patients using angiotensin-converting enzyme inhibitors (ACEI) or angiotensin receptor blockers (ARB); calcium channel blockers (CCB); thiazide and beta blockers

were 64.5%; 34.6%; 40,2% and 41.4%, respectively. The mean serum creatinine level in the study group was 1.66±0.88 mg/dL; mean tGFR was 51.2±28.0 mL/minute/1.73 m^2, mean serum magnesium level was 1.97±0.26 and the mean AIP was found to be 0.14±0.28. Serum magnesium levels were similar in patients using and not using thiazide (2.00±0.28 mg/dL vs. 1.95±0.24 mg/dL; p=0.146).

Comparison of the Groups

There were 72 patients in the high-GFR group and 142 patients in the low-GFR group. There was no difference between the groups in terms of serum magnesium level and AIP. Serum glucose, uric acid, C-reactive protein levels and proteinuria were higher in the low-GFR group; serum albumin and HDL levels were found to be lower (Table 1). The rates of hypertension and coronary artery disease were found to be higher in the low-GFR group (90.1% vs. 70.8%, p<0.001, 43.7% vs 13.9%, respectively, p<0.001). There was no difference between the groups in terms of use of ACEI/ARB, CCB, thiazide and beta blockers (p=0.666; 0.136; 0.052; 0.783, respectively).

Relationship between serum magnesium level and other parameters

When the entire study group was evaluated by correlation analysis, no correlation was found between tGFR and serum magnesium level. There was a negative correlation between serum magnesium level and serum glucose level, BMI and AIP and a positive correlation was found between serum creatinine, parathormone (PTH) and phosphorus levels. As a result of the multiple linear regression analysis performed by including serum creatinine, glucose and PTH values, AIP and BMI, it was observed that only the relationship between AIP and magnesium continued in the negative direction (OR: -0.212, p=0.006) (Table 2). Phosphorus was not included in the multiple regression analysis because the serum phosphorus level is directly affected by the serum PTH level.

Parameters affecting AIP

When the entire study group was evaluated, no correlation was found between AIP and tGFR. There was a positive correlation

Table 1. Clinical characteristics and laboratory findings of the study group

	High GFR group (n=72)	Low GFR group (n=142)	p
Age (year)	55 (22-80)	64 (19-93)	<0.001
Male n (%)	31 (43.1)	73 (51.4)	0.248
BMI (kg/m^2)	28.9 (16.9-47.9)	29.2 (19.3-45.8)	0.276
Hypertension n (%)	51 (70.8)	128 (90.1)	<0.001
CAD n (%)	10 (13.9)	62 (43.7)	<0.001
SBP (mmHg)	130 (100-230)	140 (100-220)	0.024
DBP (mmHg)	80 (60-140)	80 (55-120)	0.414
Glucose (mg/dL)	100 (76-176)	108 (76-179)	0.020
Creatinine (mg/dL)	0.85 (0.48-1.39)	1.88 (0.60-5.34)	<0.001
eGFR (mL/minute/1.73 m^2)	84 (60-125)	33 (10-59)	<0.001
Uric acid (mg/dL)	5.4 (2.3-7.7)	6.4 (3.6-14)	<0.001
Sodium (mEq/L)	139 (127-144)	139 (126-146)	0.314
Potassium (mEq/L)	4.4±0.4	4.7±0.6	<0.001
Calcium (mg/dL)	9.4±0.5	9.2±0.5	0.089
Phosphorus (mg(dL)	3.5±0.7	3.8±0.7	0.003
Magnesium (mg/dL)	1.95±0.22	1.99±0.27	0.220
Albumin (g/dL)	4.5 (2.2-5.2)	4.3 (2.5-5.2)	0.010
CRP (mg/dL)	2.8 (0.2-32)	4.5 (0.2-51)	0.006
Total cholesterol (mg/dL)	200 (122-458)	192 (98-334)	0.101
LDL (mg/dL)	117 (53-362)	110 (27-216)	0.065
HDL (mg/dL)	50 (23-100)	45 (24-83)	0.010
TG (mg/dL)	150 (43-333)	150 (44-630)	0.551
AIP (logTG/HDL)	0.10±0.28	0.16±0.29	0.116
Spot urine PCR (mg/gr)	0.17 (0.01-9.93)	0.44 (0.01-7.8)	<0.001

*Data are expressed as "mean ± SD" or median (min-max)..

AIP: Plasma atherogenic index, BMI: Body mass index, CAD: Coronary artery disease, SBP: Systolic blood pressure, DBP: Diastolic blood pressure, eGFR: Estimated glomerular filtration rate, CRP: C-reactive protein, PCR: Protein-to-creatinine ratio, LDL: Low-density lipoprotein, HDL: High-density lipoprotein, TG: Triglyceride

between AIP and serum uric acid level, BMI, systolic blood pressure (SBP) and diastolic blood pressure and a negative correlation was found with the serum magnesium level. As a result of multiple linear regression analysis in which these parameters were included, there was a positive correlation between AIP and BMI and between SBP and serum uric acid level (OR: 0.154; 0.276; 0.165, p=0.032; 0.004; 0.016, respectively). A negative correlation (OR: -0.189, p=0.006) was observed between AIP and serum magnesium level (Table 3).

DISCUSSION

In our study, high-GFR and low-GFR groups were similar in terms of serum magnesium level and AIP. No correlation was found between tGFR and serum magnesium level, and between tGFR and AIP in the entire study group. In addition, there was a negative correlation between AIP and serum magnesium level; a positive correlation was found between BMI, SBP and serum uric acid levels, and this result of our study seems to be compatible with the prevailing literature since it fits the known major cardiovascular risk profile.

Although hypomagnesemia is held responsible for increased morbidity and mortality in cardiovascular diseases and diabetes as well as in CKD, the cause-effect relationship in this area is still unclear[26,27]. The most valid and current hypothesis put forward in this process is that hypomagnesemia increases vascular calcification. In addition, it has been reported that inflammation exacerbated in the presence of hypomagnesemia has been reported to accelerate the atherosclerotic process[28,29].

Table 2. Parameters related to serum magnesium level

	Correlation		Multiple linear regression	
	r	p	OR	p
BMI	-0.171	0.017	-0.083	0.286
Creatinine	0.156	0.022	0.040	0.666
Glucose	-0.192	0.005	-0.114	0.129
PTH	0.291	<0.001	0.143	0.118
AIP	-0.217	0.001	-0.212	0.006

BMI: Body mass index, PTH: Parathyroid hormone, AIP: Atherogenic index of plasma, OR: Odds ratio (with 95% confidence interval)

Table 3. Parameters related to plasma atherogenic index

	Correlation		Multiple linear regression	
	R	p	OR	p
BMI	0.247	0.001	0.154	0.032
SBP	0.288	<0.001	0.276	0.004
DBP	0.199	0.003	-0.080	0.408
Uric acid	0.167	0.016	0.165	0.016
Magnesium	-0.217	0.001	-0.189	0.006

BMI: Body mass index, SBP: Systolic blood pressure, DBP: Diastolic blood pressure, OR: Odds ratio (with 95% confidence interval)

Accordingly, it has been reported that interventions to increase serum magnesium level reduce serum phosphorus level, reduce joint pain and inflammation by reducing soft tissue calcification, decrease carotid intima-media thickness, and reduce mortality.

However, the majority of these studies were conducted in the hemodialysis population[14,29-31]. The number of studies on this subject in patients with CKD in earlier stages is few. In one of these studies, a negative correlation was reported between serum magnesium level and endothelial dysfunction[32]. Another study in stage 3 and 4 CKD patients reported that serum magnesium level increased with decreasing GFR, and this rise increased the frequency of cardiovascular events and mortality[26]. In our study, although it was not statistically significant, a negative correlation was found between serum magnesium level and GFR (r=-0.125). On the other hand, although there was a significant positive correlation between serum magnesium and creatinine levels, it became non-significant in multiple regression analysis.

It is well known that, as eGFR declines, the frequency of cardiovascular events increases significantly, which are the main causes of death in the CKD group. Serum lipid levels are among the major components of the cardiovascular risk profile. It is accepted that dyslipidemia also contributes to CKD progression[33]. Important components of dyslipidemia in the CKD course include high serum triglyceride levels, low serum HDL levels, and HDL dysfunction even if its level is normal[34,35]. Although there are many studies evaluating the indicators of hyperlipidemia in CKD patients, the only study in the literature evaluating the relationship between GFR and AIP belongs to Zhou and Shang[36]. 15836 patients were included in this study, and a strong negative correlation was found between AIP and GFR. In addition, it has been reported that this relationship is stronger in the presence of male gender, high BMI, black race, age less than 50, hypertension and/or diabetes. Therefore, it was concluded that the risk of the decline in GFR could be predicted through AIP. In our study, although not statistically significant, a negative correlation was found between AIP and GFR (r=-0.076).

In our study, a significant negative correlation was found between AIP and serum magnesium level, and a positive significant correlation was found between uric acid, SBP and BMI, and our findings are consistent with the literature[24,36-38]. Although there are many studies reporting that magnesium is associated with atherogenicity among these parameters, information about the mechanism of this relationship, the therapeutic use of magnesium and target serum levels is not sufficient. The general conclusion in the literature is that hypomagnesemia adversely affects the serum lipid profile and related indicators[20,39]. In a study by Cambray et al.[21], it was

shown that the lipid profile worsened and carotid intima-media thickness increased in the presence of hypomagnesemia in the CKD population. In a case-control study by Dey et al.[40], it was reported that hypomagnesemia worsened atherogenic dyslipidemia parameters in the CKD group.

Study Limitations

1. In this single-center study, our number of cases was low compared to similar studies in the literature.
2. Since the relationship of magnesium and lipid parameters with GFR change over time was not evaluated, no comment could be made on the contribution of these parameters to CKD progression.

CONCLUSION

In our study, the finding of a relationship between serum magnesium level and AIP in addition to known cardiovascular risk factors supports the view that hypomagnesemia contributes to atherogenicity. Although the number of studies in this area is increasing, there is not enough data to support the use of magnesium for therapeutic purposes. Particular care should be taken in this regard, due to the predisposition to hypermagnesemia in patients with advanced CKD. In conclusion, we think that the relationship of magnesium with cardiovascular and renal survival in both healthy and CKD populations should be evaluated in large population-based cohorts with biochemical and functional parameters that will be repeated over a long period of time.

Ethics

Ethics Committee Approval: The study protocol was prepared in accordance with the Declaration of Helsinki and approved by the Tekirdağ Namık Kemal University Non-Interventional Research Ethics Committee (protocol no: 2021.280.12.03, date: 28.12.2021).

Informed Consent: Retrospective study.

Peer-review: Externally peer-reviewed.

Authorship Contributions

Concept: N.B., Design: N.B., Data Collection or Processing: N.B., Ö.E., V.Y.K., A.Ç., Analysis or Interpretation: N.B., G.Ö., Literature Search: N.B., V.Y.K., Writing: N.B.

Conflict of Interest: No conflict of interest was declared by the authors.

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