

ORIGINAL ARTICLE

The Relation between Serum Magnesium Level and Severity of Hypertension

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ABSTRACT

Background: Hypertension is a significant global health problem that is mainly related to cardiovascular diseases and cerebrovascular complications. Variations in the levels of risk factors for this condition, such as a diet high in salt and low in potassium, obesity, consumption of alcohol, lack of activity, and poor lifestyle habits, might be responsible for a portion of the regional heterogeneity in the prevalence of hypertension. This study assessed the relation between serum magnesium level and the severity of hypertension.

Methods: In a cross sectional study from 2nd October 2024 to the end of December 2024, 150 patients with hypertension from Al-Hakim General Hospital were enrolled. The number of antihypertensive drugs used for the treatment was considered as a marker for severity of hypertension. All participants underwent evaluation for coronary artery disease and stroke. The xylidyl blue method (magnesium liquicolor-human kit) was used to assess the level of serum magnesium in sera of the patients.

Results: Patients who needed a multiple antihypertensive medication had lower serum magnesium level and demonstrated a significant inverse relationship between serum magnesium level and the number of antihypertensive medications taken. Patients with managed and uncontrolled hypertension exhibited a significant difference in serum magnesium concentration, with those with uncontrolled blood pressure having a lower magnesium level. Serum magnesium level was significantly lower in obese patients than in normal-weight and overweight persons.

Conclusion: Hypertensive patients who needed multiple antihypertensive drugs showed a significantly lower serum magnesium level.

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Introduction

Gradually, almost all cardiovascular disorders, particularly valvular cardiac disease, coronary artery disease, left ventricular hypertrophy, and kidney damage, may be influenced by hypertension, a very recurrent and progressive condition with several contributing variables (1-3). In addition,

non-communicable disorders (NCDs) that include overweight, dyslipidemia, intolerance to glucose, and the metabolism syndrome have all been associated with increased blood pressure (4). About 30-35% decrease in stroke and 20% decrease in myocardial infarction (MI) are associated with the control of hypertension (5). The vast majority of the main

guidelines recommended diagnosing hypertension when a patient's systolic blood pressure (SBP) in the office or clinic is ≥ 140 mmHg and/or their diastolic blood pressure (DBP) is ≥ 90 mmHg based on multiple evaluations (6).

In contrast to office blood pressure level, ambulatory blood pressure monitoring (ABPM) data accurately represented the risk of cardiovascular events. Several patients with high blood pressure in the office had normal blood pressure levels on ABPM and a prognosis comparable to those who were normotensive in both circumstances, a phenomenon referred to as "white coat hypertension". For all of these reasons, the US Preventive Services Task Force suggested utilizing ABPM to confirm the diagnosis of hypertension in people with high office blood pressure prior to beginning medication therapy (7). A comprehensive neurohumoral system comprising the endothelium, natriuretic peptides, the sympathetic nervous system (SNS), and the renin-angiotensin-aldosterone system (RAAS) maintains the physiological blood pressure level. An either indirect or direct rise in mean blood pressure may result from a defect or fluctuation in these components, and over time, this may lead to damage to target organs and cardiovascular disease (CVD) consequences (8-10).

Excessive consumption of sodium in salt-sensitive people decreases bioavailable nitric oxide (NO) and raises transforming growth factor beta (TGF- β) production, elevating the risk of oxidative stress and fibrosis (11, 12). Moreover, blood pressure increases when constantly expressed endothelial NO synthase (eNOS) is diminished, resulting in decreased NO generation (13). More than 95% of hypertensive patients are without an apparent cause for hypertension; and these individuals are therefore specified to have essential hypertension (14). Magnesium's calcium antagonist characteristics and their consequences on endothelial cell function, arterial tone, vascular cell growth, calcification of arteries, oxidative stress, chronic inflammation, and glucose metabolism might all serve as contributing factors to the relationship that exists between magnesium and high blood pressure (15, 16).

Mg²⁺ interferes with the activity of the sympathetic nervous system, whereas having a shortage in Mg²⁺ enhances sympathetic tone. Mg²⁺ primarily impacts the sympathetic nervous system through modifying the action of N-methyl-D-aspartate (NMDA) receptors. The NMDA receptor is a Ca²⁺-selective ion channel that is triggered to open by both NMDA and L-glutamate (L-Glu). Activation of the NMDA receptor in the rostral ventrolateral medulla (RVLM) and hypothalamic

paraventricular nucleus (PVN) raises sympathetic output and arterial pressure. Therefore, it is believed that Mg²⁺'s inhibition of NMDA receptor activity could result in lower blood pressure (17-19).

Moreover, the body might turn more reactive to vasoconstrictors as angiotensin II and catecholamines when there is a magnesium shortage. Many hypertension patients undergo therapy with thiazides and loop diuretics, which deplete magnesium from the body. Whenever patients with hypertension consume thiazide diuretics for a longer period of time, administering oral magnesium supplements dramatically lowers their blood pressure. In fact, the increased intracellular calcium brought on by a magnesium deficit might contribute to insulin resistance and hypertension (20). Since lipid and lipoprotein abnormalities are modifiable risk factors for cardiovascular diseases due to their detrimental impact on atherosclerosis, magnesium significantly influences the metabolism of lipids by improving the activity of lecithin-cholesterol acyltransferase and lipoprotein lipase by modulating the activity of 3-hydroxy-3-methylglutaryl coenzyme A (HMG CoA) reductase (21).

Materials and Methods

The study was a cross sectional study enrolling 150 hypertensive patients with or without ischemic heart diseases and administered antihypertensive medications. About 102 of the participants were female and 48 were male aged 25-75 years. All patients were recruited from Al-Hakim General Hospital in Al-Najaf City, Iraq between 2nd October and the end of December 2024. The study was granted ethical approval from the Health Directorate in the province of Najaf and the Internal Ethical Committee of the Medical Physiology Department/ Faculty of Medicine at the University of Kufa, Kufa, Iraq (Reference #: MEC-93). Moreover, all patients who engaged in in this trial provided their informed consent as a voluntary agreement. Each patient underwent a thorough evaluation, which included a complete medical history, clinical examination, body mass index (BMI) calculation, blood pressure measurement and electrocardiogram (ECG). However, patients with chronic kidney diseases, inflammatory bowel diseases, chronic diarrhea, and pregnant women were excluded from this research endeavor.

Measurement of serum magnesium level was done by utilizing disposable syringes and needles, while three-milliliter venous blood samples were obtained from every participant and inserted into a gel tube. The sample went through a centrifuge for 4 minutes (4000 rounds per minute) in order to

separate the serum from the whole blood after have been left for five minutes. Following that, the serum was taken for biochemical analysis. Measuring the serum magnesium level of each participant was undertaken by employment of Magnesium Liquicolor kit (Human, Germany) by the xylidyl blue method with the Spectrophotometer V-1000 machine (AOELAB, China).

Data were entered and analyzed using SPSS software (version 26, Chicago, IL, USA). Descriptive statistics included frequencies and percentages for categorical variables, and means with standard deviations for continuous variables. The Shapiro-Wilk test assessed data normality. Independent t-tests were used to compare mean serum magnesium level with blood pressure control, comorbidities, smoking history and sex. ANOVA with post-hoc test was utilized to compare mean serum magnesium level with the number of antihypertensive drugs, age groups and BMI categories. A *p* value of ≤ 0.05 was

considered statistically significant (22).

Results

This study included 150 hypertensive patients aged 59.53 ± 10.4 years; while the female participants were 102 (68%) and males were 48 (32%) and the BMI was 30.9 ± 6.4 kg/m². About 33 (22%) patients were smokers, 56 (37.3%) had diabetes mellitus (DM), 60 (40%) suffered from ischemic heart disease (IHD) and 55 (36.7%) had cerebrovascular accident (CVA) (Table 1). Age, sex, and smoking status were not significantly correlated with mean serum magnesium level ($p > 0.05$). Conversely, as shown in Table 2, patients classified as obese had significantly lower mean serum magnesium level when compared to individuals who were overweight or of normal body weight ($p = 0.04$). As illustrated in Table 3, there was no statistically significant correlation between serum magnesium level and the occurrence of DM, IHD, or CVA ($p > 0.05$).

Table 1: Demographic and clinical characteristics of the patients (N=150).

Variable	Subgroup	No.	%
Age (years)	25-40	10	6.7
	41-60	66	44
	61-75	74	49.3
Sex	Female	102	68
	Male	48	32
BMI (Kg/m ²)	Normal	20	13.3
	Overweight	49	32.7
	Obese	81	54
DM	Yes	56	37.3
	No	94	62.7
IHD	Yes	60	40
	No	90	60
CVA	Yes	55	36.7
	No	95	63.3
Smoking	Yes	33	22
	No	117	78

BMI: Body mass index, CVA: Cerebrovascular accident, DM: Diabetes mellitus, IHD: Ischemic heart disease, SD: Standard deviation.

Table 2: The effect of age, sex, BMI, and smoking on mean serum magnesium level.

Variable	Subgroup	Serum Mg (mg/dL) (Mean $\pm$ SD)	P value
Age (year)	25-40	2.124 $\pm$ 0.4	0.6
	41-60	2.034 $\pm$ 0.3	
	61-75	2.004 $\pm$ 0.4	
Sex	Female	1.991 $\pm$ 0.4	0.1
	Male	2.095 $\pm$ 0.4	
BMI (Kg/m ²)	Normal	2.107 $\pm$ 0.4	0.04*
	Overweight	2.109 $\pm$ 0.4	
	Obese	1.955 $\pm$ 0.3	
Smoking	Yes	2.003 $\pm$ 0.4	0.7
	No	2.031 $\pm$ 0.4	

BMI: Body mass index, SD: Standard deviation.

Table 3: Comparison between mean serum magnesium level and associated comorbid conditions.

Variable	Subgroup	No.	Serum Mg (mg/dL) (Mean±SD)	P value
DM	Yes	56	2.058±0.4	0.4
	No	94	2.005±0.4	
IHD	Yes	60	1.976±0.4	0.2
	No	90	2.058±0.4	
CVA	Yes	55	2.0004±0.4	0.5
	No	95	2.039±0.4	

CVA: Cerebrovascular accident, DM: Diabetes mellitus, IHD: Ischemic heart disease, SD: Standard deviation.

As shown in Figure 1, the mean serum magnesium level was significantly lower in patients receiving much more antihypertensive drugs. As consumption of antihypertensive medications increased, blood magnesium gradually decreased, thus suggesting an inverse relationship between serum magnesium concentration and the severity of hypertension ($p<0.05$). The mean level of serum magnesium among patients with uncontrolled hypertension was substantially lower than those of patients with controlled hypertension, demonstrating a causal association between poor blood pressure control and lower serum magnesium concentration ($p<0.05$) (Figure 2).

Discussion

Mg²⁺ has protective effects on the circulatory system and protects against arrhythmia and ischemia. Multiple factors lead to the anti-ischemic benefits. It interacts with Ca²⁺ binding sites in order to avoid Ca²⁺ overload and its vasodilatory effects. It lowers catecholamine-induced demand for oxygen, heartbeat rate, and contractility. Furthermore, it acts as an antioxidant and modulates ATP-dependent mechanisms that prevent future coronary artery disease (23). According to research by Cameron *et al.*, average serum magnesium level tended to remain stable as people aged, while intracellular magnesium level, particularly those found in

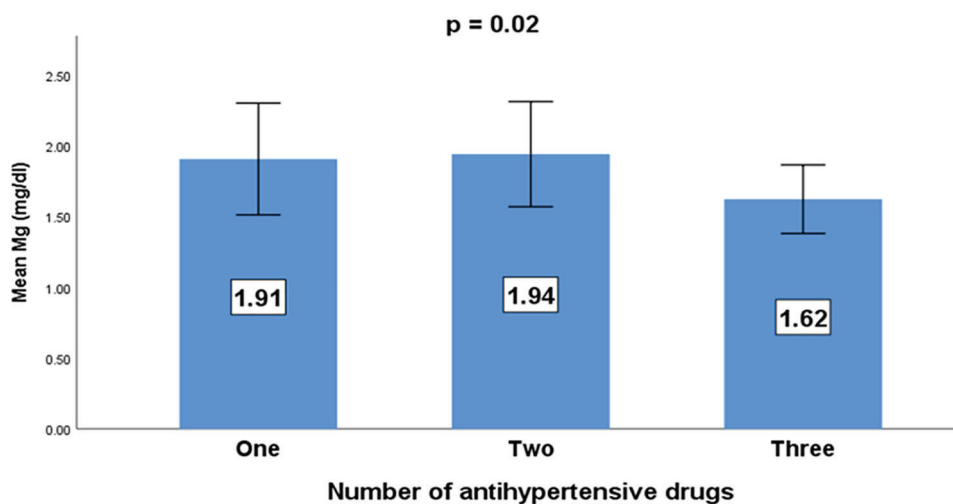


Figure 1: The relation between mean serum magnesium level and number of antihypertensive drugs.

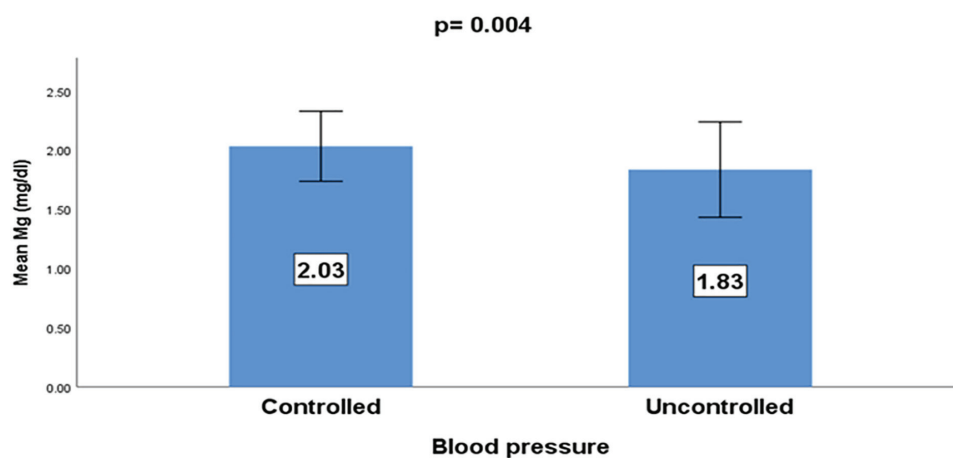


Figure 2: The effect of mean serum magnesium level on the control of hypertension.

muscle and red blood cells that continued to decline. This suggests that whereas cell magnesium level varies significantly by age, serum magnesium concentration appears to be unaffected (24).

A positive correlation was noticed between low dietary Mg^{2+} intake and metabolic risk, regardless of other potential risk factors like gender, age, BMI, ethnicity, level of education, and smoking (25). In accordance with Nielsen's findings, obesity could stimulate pro-inflammatory cytokines including tumor necrosis factor- α (TNF- α) and interleukin-6 (IL-6), and this may have a direct effect on magnesium balance. These markers of inflammation may be a factor in magnesium shortage because they promote urine excretion and decrease magnesium cell retention. When investigating if magnesium and chronic low-grade inflammation are associated, being overweight or obese could play a significant role (26).

Obese people acquire the majority of their bodily energy from carbohydrates that are refined and simple sugars, and as a consequence, their catabolism in liver is extremely robust. Many major enzymes in glucose oxidation pathways require Mg^{2+} , while Mg^{2+} is additionally needed for the conversion of vitamin B1 into thiamine diphosphate (TDP), a further vital coenzyme in oxidative metabolism. Reduced intracellular concentrations of Mg^{2+} and/or TDP could alter the oxidative metabolism of glucose (27). The National Health and Nutrition Examination Survey-3 (NHANES-3) study emphasized that Mg^{2+} deficiency is significantly greater in individuals with a BMI in the obese range rather than in the typical American population (28). Meanwhile, the research results agree with Rebat *et al.* suggesting no noticeable effect for smoking on serum magnesium level (29).

Hypomagnesemia was associated with further aggravated hypertension in an observational study of 1,000 ambulatory hypertensive patients, as demonstrated by a higher number of antihypertensive prescribed medications (30). Clinicians must deliver medications that minimize urinary Mg^{2+} -wasting with a low threshold, including Sodium-glucose cotransporter-2 (SGLT-2) inhibitors and K^+ and Mg^{2+} -sparing diuretics (for example, amiloride or spironolactone) (31). As reported by Frumuzachi *et al.*, whenever patients with stage 1 and stage 2 hypertension received an oral magnesium supplement to their usual antihypertensive drugs, their blood pressure decreased. For patients whose conventional medical therapy was incapable of providing optimal management of blood pressure, prescribing magnesium supplement may be an effective approach to lower blood pressure without

prescribing additional antihypertensive medications, increasing the risk of associated consequences (32).

Based on an investigation by Gonuguntla *et al.*, insulin resistance and renal magnesium insufficiency via glycosuria were the primary determinants of magnesium deficiency in diabetic patients. Despite having a high probability of hypomagnesemia in individuals with diabetes; magnesium level has not demonstrated any discernible correlation (33). Because of processing of food and refinement, the western diet is exceptionally often deficient in magnesium, and hypomagnesemia in hospitalized individuals is frequently incorrectly diagnosed (34, 35). A further research assessed the relationship among patients with high serum magnesium level, osteoporosis, and cardiometabolic risks that were type 2 diabetes mellitus (T2DM), coronary artery disease, atrial fibrillation (AF), and heart failure. The investigation revealed no connection between cardiometabolic risks and serum magnesium level; while an obvious causal connection was discovered in the incident of osteoporosis (36). Also utilizing intravenous magnesium supplementation as a therapy for acute heart failure might not be beneficial to patients or enhance patient care (37).

The findings of Zhang *et al.* indicated that a dose of 368 mg per day for 12 weeks was effective for lowering DBP by 1.78 mmHg and SBP by 2 mmHg (38). Particularly, only people who were already taking antihypertensive medications exhibited substantial reductions in this trial. Another research investigation by Rosanoff and Plesset included hypertension participants with a mean baseline SBP of more than 155 mmHg and utilized antihypertensive drugs regularly for at least six months. In their study, the investigators showed that magnesium supplementation significantly decreased blood pressure, with mean reductions in mmHg for systolic and diastolic blood pressure of 18.7 and 10.9 mmHg, respectively (39).

There was a limitation in this study. First, it was not possible to demonstrate a causal association between the severity of hypertension and serum magnesium level owing to its cross-sectional design. Second, the results may not be considered relevant to other groups because the study was conducted at one specific site with a relatively small sample size.

Conclusion

The findings of this study revealed the requirement to use multiple antihypertensive drugs and indicated a significant connection between lower serum magnesium levels and the presence of more severe hypertension. Obese hypertension patients were shown to have a significantly decreased serum

magnesium level compared to non-obese individuals highlighting the likelihood that magnesium can play a part in the pathogenesis of hypertension and may provide evidence that lower blood magnesium levels can possibly be linked to more challenging control of hypertension. A routine magnesium supplementation is not recommended; however, after following a suitable clinical examination, measuring blood magnesium is potentially beneficial in certain hypertension individuals.

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Authors' Contribution

Each author contributed equally to this study.

Conflict of Interest

The authors clearly state that they actually possess no conflicts of interest.

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