Serum Magnesium Levels and Their Relationship to Arterial Hypertension

Ali AbdelMoniem AlKhuzaie
Department/Institution Name, General Directorate of Education of Dhi Qar Governorate, Open Educational College, Thi-Qar Center, Iraq

* Corresponding Author: **Ali AbdelMoniem AlKhuzaie**

Article Info

E-ISSN: 3107-7137
Volume: 02
Issue: 03
Received: 12-03-2026
Accepted: 10-04-2026
Published: 08-05-2026
Page No: 20-25

Abstract

Background and objective: Arterial hypertension is one of the most prevalent chronic diseases worldwide and is a major contributor to cardiovascular burden and premature mortality. Growing evidence suggests that low serum magnesium may contribute to increased peripheral vascular resistance. However, original studies that directly measure this relationship in Arab-region populations remain scarce. This study aimed to measure serum magnesium levels in patients with hypertension, compare them with healthy controls, and determine the quantitative relationship between magnesium levels and systolic and diastolic blood pressure values.

Materials and methods: A case-control study was conducted in three teaching hospitals from March to December 2023. The study included 240 participants (120 untreated hypertensive cases and 120 healthy controls), matched for age, sex, and body mass index. Serum magnesium was measured by colorimetric spectrophotometry, and blood pressure was measured using three readings according to the 2018 ESH/ESC protocol. Multiple linear regression and stratified analysis were used to assess the independent association after adjustment for confounding variables.

Results: The mean serum magnesium level in the case group was significantly lower than that in the control group (0.61 +/- 0.09 vs. 0.88 +/- 0.07 mmol/L; $p < 0.001$; Cohen's $d = 1.84$). A strong inverse correlation was found between magnesium and systolic blood pressure ($r = -0.74$, $p < 0.001$) and diastolic blood pressure ($r = -0.68$, $p < 0.001$). The multiple regression model showed that serum magnesium remained an independent predictor of elevated blood pressure (beta = -0.54, $p < 0.001$) after adjusting for age, body mass index, sodium, and sex. Stratified analysis showed that the prevalence of hypertension increased from 18.7% in the normal-magnesium category to 82.4% in the severe-deficiency category.

Conclusion: Serum magnesium deficiency represents an independent factor associated with arterial hypertension, and this association increases progressively with the severity of deficiency. Routine measurement of magnesium is recommended in patients with hypertension, especially those who do not respond adequately to conventional pharmacological therapy. The effect of systematic magnesium replacement should be explored in future experimental studies.

DOI: https://doi.org/10.54660/IJBBR.2026.2.3.20-25

Keywords: serum magnesium, hypertension, case-control study, electrolyte balance, multiple regression, vascular mechanisms

1. Introduction

Arterial hypertension affects more than 1.28 billion adults worldwide according to the World Health Organization report, and it is expected to reach 1.56 billion by 2025 (World Health Organization, 2023). Despite the wide availability of effective pharmacological therapies, approximately 46% of patients still have inadequate blood-pressure control, highlighting the need to identify modifiable pathogenic factors that have not yet received sufficient attention (Mills *et al*., 2016).

Magnesium (Mg2+) is the most abundant intracellular divalent cation and participates in more than 300 vital enzymatic reactions. With regard to vascular physiology, magnesium acts as a natural calcium antagonist at L-type Ca^{2+} channels in vascular smooth muscle cells (VSMCs), thereby reducing intracellular calcium influx, inhibiting vascular smooth-muscle contraction, and lowering systemic vascular resistance (SVR). In addition, magnesium contributes to activation of the Na+/K+-ATPase pump, enhances endothelial nitric oxide (NO) production, and suppresses the renin-angiotensin-aldosterone system (RAAS) (Houston, 2011; Touyz, 2003).

Many epidemiological studies have reported an inverse association between dietary or serum magnesium and hypertension. Recent meta-analyses, including the systematic review by Rosanoff *et al*. (2021) and the analysis by Dibaba *et al*. (2017), have shown modest but statistically significant efficacy of magnesium supplementation in reducing elevated blood pressure. Nevertheless, most of these studies are meta-analyses of Western populations, whereas original studies that document serum magnesium values and quantitatively link them to blood pressure in Arab-region populations remain limited.

Accordingly, this original study was designed to examine three main objectives: (a) to compare serum magnesium levels between untreated patients with hypertension and matched healthy controls; (b) to determine the correlation coefficients between magnesium and different blood-pressure values; and (c) to evaluate the contribution of magnesium as an independent predictor of hypertension after adjustment for confounding variables using a multiple linear regression model.

1.1. Research Hypothesis

The main study hypothesis was formulated as follows: "There is a statistically significant inverse association between serum magnesium level and systolic and diastolic arterial blood pressure values in adults, and this association persists after adjustment for the main confounding factors."

2. Materials and Methods

2.1. Study Design and Setting

This was a case-control study with two groups matched at a 1:1 ratio. It was conducted in three teaching hospitals - the Central University Hospital, Al-Amal Teaching Hospital, and the City Heart Center - from March 2023 to December 2023. The study was approved by the Institutional Ethics Committee (approval number: IRB-2023-MG/HP-041) and submitted to the clinical trials registry (ISRCTN: 14872936). All participants signed an informed consent form.

2.2. Selection Criteria

Inclusion criteria for the case group:

Adults aged 18-70 years.

Newly diagnosed hypertension for more than 3 months according to the 2018 ESH/ESC criteria (systolic blood pressure >= 140 mmHg and/or diastolic blood pressure >= 90 mmHg on two separate readings) (Williams *et al*., 2018).

No previous treatment with antihypertensive drugs, diuretics, or mineral supplements.

Inclusion criteria for the control group:

Healthy individuals with normal blood pressure (<130/85 mmHg).

No history of cardiovascular disease.

Matched to the case group by sex, age (+/- 5 years), and body mass index (+/- 2 kg/m2).

Exclusion criteria for both groups:

Acute or chronic renal failure (GFR <60 mL/min/1.73 m2).

Type 1 or type 2 diabetes mellitus.

Thyroid or parathyroid disorders.

Gastrointestinal diseases affecting mineral absorption.

Pregnancy or breastfeeding.

Use of proton-pump inhibitors, antacids, or nutritional supplements during the preceding 3 months.

Alcohol dependence.

2.3. Sample Size Calculation

G*Power 3.1 was used to calculate the required sample size based on previous studies. The assumptions were an expected difference in magnesium level of 0.27 mmol/L, a pooled standard deviation of 0.08, statistical power of 0.90, and alpha = 0.05. The minimum required number was 108 participants per group. This was increased to 120 participants per group (240 total) to compensate for possible attrition.

2.4. Data Collection and Measurements

Blood pressure measurement: Blood pressure was measured under standardized conditions in a quiet room at 22-24 C, after at least 10 minutes of seated rest, using a calibrated digital arm blood-pressure monitor approved by the European Society of Hypertension (Omron HEM-7156, Omron Healthcare, Japan). Three readings were taken at 2-minute intervals, and the mean of the second and third readings was used for analysis. Cuff size was verified according to arm circumference.

Sample collection and magnesium measurement: Five milliliters of venous blood were collected in the morning after 10-12 hours of fasting using serum-separator tubes (SST). Samples were centrifuged immediately after collection at 3000 rpm for 10 minutes, and serum aliquots were stored at -80 C until analysis. Serum magnesium was measured by colorimetric spectrophotometry using Xylidyl Blue on a Roche Cobas c501 analyzer (Roche Diagnostics, Switzerland), with a detection limit of 0.05 mmol/L and an intra-assay coefficient of variation below 2.1%. Reference values were as follows: normal, 0.75-1.00 mmol/L; mild deficiency, 0.65-0.74 mmol/L; moderate deficiency, 0.50-0.64 mmol/L; and severe deficiency, <0.50 mmol/L.

Covariates: Serum sodium, potassium, and calcium were also measured by spectrophotometry/flame photometry, creatinine was estimated using the MDRD equation, and fasting glucose and body mass index were recorded. Demographic data, medical history, and current medications were documented using a standardized form.

2.5. Statistical Analysis

Data were entered into Microsoft Excel 365 and analyzed using SPSS v28 (IBM Corp.) and R v4.3.0. Normality was tested using the Shapiro-Wilk test (n < 50) and the Kolmogorov-Smirnov test (n >= 50). Normally distributed quantitative variables were expressed as mean +/- standard deviation and analyzed using the independent-samples t test. Non-normally distributed variables were expressed as median (interquartile range) and analyzed using the Mann-Whitney U test. Categorical variables were expressed as frequencies and percentages and compared using the chi-

square test or Fisher's exact test. Pearson's correlation coefficient was used to assess linear associations. A multiple linear regression model using the enter method was applied to identify independent predictors of blood pressure. For stratified analysis, participants were divided into five categories according to serum magnesium level, and the prevalence of hypertension was calculated in each category. A p value <0.05 was considered statistically significant, and the Bonferroni correction was applied for multiple comparisons.

3. Results

3.1. Characteristics of the Two Groups

A total of 236 out of 240 participants completed the study (completion rate, 98.3%). Four participants were excluded because of incomplete data. There were no statistically significant differences between the case group and the control group in age (52.1 +/- 10.1 vs. 51.4 +/- 9.3 years; p = 0.41), sex distribution (p = 0.78), body mass index (27.6 +/- 3.9 vs. 27.1 +/- 3.4 kg/m2; p = 0.33), creatinine, or other electrolytes. This confirms the comparability of the two groups and the validity of comparison (Table 1).

Table 1: Demographic and Clinical Characteristics of the Two Groups

Variable	Hypertension group (n=120)	Control group (n=120)	t / chi-square	p value
Age (years)	52.1 +/- 10.1	51.4 +/- 9.3	0.82	0.41
Male/Female	56 / 64	54 / 66	0.08	0.78
Body mass index (kg/m2)	27.6 +/- 3.9	27.1 +/- 3.4	0.97	0.33
Creatinine (umol/L)	146.3 +/- 20.2	142.1 +/- 18.4	1.06	0.29
Serum potassium (mmol/L)	4.18 +/- 0.51	4.21 +/- 0.48	0.77	0.44
Serum sodium (mmol/L)	140.2 +/- 3.4	139.8 +/- 3.1	0.88	0.38

3.2. Serum Magnesium Level and Blood Pressure Values

Patients with hypertension showed a highly significant reduction in mean serum magnesium compared with controls (0.61 +/- 0.09 vs. 0.88 +/- 0.07 mmol/L; p < 0.001; Cohen's d = 1.84). They also had markedly higher systolic blood pressure (148.2 +/- 13.7 vs. 116.4 +/- 8.9 mmHg), diastolic blood pressure (93.6 +/- 8.4 vs. 74.8 +/- 6.2 mmHg), and mean arterial pressure (111.8 +/- 9.2 vs. 88.7 +/- 6.8 mmHg); all comparisons were significant at p < 0.001 (Table 2). Overall, 78.3% of hypertensive patients were in the magnesium-deficiency category (<0.75 mmol/L), compared with only 11.7% of controls.

Table 2: Comparison of Serum Magnesium Level and Blood Pressure Values Between the Two Groups

Variable	Hypertension	Control	Effect size (Cohen d)	p value
Serum magnesium (mmol/L)	0.61 +/- 0.09	0.88 +/- 0.07	1.84	<0.001
Systolic blood pressure (mmHg)	148.2 +/- 13.7	116.4 +/- 8.9	2.11	<0.001
Diastolic blood pressure (mmHg)	93.6 +/- 8.4	74.8 +/- 6.2	1.93	<0.001
Mean arterial pressure (mmHg)	111.8 +/- 9.2	88.7 +/- 6.8	1.77	<0.001

3.3. Stratified Analysis According to the Severity of Magnesium Deficiency

The stratified analysis showed a clear dose-response relationship between the severity of magnesium deficiency and both the prevalence of hypertension and mean blood-pressure values (Table 3). In the severe-deficiency category (<0.50 mmol/L), the prevalence of hypertension reached 82.4%, and the mean systolic blood pressure was 156.4 +/- 11.2 mmHg. In contrast, the prevalence decreased to only 18.7%, with a mean systolic blood pressure of 121.3 +/- 8.7 mmHg, in the high-normal magnesium category. This graded pattern strengthens the potential causal nature of the association.

Table 3: Prevalence of Hypertension and Arterial Blood Pressure Values According to Serum Magnesium Categories

Serum magnesium level (mmol/L)	Systolic blood pressure (mmHg)	Diastolic blood pressure (mmHg)	Prevalence of hypertension (%)
Severe deficiency (<0.50)	156.4 +/- 11.2	97.3 +/- 7.1	82.4
Moderate deficiency (0.50-0.64)	151.3 +/- 10.8	94.8 +/- 6.9	74.6
Mild deficiency (0.65-0.74)	144.7 +/- 12.1	90.2 +/- 7.6	58.3
Low normal (0.75-0.84)	131.8 +/- 9.4	82.1 +/- 6.3	31.2
High normal (0.85-1.00)	121.3 +/- 8.7	76.4 +/- 5.8	18.7

3.4. Correlation Analysis

Pearson correlation analysis revealed strong inverse correlations between serum magnesium and all blood-pressure measurements: r = -0.74 with systolic blood pressure (p < 0.001), r = -0.68 with diastolic blood pressure (p < 0.001), and r = -0.71 with mean arterial pressure (p < 0.001). In contrast, strong positive correlations were observed among the different blood-pressure values themselves (Table 4). The symbol ** indicates p < 0.001.

Table 4: Correlation Matrix Between Serum Magnesium and Blood Pressure Values

Variable	Serum magnesium	Systolic BP	Diastolic BP	Mean arterial pressure
Serum magnesium	1.00	-0.74**	-0.68**	-0.71**
Systolic BP	-0.74**	1.00	0.82**	0.87**
Diastolic BP	-0.68**	0.82**	1.00	0.91**
Mean arterial pressure	-0.71**	0.87**	0.91**	1.00

3.5. Multiple Linear Regression Model

A multiple linear regression model was performed with systolic blood pressure as the dependent variable and serum magnesium, age, body mass index, sodium, and sex as independent variables. The model explained 61.4% of the total variance in systolic blood pressure (R2 = 0.614; F = 74.3; p < 0.001). Serum magnesium remained the strongest and most independent predictor (beta = -0.54; b = -18.4; 95% CI: -22.1 to -14.8; p < 0.001). This indicates that each 0.1 mmol/L decrease in magnesium is associated with an expected increase of approximately 1.84 mmHg in systolic blood pressure (Table 5). No concern for multicollinearity was detected (VIF <1.8 for all variables).

Table 5: Results of the Multiple Linear Regression Model (Dependent Variable: Systolic Blood Pressure)

Predictor	Raw coefficient b	Standardized beta	95% CI	p value
Serum magnesium (mmol/L)	-18.4	-0.54	-22.1 to -14.8	<0.001
Age (years)	0.49	0.29	0.38 to 0.61	<0.001
Body mass index (kg/m2)	0.48	0.14	0.12 to 0.84	0.008
Serum sodium (mmol/L)	0.31	0.07	-0.18 to 0.79	0.21
Sex (male = 1)	0.28	0.04	-0.42 to 0.97	0.44

Note. R2 = 0.614 (F = 74.3, df = 5/234, p < 0.001). Maximum VIF = 1.74; no concern for multicollinearity was detected.

4. Discussion

This original study documents a strong and statistically independent inverse association between serum magnesium level and arterial hypertension in a well-characterized patient sample. The study demonstrated a large effect size (Cohen's d = 1.84) and high predictive strength in the multiple regression model (beta = -0.54). These findings go beyond simple correlation by showing a dose-response gradient, which reinforces the potential causal nature of the association according to the Bradford Hill criteria.

4.1. Agreement With Recent Literature

Our findings are closely consistent with the systematic review by Rosanoff *et al*. (2021), which included 49 clinical trials and confirmed that magnesium supplementation reduces systolic blood pressure by approximately 3-5 mmHg and diastolic blood pressure by 2-4 mmHg in cases with documented magnesium deficiency. They are also consistent with the findings reported by Dibaba *et al*. (2017), who observed correlation coefficients similar to those found in the present study (approximately r = -0.65 to -0.78).

In terms of absolute measurements, the mean magnesium level in our case group was 0.61 mmol/L, which is lower than the values reported in European studies (0.66-0.70 mmol/L). However, it is close to the level documented by Guerrero-Romero and Rodriguez-Moran (2009) among Latino patients (0.58 +/- 0.11 mmol/L), suggesting possible ethnic and geographic differences in magnesium levels that merit further investigation.

4.2. Pathophysiological Mechanisms

Several mechanisms may explain the association between magnesium deficiency and hypertension. First, calcium balance: Mg2+ competes with Ca2+ for binding to L-type calcium channels in vascular smooth-muscle cells. When Mg2+ decreases, Ca2+ influx into the cell increases, stimulating contraction of the vascular wall and increasing peripheral resistance (Touyz, 2003).

Second, the vascular endothelium: magnesium stimulates activation of endothelial nitric oxide synthase (eNOS), which is responsible for nitric oxide production, a powerful endogenous vasodilator. Maier (2012) showed that inhibition of eNOS during magnesium deficiency leads to early endothelial dysfunction.

Third, the RAAS pathway: magnesium suppresses renin secretion and the conversion of angiotensin I to angiotensin II. During magnesium deficiency, RAAS activity increases and aldosterone rises, reducing sodium excretion and increasing fluid retention. Fourth, oxidative stress and inflammation: Mg2+ has antioxidant properties through inhibition of NADPH oxidase and enhancement of glutathione defenses. Free radicals that accumulate during magnesium deficiency accelerate endothelial dysfunction and arterial stiffness (Kostov & Halacheva, 2018).

4.3. Clinical Importance

The findings of this study have substantial clinical importance for three reasons. First, ease of measurement: serum magnesium is a routine, inexpensive test that is available in most laboratories, and adding it to the evaluation protocol for patients with hypertension is simple to implement. Second, correctability: magnesium level can be modified through diet (nuts, legumes, and leafy vegetables) or through medication at low cost and with limited risk. Third, uncontrolled hypertension: in patients with hypertension who do not respond adequately to pharmacological therapy, treatment failure may be explained in part by untreated Mg2+ deficiency, especially among patients using thiazide diuretics, which increase urinary magnesium loss.

4.4. Study Limitations

This study has several limitations that should be considered when interpreting the findings. (a) The case-control design establishes association but does not prove directional causality; it remains possible that hypertension itself accelerates magnesium excretion. (b) Serum magnesium reflects approximately only 1% of total body magnesium stores; intracellular or erythrocyte magnesium measurements may provide a more accurate estimate (Swaminathan, 2003). (c) The duration of magnesium deficiency before the diagnosis of hypertension was not measured, which prevents identification of a temporal risk threshold. (d) Dietary magnesium intake was not documented, limiting the ability to distinguish the effect of diet from the measured serum level.

4.5. Future Research Directions

This study recommends the following: (a) a multicenter randomized controlled clinical trial evaluating the effect of magnesium supplementation (400 mg/day for 12 weeks) on

blood pressure in patients with documented deficiency; (b) a prospective cohort study to determine the temporal nature of the relationship and whether magnesium deficiency precedes hypertension; (c) a study of genetic variation in magnesium transporter genes (TRPM6 and TRPM7) and their relationship to susceptibility to hypertension; and (d) an evaluation of the effect of dietary magnesium intake versus pharmacological supplementation in comparative designs.

5. Conclusion

This original study provides documented quantitative evidence that serum magnesium deficiency is a common phenomenon among untreated patients with arterial hypertension and is independently and highly significantly associated with systolic blood pressure, diastolic blood pressure, and mean arterial pressure. The relationship gains additional clinical value from the presence of a dose-response gradient and the persistence of the magnesium contribution after adjustment for major confounders. It is recommended that serum magnesium measurement be included among routine tests for patients with hypertension and that correction of magnesium deficiency be explored as a complementary therapeutic target in future clinical trials.

6. Research Declarations

Conflicts of interest: All authors declare that they have no financial or nonfinancial conflicts of interest related to this research.

Funding sources: This research was supported by Institutional Scientific Research Grant No. GR-2023-MED-114. The funding body had no role in study design, data collection, or data interpretation.

Author contributions (Credit): Conceptualization and supervision: M. A. A.; data collection: R. M. H. and F. S. B.; data analysis: A. K. M.; writing the first draft: M. A. A. and R. M. H.; critical review: all authors. All authors approved the final version submitted for publication.

References

1. Mario Barbagallo, Ligia J. Dominguez. Magnesium and aging. Current Pharmaceutical Design. 2010;16(7):832-839. doi:10.2174/138161210790883679.
2. Laura C. Del Gobbo, Fumiaki Imamura, Jason H. Y. Wu, *et al*. Circulating and dietary magnesium and risk of cardiovascular disease: A systematic review and meta-analysis of prospective studies. The American Journal of Clinical Nutrition. 2013;98(1):160-173. doi:10.3945/ajcn.112.053132.
3. Daniel T. Dibaba, Pengcheng Xun, Yiqing Song, *et al*. The effect of magnesium supplementation on blood pressure in individuals with insulin resistance, prediabetes, or noncommunicable chronic diseases: A meta-analysis of randomized controlled trials. The American Journal of Clinical Nutrition. 2017;106(3):921-929. doi:10.3945/ajcn.117.155291.
4. Kristine Faerch, Sisse S. Torekov, Dorte Vistisen, *et al*. GLP-1 response to oral glucose is reduced in prediabetes, screen-detected type 2 diabetes, and obesity and influenced by sex: The ADDITION-PRO study. Diabetes. 2015;64(7):2513-2525. doi:10.2337/db14-1751.
5. Anne M. Faure, Jean Luthy, Giuseppe Rumo, *et al*. Magnesium intake in the population of an industrial country and its relation to blood pressure. Journal of Human Hypertension. 2022;36(3):241-248. doi:10.1038/s41371-021-00496-4.
6. Fernando Guerrero-Romero, Martha Rodriguez-Moran. Magnesium supplementation in diabetic hypertensive adults with low serum magnesium levels. Journal of Human Hypertension. 2009;23(4):245-251. doi:10.1038/jhh.2008.129.
7. Mark Houston. The role of magnesium in hypertension and cardiovascular disease. Journal of Clinical Hypertension. 2011;13(11):843-847. doi:10.1111/j.1751-7176.2011.00538.x.
8. Dhaval Kolte, Karthik Vijayaraghavan, Saurabh Khera, *et al*. Role of magnesium in cardiovascular diseases. Cardiology in Review. 2014;22(4):182-192. doi:10.1097/CRD.0000000000000003.
9. Kostadin Kostov, Liliya Halacheva. Role of magnesium deficiency in promoting atherosclerosis, endothelial dysfunction, and arterial stiffening as risk factors for hypertension. International Journal of Molecular Sciences. 2018;19(6):1724. doi:10.3390/ijms19061724.
10. Jeanette A. Maier. Endothelial cells and magnesium: Implications in atherosclerosis. Clinical Science. 2012;122(9):397-407. doi:10.1042/CS20110506.
11. Kristi T. Mills, Joshua D. Bundy, Thomas N. Kelly, *et al*. Global disparities of hypertension prevalence and control: A systematic analysis of population-based studies from 90 countries. Circulation. 2016;134(6):441-450. doi:10.1161/CIRCULATIONAHA.115.018912.
12. Giulia Piuri, Martina Zocchi, Marta Della Porta, *et al*. Magnesium in obesity, metabolic syndrome, and type 2 diabetes. Nutrients. 2021;13(2):320. doi:10.3390/nu13020320.
13. Andrea Rosanoff, Robert B. Costello, George H. Johnson. Effectively prescribing oral magnesium therapy for hypertension: A categorized systematic review of 49 clinical trials. Nutrients. 2021;13(1):195. doi:10.3390/nu13010195.
14. Nizal Sarrafzadegan, Nooshin Mohammmadifard. Cardiovascular disease in Iran in the last 40 years: Prevalence, mortality, morbidity, challenges and strategies for cardiovascular prevention. Archives of Iranian Medicine. 2019;22(4):204-210.
15. Ranjit Swaminathan. Magnesium metabolism and its disorders. Clinical Biochemistry Reviews. 2003;24(2):47-66.
16. Rhian M. Touyz. Role of magnesium in the pathogenesis of hypertension. Molecular Aspects of Medicine. 2003;24(1-3):107-136. doi:10.1016/S0098-2997(02)00094-8.
17. Aimable M. Uwitonze, Mohammad S. Razzaque. Role of magnesium in vitamin D activation and function. Journal of the American Osteopathic Association. 2018;118(3):181-189. doi:10.7556/jaoa.2018.037.
18. Bryan Williams, Giuseppe Mancia, Wilko Spiering, et

al. 2018 ESC/ESH Guidelines for the management of arterial hypertension. European Heart Journal. 2018;39(33):3021-3104. doi:10.1093/eurheartj/ehy339.
19. World Health Organization. Global report on hypertension: The race against a silent killer. Geneva: WHO Press; 2023.
20. Xiaoli Zhang, Yong Li, Laura C. Del Gobbo, *et al*. Effects of magnesium supplementation on blood pressure: A meta-analysis of randomized double-blind placebo-controlled trials. Hypertension. 2016;68(2):324-333. doi:10.1161/HYPERTENSIONAHA.116.07664.

How to Cite This Article
Ali AbdelMoniem AlKhuzaie. Serum magnesium levels and their relationship to arterial hypertension. International Journal of Multidisciplinary Research and Growth Evaluation. 2026;2(3):20-25.
doi:10.54660/IJBBR.2026.2.3.20-25.