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**ORIGINAL ARTICLE**

# Evaluation of Serum Magnesium Levels in Patients with Hypertension: A Case-control Study

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**Abstract**: *There are conflicting data on the effects of magnesium on hypertension in the literature. Therefore we aim this study to investigate whether there is a relationship between serum magnesium levels and hypertension in newly diagnosed. This is a case-control study. The patients were selected from the records of patients who applied to the family medicine outpatient clinic between June 2016 and May 2017. A total of 276 patients, 68 patients with newly diagnosed hypertension in the case group and 208 patients without any disease in the control group were included in the study. Binary logistic regression analysis was performed between the case and control groups. The odds ratio for magnesium was 0.1 (0.012-0.810). When the value of the magnesium variable increases by 1mg/dl, the risk of hypertension decreases by 90%. However, in the presence of magnesium as an independent variable, the predictability rate of our model was expected to increase but decreased. The odds ratio for age was 1.14 (1.092-1.186). The odds ratio for exercise was 0.18 (0.03-0.99). The odds ratio for glucose was 1.03 (1.001-1.065). In our study that magnesium deficiency causes an increased risk of hypertension, the predictability rate of our model was expected to increase instead of decrease. These findings show that a cause-effect approach or incidental association cannot explain the relationship between magnesium and hypertension*.

**Keywords**: *hypertension; magnesium; age; exercise; glucose*

## INTRODUCTION

The prevalence of hypertension continues to increase globally [1]. The prevalence of hypertension in the adult population in the world is 31% [2]. For over forty years, the causes of most hypertensive diseases have been unknown and are defined as primary or essential hypertension. Secondary causes are accepted as 5-10% [3].

Recently, risk factors such as age, sex, race, family history, genetic composition, obesity, excessive salt intake, inactivity or lack of exercise, high-fat diet, tobacco use, and alcohol consumption have been proven to lead to the development of hypertension [4].

Magnesium (Mg) can be found in the fourth sequence in the human body and the second after potassium in the intracellular area [5]. Magnesium is an essential cation playing a crucial role in many physiological functions. It is critical in energy-requiring metabolic processes, protein synthesis, membrane integrity, nervous tissue conduction, neuro-muscular excitability, muscle contraction, and hormone secretion [6]. Given the role of magnesium in calcium and potassium transport, cell signaling, energy metabolism, genome stability, DNA repair, and replication, it should be expected that hypomagnesemia is associated with many diseases, including neurological disorders, osteoporosis, diabetes, coronary heart disease, and hypertension [7].

Magnesium levels were lower in white hypertensives than in normotensives, while no significant difference was found in black hypertensives compared to normotensives [8]. It has been shown that those with essential hypertension have a significantly higher intra-erythrocytic magnesium level than healthy individuals [9]. A systemic review and meta-analysis study suggested no relationship between serum magnesium and hypertension but that dietary magnesium supplementation reduces blood pressure [10]. Magnesium deficiency was found to be responsible for calcium and magnesium disorders in those with pregnancy-induced hypertension, and magnesium sulfate therapy was effective in

these patients [11]. In a meta-analysis of twelve studies, a reduction in diastolic blood pressure was observed, while a decrease in systolic blood pressure was not observed [12]. Again, a meta-analysis of 20 studies found a reduction in systolic blood pressure while no change in diastolic blood pressure [13]. A meta-analysis of 22 studies noted a reduction in both systolic and diastolic blood pressure [14].

The fact that there are conflicting data on the effects of magnesium on hypertension in the literature shows a need for research on this subject. For this purpose, we wanted to evaluate magnesium levels in patients with newly diagnosed hypertension. This study aims to investigate whether there is a relationship between serum magnesium levels and hypertension.

## MATERIALS AND METHODS

### Study Design

This study is a case-control study. The sample size was determined for logistic regression analysis, considering the number of seven independent variables, such as age, gender, body mass index (BMI), smoking, alcohol consumption, exercise status, and magnesium. For each group, using the power analysis method, the minimum sample size of at least 48 which is needed to detect a significant difference when taken into account at 0.05 type-I error (Alpha), 0.35 effect size, 0,80 power (1-beta).

### Population selection

The patients included in the study were selected from those who applied to the family medicine outpatient clinic between June 2016 and May 2017 from the hospital information system and patient records. Sixty-eight people with hypertension constituted the case group. Two hundred-eight people without hypertension formed the control group. The control group was selected considering demographic characteristics such as age and gender.

### Inclusion and exclusion criteria

Among the patients tested for magnesium at the time of admission, patients with hypertension newly diagnosed were included in the case group, and those with no chronic disease and no medication were included in the control group. The study did not include those with any illness and drug use before the application, those with a previous hypertension diagnosis and treatment, and those without magnesium checked at the time of application.

### Statistical analysis

All statistical analyses were performed using IBM SPSS Statistics (V25). A P-value below 0.05 was considered statistically significant. In the data expression, categorical data, numbers and percentages, and numerical data were expressed with averages and standard deviation. The distribution of demographic data was analyzed by frequency tests, comparison of categorical data by chi-square test, and comparison of numerical data by independent sample t-test. The stepwise enter model was used in the binary logistic regression test to evaluate the effect of magnesium on hypertension. Skewness and kurtosis analyses were used to conform the data to the normal distribution.

### Ethical Considerations

Ethical approval was obtained from the İstanbul Training Research Hospital Clinical Research Ethics Committee (Decision no. 14.10.2022, 318).

## RESULTS

A total of 276 people, including 68 cases and 208 control groups, were included in the study. There was no found difference between those with hypertension and those without it in terms of sex, smoking, and alcohol consumption. However, the proportion of those without exercise was found to be lower in the hypertension group (Table 1).

**Table 1:** Comparison of the case and control groups in terms of gender, smoking, alcohol consumption, and exercise status

| Characteristics of participants | | Hypertension N (%) | | | |
|---|---|---|---|---|---|
| | | No | Yes | Total | p |
| Gender | Women | 149 (71.6) | 52 (76.5) | 201 (72.8) | 0.530 |
| | Men | 59 (28.4) | 16 (23.5) | 75 (27.2) | |
| Smoking | No | 167 (80.3) | 58 (85.3) | 225 (81.5) | 0.472 |
| | Yes | 41 (19.7) | 10 (14.7) | 51 (18.5) | |
| Alcohol | No | 204 (98.1) | 68 (100.0) | 272 (98.6) | 0.575 |
| | Yes | 4 (1.9) | 0 (0.0) | 4 (1.4) | |
| Exercise | No | 168 (80.8) | 66 (97.1) | 234 (84.8) | < 0.001* |
| | Yes | 40 (19.2) | 2 (2.9) | 42 (15.2) | |

*Chi-Square Tests: The statistically significant difference was accepted as p < 0.05*

The average age and BMI were found to be higher in the group with hypertension. Additionally, glucose, total cholesterol, triglycerides, and LDL- cholesterol (LDL-C) were found to be increased in the case group. Magnesium levels were found to be higher in the control group than in the case group (Table 2).

**Table 2:** Comparison of case and control groups in terms of age, body mass, and biochemical parameters

| | Hypertension | | | |
|---|---|---|---|---|
| | No | Yes | | |
| | Mean ± SD | | t. | p |
| Glucose (mg/dl) | 95.25 ± 11.04 | 107.79 ± 15.96 | -6.02 | < 0.001* |
| Cholesterol (mg/dl) | 206.55 ± 44.32 | 229.43 ± 35.59 | -3.86 | < 0.001* |
| Triglycerides (mg/dl) | 111.96 ± 61.58 | 154.07 ± 60.27 | -4.92 | < 0.001* |
| HDL-C (mg/dl) | 54.55 ± 12.66 | 52.68 ± 7.40 | 1.49 | 0.137 |
| LDL-C (mg/dl) | 129.35 ± 37.07 | 143.49 ± 29.66 | -3.19 | 0.002* |
| Calcium (mg/dl) | 9.47 ± 0.46 | 9.54 ± 0.43 | -1.11 | 0.266 |
| Magnesium (mg/dl) | 1.99 ± 0.19 | 1.92 ± 0.15 | 2.82 | 0.005* |
| Sodium (mmol/L) | 139.14 ± 1.99 | 145.24 ± 32.97 | -1.52 | 0.133 |
| TSH (mIU/L) | 1.91 ± 1.29 | 1.90 ± 1.20 | 0.06 | 0.950 |
| Vitamin B12 (ng/L) | 231.69 ± 127.26 | 207.32 ± 90.38 | 1.73 | 0.085 |
| Vitamin D (ng/ml) | 20.08 ± 10.69 | 19.32 ± 9.60 | 0.52 | 0.602 |
| Age (years) | 39.69 ± 12.80 | 59.71 ± 11.65 | -11.42 | < 0.001* |
| BMI | 25.83 ± 4.48 | 29.50 ± 6.25 | -4.47 | < 0.001* |

*SD: Standard Deviation; BMI: Body Mass Index; *Independent sample t-test is significant at the 0.05 level (2-tailed)*

Binary logistic regression analysis was performed by considering the different parameters between the case and control groups. In addition to the magnesium, age, exercise, BMI, glucose, triglyceride, and LDL-C variables which were different between the case and control groups, gender and sodium variables were also included in the analysis according to the literature [4]. Total cholesterol was not included in the study because it is affected by LDL-C and triglyceride.

In the analysis in which only magnesium as an independent variable was added, the model is insignificant because the Cox-Snell R2 and Nagelkerke R2 values are smaller than the 0.2 value. However, the model is significant when other independent variables are added. Except for the step in which triglyceride was added, the model's goodness of fit is acceptable since the p-values are bigger than 0.05 in the Hosmer-Lemeshow test. Changes in -2 Log Likelihood values (Chi-square, $p < 0.05$) are significant in magnesium, age, exercise, and glucose steps.

These results show that the logistic regression analysis is generally valid (Table 3).

**Table 3:** Distribution of data showing the validity of the logistic regression analysis

| Metod=Enter: stepwise | -2 Log Likelihood | Chi-square | P | Cox and Snell R Square | Nagelkerke R Square | Hosmer and Lemeshow Test | Predicted percentage |
|---|---|---|---|---|---|---|---|
| Beginning | 308.192 | | | | | | 75.4 |
| + Magnesium | 299.999 | 8.193 | 0.004* | 0.029 | 0.043 | 0.150 | 74.6 |
| + Age | 194.958 | 105.041 | < 0.001* | 0.337 | 0.500 | 0.493 | 82.6 |
| + Exercise | 186.608 | 8.350 | 0.004* | 0.356 | 0.530 | 0.642 | 81.9 |
| + Gender | 186.341 | 0.267 | 0.606 | 0.357 | 0.531 | 0.592 | 81.9 |
| + BMI | 183.822 | 2.519 | 0.112 | 0.363 | 0.539 | 0.695 | 83.3 |
| + Glucose | 179.281 | 4.541 | 0.033* | 0.373 | 0.555 | 0.398 | 84.1 |
| + Triglycerides | 179.265 | 0.015 | 0.901 | 0.373 | 0.555 | 0.044* | 83.3 |
| + LDL-C | 178.064 | 1.201 | 0.273 | 0.376 | 0.559 | 0.744 | 82.6 |
| + Sodium | 178.059 | 0.005 | 0.944 | 0.376 | 0.559 | 0.746 | 82.6 |

**The statistically significant difference was accepted as $p < 0.05$*

The odds ratio for magnesium was found to be 0.1 (0.012-0.810). When the value of the magnesium variable increases by 1mg/dl, the risk of hypertension decreases by 90% (Table 4). However, in the presence of magnesium as an independent variable, the predictability rate of our model was expected to increase but decreased (Table 3). The odds ratio for age was found to be 1.14 (1.092-1.186). When the value of the age variable increases by one unit, the risk of hypertension increases by 1.14 times. The odds ratio for exercise was found to be 0.18 (0.03-0.99). When the reference group is taken as those with non-exercise, the risk of hypertension was found to be 82% less in those with exercise. The odds ratio for glucose was found to be 1.03 (1.001-1.065). When the value of the glucose variable increases by 1 mg/dl, the risk of hypertension increases by 1.03 times (Table 4).between vitamin D and total cholesterol and LDL-C in the young age group. However, serum vitamin D had no significant correlation with serum glucose, triglyceride, HDL-C, calcium, magnesium, sodium, TSH levels, and BMI scores (Table 4).

**Table 4:** Logistic regression analysis showing the relationship between hypertension and variables of magnesium, age, exercise, gender, BMI, glucose, triglyceride, LDL-C, and sodium

| Hypertension[a] | B | SE | Wald | P | Risk (Odds) coefficient | 95% C.I.for (Exp B) | |
|---|---|---|---|---|---|---|---|
| | | | | | | Lower | Upper |
| Magnesium (mg/dl) | -2.325 | 1.079 | 4.645 | 0.031* | 0.1 | 0.012 | 0.810 |
| Age (years) | 0.129 | 0.021 | 37.532 | < 0.001* | 1.14 | 1.092 | 1.186 |
| Exercise (1: -/+) | -1.723 | 0.874 | 3.882 | 0.049* | 0.18 | 0.032 | 0.991 |
| Gender (1: Women/Men) | -0.628 | 0.456 | 1.898 | 0.168 | 0.53 | 0.219 | 1.304 |
| BMI | 0.031 | 0.040 | 0.621 | 0.431 | 1.03 | 0.955 | 1.115 |
| Glucose (mg/dl) | 0.032 | 0.016 | 4.140 | 0.042* | 1.03 | 1.001 | 1.065 |
| Triglycerides (mg/dl) | 0.001 | 0.003 | 0.019 | 0.890 | 1.00 | 0.994 | 1.007 |
| LDL-C (mg/dl) | -0.006 | 0.006 | 1.172 | 0.279 | 0.99 | 0.983 | 1.005 |
| Sodium (mmol/L) | 0.002 | 0.034 | 1.897 | 0.947 | 1.00 | 0.938 | 1.071 |

*[a]Dependent Variable: Hypertension; BMI: Body Mass Index; B: Estimated Coefficients; CI: Confidence Interval; SE: Standard Error; *Logistic regression analysis is significant at the p < 0.05 level (2-tailed)*

**DISCUSSION**

The fact that the age and BMI values, glucose, total cholesterol, triglycerides, and LDL-C levels are high in those with hypertension and the low rate of exercisers shows that the study groups are compatible with the general population.

In our study, magnesium levels were lower in those with hypertension. When the value of the magnesium variable increases by 1mg/dl, the risk of hypertension decreases by 90%. However, in the presence of magnesium as an independent variable, the predictability rate of our model for hypertensive patients decreased, while it was expected to increase [2]. In two meta-analysis studies involving seven and 11 studies, it has been suggested that magnesium supplementation provides a significant, albeit small, decrease in blood pressure [15,16]. In a meta-analysis in which thirty-four studies were evaluated, it was found that there was a decrease in both systolic and diastolic blood pressure [17]. A recent meta-analysis study claimed that oral magnesium supplementation does not affect blood pressure in normotensives and controlled hypertensives but reduces blood pressure in uncontrolled hypertensives [18]. In addition, there was no difference in magnesium levels between those with hypertensive crisis and the control group [19]. However, in light of these contradictory findings, considering that the number of participants in meta-analysis studies is high, it is a point that should not be overlooked that statistical significance will be high, even if there is no significance in reality.

In our study, it was found that when the value of the age variable increased by one unit, the risk of hypertension increased by 1.14 times. In our working model, the probability of predicting hypertensive patients in the presence of magnesium and age is relatively high. This result indicates that the age variable is an important risk factor. In the literature, age is recognized as a risk factor for hypertension [4]. Studies have been reported showing that magnesium decreases with age [20,21]. It has been suggested that this relationship may be related to decreased absorption with age and slower kidney reabsorption [22,23]. In addition, recent reports indicate that many Americans, especially older people, do not consume the recommended daily amount of magnesium [24]. In countries with high incomes, the rate of hypertension is lower than in poor countries [2]. Again, it is stated that an unhealthy diet causes hypertension [25]. All these findings and literature information suggest that the relationship between magnesium and hypertension may be an incidental association.

Our study found that the risk of hypertension decreased by 82% in those who exercised. In the literature, it is accepted that inactivity or lack of exercise is a risk factor for

hypertension [4]. It is stated that regular exercise causes a decrease in blood pressure of 5 mmHg [26]. In addition, emphasis has been placed on the curative effect of both aerobic and moderate-weight exercise on hypertension [27]. Aerobic exercise is assumed to positively affect hypertension by reducing oxidative stress [28]. A recent meta-analysis indicates that aerobic exercise is an effective adjunctive treatment method for lowering ambulatory blood pressure in hypertension patients receiving drug therapy [29].

Our study found no relationship between body mass index and hypertension. However, when the value of the glucose variable increased by one unit, the risk of hypertension increased 1.03 times, albeit very slightly. Obesity is accepted as a risk factor for hypertension in the literature [4,30]. Mechanisms of obesity causing hypertension are explained by the activation of the sympathetic nervous system activated by hormones such as leptin, insulin, melanocortin, and the activation of the renin-angiotensinogen system due to oxidative effects caused by proinflammatory cytokines released as a result of dyslipidemia [31]. Many pathophysiological mechanisms explain the relationship between diabetes and hypertension. These mechanisms are mainly hyperinsulinemia-mediated sympathetic activation, hyperglycemia-induced sodium-fluid retention, the stimulatory effect of hyperglycemia on the renin-angiotensin-aldosterone system, and increased insulin resistance as a result of oxidative stress [32,33].

An increased risk of type-2 diabetes and increased LDL-C levels were found in low magnesium [34]. It has been suggested that the positive effects of magnesium on diabetes occur by inhibiting oxidative stress [35]. It is also stated that the primary pathology in gestational hypertension in which magnesium sulfate is used in the treatment is oxidative stress [36]. The positive effects of regular and aerobic exercise on oxidative stress and its curative effects on diabetes and obesity have been emphasized [37]. Both magnesium deficiency and oxidative stress have been identified as pathogenic factors in aging and age-related diseases. Additionally, severe magnesium deficiency has been shown to increase oxidative stress in experimental animals [38]. In light of these findings, it seems complicated to define the relationship of magnesium with hypertension without fully elucidating the complex effects of oxidative stress on hypertension.

## CONCLUSIONS

As a result, it was found in our study that magnesium deficiency causes an increased risk of hypertension. However, in the presence of magnesium as an independent variable, the predictability rate of our model was expected to increase but decreased. These findings show that the relationship between magnesium and hypertension cannot be explained only by a cause-effect connection or incidental association. This may explain the reason for the conflicting results in the literature. More detailed analyzes are needed in this regard.

***Conflicts of interest and sources of funding***

*The authors declare no conflict of interest. The funders had no role in the design of the study; in the collection, analyses, or interpretation of data; in the writing of the manuscript; or in the decision to publish the results. This research received no external funding.*

***Authors' contribution***

*All authors made substantial contributions to the conception or design of the work or the acquisition, analysis, or interpretation of data for the work. All authors have read and agreed to the published version of the manuscript.*

***Patient consent for publication***

*Because of the retrospective study, patient consent was not obtained.*

***Ethics approval and consent to participate***

*Ethical approval was obtained from the İstanbul Training Research Hospital Clinical Research Ethics Committee (Decision no. 14.10.2022, 318).*

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