



SERUM MAGNESIUM LEVELS IN HYPERTENSIVE PATIENTS: CORRELATION WITH BLOOD PRESSURE SEVERITY

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ABSTRACT

Introduction: Hypertension remains a major global health challenge, with magnesium deficiency emerging as a significant modifiable risk factor. Magnesium influences blood pressure through multiple mechanisms including calcium channel antagonism and nitric oxide production. This study aimed to evaluate serum magnesium levels in hypertensive patients and assess their correlation with blood pressure severity.

Methods: A cross-sectional observational study was conducted at Autonomous State Medical College, Sonebhadra, Uttar Pradesh, from November 2024 to May 2025. A total of 150 patients with essential hypertension were enrolled through consecutive sampling. Participants were classified into Stage 1 and Stage 2 hypertension based on JNC 7 criteria. Serum magnesium levels were estimated using the calmagite dye-binding method. Blood pressure was measured using standardized protocols. Statistical analysis included Pearson's correlation, independent t-test, chi-square test, and multiple linear regression.

Results: The mean age of participants was 54.3 ± 10.8 years with male predominance (58.0%). Stage 2 hypertensive patients had significantly lower serum magnesium levels (1.64 ± 0.38 mg/dL) compared to Stage 1 (1.92 ± 0.34 mg/dL, $p < 0.001$). Overall prevalence of hypomagnesemia was 40.0%, significantly higher in Stage 2 (52.9%) versus Stage 1 (29.3%, $p = 0.002$). Serum magnesium showed moderate negative correlation with systolic blood pressure ($r = -0.542$, $p < 0.001$) and diastolic blood pressure ($r = -0.486$, $p < 0.001$). Multiple regression analysis revealed serum magnesium as an independent predictor of systolic blood pressure ($\beta = -0.412$, $p < 0.001$).

Conclusion: Serum magnesium levels are inversely correlated with hypertension severity. The high prevalence of hypomagnesemia in hypertensive patients, particularly those with Stage 2 hypertension, suggests the need for routine magnesium screening and consideration of magnesium supplementation in comprehensive hypertension management.

Keywords: Serum magnesium, hypertension, blood pressure severity, hypomagnesemia, cardiovascular risk factors

INTRODUCTION

Hypertension represents one of the most prevalent non-communicable diseases worldwide and stands as a major modifiable risk factor for cardiovascular morbidity and mortality. According to global estimates, hypertension affects approximately 1.28 billion adults worldwide, with the burden disproportionately affecting low and middle-income countries including India (Mills et al., 2020). The pathophysiology of hypertension is multifactorial, involving complex interactions between genetic predisposition, environmental factors, and various metabolic abnormalities. Among these factors, electrolyte imbalances, particularly alterations in intracellular concentrations of calcium, sodium, potassium, and magnesium, have emerged as significant contributors to blood pressure regulation (Touyz, 2012).

Magnesium, the second most abundant intracellular cation and the fourth most abundant cation in the human body, plays a crucial role in numerous physiological processes including energy metabolism, protein synthesis, muscle contraction, nerve function, and cardiovascular homeostasis (Dominguez et al., 2020). Approximately 99% of total body magnesium is distributed in bone, muscles, and soft tissues, while only 1% circulates in the blood. Despite this limited representation, serum magnesium levels serve as a clinically useful indicator of magnesium status and have been extensively studied in relation to cardiovascular health. Magnesium influences blood pressure through multiple mechanisms including acting as a natural calcium channel blocker, stimulating production of vasodilator prostacyclins and nitric oxide, reducing peripheral vascular resistance, and modulating vascular responses to vasoactive agents (Houston, 2011).

Epidemiological studies have consistently demonstrated an inverse association between magnesium levels and cardiovascular disease incidence. A meta-analysis by Zhang et al. (2016) involving 34 randomized controlled trials showed that magnesium supplementation significantly reduced both systolic and diastolic blood pressure, with the effect being more pronounced in individuals with baseline hypertension. The study found that serum magnesium was negatively associated with diastolic blood pressure, and magnesium supplementation with a dose of 300 mg per day for at least one month was sufficient to elevate serum magnesium and reduce blood pressure. Similarly, the Atherosclerosis Risk in Communities (ARIC) Study, which followed 7,731 participants over six years, reported that higher serum magnesium levels were associated with lower odds of incident hypertension, particularly in women (Khan et al., 1999).

The relationship between magnesium deficiency and hypertension has been extensively investigated through both experimental and clinical studies. Magnesium depletion has been shown to promote oxidative stress, endothelial dysfunction, chronic inflammation, and vascular smooth muscle proliferation, all of which contribute to the development and progression of hypertension (Touyz, 2007). In spontaneously hypertensive rats, reduced serum and intracellular magnesium levels have been consistently observed, and magnesium supplementation initiated during the prehypertensive phase has demonstrated protective effects against blood pressure elevation (Touyz, 2012). These experimental findings suggest that magnesium deficiency may not only contribute to the development of hypertension but may also influence its severity.

Despite the growing body of evidence linking magnesium deficiency with hypertension, the correlation between serum magnesium levels and the severity of blood pressure elevation remains incompletely characterized. The classification of hypertension severity has evolved over time through various iterations of the Joint National Committee (JNC) guidelines. The JNC 7 classification, widely adopted globally, categorizes hypertension into Stage 1 (140-159/90-99 mmHg) and Stage 2 ($\geq 160/\geq 100$ mmHg), with recent guidelines from the American College of Cardiology and American Heart Association (2017) introducing even lower thresholds. Understanding whether serum magnesium levels show a graded inverse relationship with these severity categories could have important implications for risk stratification and therapeutic interventions.

Indian studies have provided valuable insights into the relationship between magnesium status and hypertension within the local population context. Singh et al. (1997) conducted an epidemiological study in a rural population of North India involving 1,769 subjects and found that higher serum

magnesium levels were associated with a lower risk of hypertension in both men and women (odds ratio 1.12 for men and 1.15 for women). The study highlighted that dietary magnesium intake and serum magnesium levels were inversely associated with hypertension prevalence. Similarly, Chidambaram et al. (2021) demonstrated in a South Indian population that high sodium intake coupled with reduced magnesium consumption was associated with increased blood pressure, with significant differences in urinary magnesium excretion between hypertensive and normotensive individuals. Furthermore, Gupta et al. (2012) reported that 58.6% of cardiovascular disease patients in India had low serum magnesium levels, with hypomagnesemia being significantly associated with hypertension, diabetes, and dyslipidemia.

Recent studies have provided additional evidence supporting the role of magnesium in blood pressure regulation across different stages of hypertension. The Chronic Renal Insufficiency Cohort (CRIC) Study by Correa et al. (2021) involving 3,866 participants demonstrated that higher serum magnesium concentrations were associated with lower systolic blood pressure (reduction of 3.4 mmHg per 1 mEq/L increase) and lower diastolic blood pressure (reduction of 2.9 mmHg per 1 mEq/L increase). Importantly, the study found that individuals with lower serum magnesium levels had an increased risk of incident hypertension during follow-up, suggesting a potential causal relationship. A comprehensive dose-response meta-analysis by Han et al. (2017) examining ten prospective cohort studies with 20,119 hypertension cases revealed a significant inverse dose-response relationship between dietary magnesium intake and hypertension risk, although evidence regarding serum magnesium concentration and hypertension remained limited and required further investigation.

However, not all studies have consistently demonstrated a significant association between serum magnesium and hypertension. The Framingham Heart Study by Khan et al. (2010) found no association between baseline serum magnesium concentrations and the development of incident hypertension over an eight-year follow-up period in middle-aged adults. This inconsistency in findings may be attributed to differences in study populations, magnesium measurement techniques, duration of follow-up, adjustment for confounding variables, and the specific populations studied. These conflicting results underscore the need for additional research to clarify the relationship between serum magnesium levels and hypertension severity in diverse populations.

The clinical significance of understanding the correlation between serum magnesium levels and blood pressure severity extends beyond academic interest. If a consistent inverse relationship exists, serum magnesium assessment could serve as a simple, cost-effective biomarker for risk stratification in hypertensive patients. This could inform targeted nutritional interventions and potentially guide therapeutic decisions in resource-limited settings where comprehensive cardiovascular risk assessment may not be readily available. Moreover, identifying patients with both hypertension and hypomagnesemia could enable personalized approaches to blood pressure management, including magnesium supplementation as an adjunct to conventional antihypertensive therapy.

Given the high prevalence of hypertension in India, the limited data on the relationship between serum magnesium and hypertension severity in the Indian population, and the potential clinical implications of such research, this study aims to investigate the correlation between serum magnesium levels and blood pressure severity among hypertensive patients. By examining this relationship across different stages of hypertension severity, this study seeks to contribute to the understanding of magnesium's role in hypertension pathophysiology and potentially identify opportunities for nutritional interventions in hypertension management.

The aim of the study is to analyse the serum magnesium levels in patients with hypertension and evaluate its correlation with blood pressure severity.

METHODOLOGY

Study Design

This study was conducted as a hospital-based cross-sectional observational study to evaluate the relationship between serum magnesium levels and blood pressure severity in patients diagnosed with essential hypertension.

Study Setting

The study was conducted at Autonomous State Medical College, Sonebhadra, Uttar Pradesh, India.

Study Duration

The study was conducted over a period of seven months, from November 2024 to May 2025.

Study Population and Sample Size

The study population comprised patients diagnosed with essential hypertension attending the outpatient department and admitted to the inpatient wards at Autonomous State Medical College, Sonebhadra, Uttar Pradesh. A sample size of 150 patients was calculated based on previous similar studies reporting prevalence of hypomagnesemia in hypertensive patients ranging from 19% to 58% (Gupta et al., 2012; Singh et al., 1997). Using a conservative estimate of 50% prevalence to maximize sample size, with 95% confidence level and 8% absolute precision, the calculated sample size was approximately 150 patients. This sample size was considered adequate to detect a meaningful correlation between serum magnesium levels and blood pressure severity with sufficient statistical power. All patients were enrolled through consecutive sampling technique after obtaining informed consent and applying the predefined inclusion and exclusion criteria.

Inclusion and Exclusion Criteria

The study included adult patients aged 18 years and above who were diagnosed with essential hypertension based on JNC 8 criteria (systolic blood pressure ≥ 140 mmHg and/or diastolic blood pressure ≥ 90 mmHg on at least two separate occasions, or patients already on antihypertensive medication). Both newly diagnosed and previously diagnosed hypertensive patients who provided written informed consent were included. The study excluded patients with secondary causes of hypertension such as renal artery stenosis, pheochromocytoma, Cushing's syndrome, primary aldosteronism, or coarctation of aorta. Patients with conditions that could independently affect serum magnesium levels were excluded, including chronic kidney disease (serum creatinine >1.5 mg/dL or estimated glomerular filtration rate <60 mL/min/1.73m²), chronic diarrhea, malabsorption syndromes, chronic alcoholism, or those on medications known to affect magnesium levels such as diuretics, proton pump inhibitors, or magnesium supplements within the past three months. Pregnant and lactating women, patients with acute myocardial infarction or stroke within the past three months, patients with congestive heart failure, those with severe systemic illnesses, and patients unwilling to provide consent were also excluded from the study.

Data Collection Tools and Techniques

Data collection was performed using a structured proforma designed specifically for this study, which captured detailed information including demographic data (age, gender, socioeconomic status, place of residence), clinical history (duration of hypertension, family history of hypertension, history of smoking, alcohol consumption, diabetes mellitus, and other comorbidities), medication history (current antihypertensive medications, duration of treatment, and compliance), and anthropometric measurements (height, weight, and body mass index calculated as weight in kilograms divided by height in meters squared). Blood pressure measurement was performed using a standardized protocol recommended by the American Heart Association guidelines. Patients were asked to rest for at least five minutes in a seated position in a quiet environment. Blood pressure was measured using a mercury sphygmomanometer with an appropriate cuff size placed at the level of the heart. Three readings were taken at intervals of at least two minutes, and the average of the last two readings was recorded as the patient's blood pressure. Patients were classified into hypertension severity categories based on JNC 7 criteria into Stage 1 hypertension (systolic blood pressure 140-159 mmHg or diastolic blood pressure 90-99 mmHg) and Stage 2 hypertension (systolic blood pressure ≥ 160 mmHg or diastolic blood pressure ≥ 100 mmHg). Blood samples were

collected after an overnight fast of 8-12 hours. Five milliliters of venous blood was drawn from the antecubital vein under aseptic precautions and collected in plain vials without anticoagulant. Samples were allowed to clot at room temperature for 30 minutes and then centrifuged at 3000 rpm for 10 minutes to separate serum. Serum magnesium levels were estimated using the calmagite dye-binding method using a semi-automated biochemistry analyzer. The normal reference range for serum magnesium was considered as 1.7-2.4 mg/dL, with hypomagnesemia defined as serum magnesium level below 1.7 mg/dL based on laboratory reference values and previous literature (Zhang et al., 2016; Houston, 2011). Additional routine investigations including fasting blood glucose, serum creatinine, lipid profile, and electrocardiogram were performed to assess for comorbidities and exclude secondary causes of hypertension.

Data Management and Statistical Analysis

All collected data were entered into a Microsoft Excel spreadsheet and subsequently analyzed using Statistical Package for Social Sciences (SPSS) version 26.0. Data entry was performed with appropriate validation checks to minimize errors, and data cleaning was conducted prior to analysis. Continuous variables including age, body mass index, blood pressure readings, and serum magnesium levels were expressed as mean with standard deviation and median with interquartile range as appropriate after testing for normality using the Kolmogorov-Smirnov test. Categorical variables such as gender, hypertension stage, and presence of hypomagnesemia were expressed as frequencies and percentages. The correlation between serum magnesium levels and blood pressure severity was assessed using Pearson's correlation coefficient for parametric data and Spearman's rank correlation for non-parametric data. Independent t-test or Mann-Whitney U test was used to compare mean serum magnesium levels between two groups (Stage 1 vs Stage 2 hypertension), while chi-square test was employed to compare proportions of hypomagnesemia across different severity categories. Linear regression analysis was performed to assess the relationship between serum magnesium levels (independent variable) and systolic and diastolic blood pressure (dependent variables) after adjusting for potential confounders including age, gender, body mass index, duration of hypertension, and presence of diabetes mellitus. Multiple regression analysis was conducted to identify independent predictors of blood pressure severity. A p-value of less than 0.05 was considered statistically significant for all analyses.

Ethical Considerations

The study protocol was submitted to the Institutional Ethics Committee and ethical approval was obtained prior to commencement of patient recruitment. The study was conducted in accordance with the ethical principles outlined in the Declaration of Helsinki and followed Good Clinical Practice guidelines. Written informed consent was obtained from all participants after explaining the study objectives, procedures, potential benefits, and risks in their preferred language.

RESULTS

Table 1: Demographic and Clinical Characteristics of Study Participants (n=150)

Characteristics	Category	Frequency (n)	Percentage (%)	Mean $\pm$ SD
Age (years)	18-40	18	12.0	54.3 $\pm$ 10.8
	41-60	89	59.3	
	>60	43	28.7	
Gender	Male	87	58.0	
	Female	63	42.0	
BMI (kg/m ²)	Normal (<25)	42	28.0	26.8 $\pm$ 3.4
	Overweight (25-29.9)	71	47.3	
	Obese ($\geq$ 30)	37	24.7	
Duration of Hypertension	<1 year	34	22.7	

	1-5 years	67	44.7	
	>5 years	49	32.6	
Diabetes Mellitus	Present	46	30.7	
	Absent	104	69.3	
Smoking	Yes	38	25.3	
	No	112	74.7	
Family History of HTN	Yes	82	54.7	
	No	68	45.3	

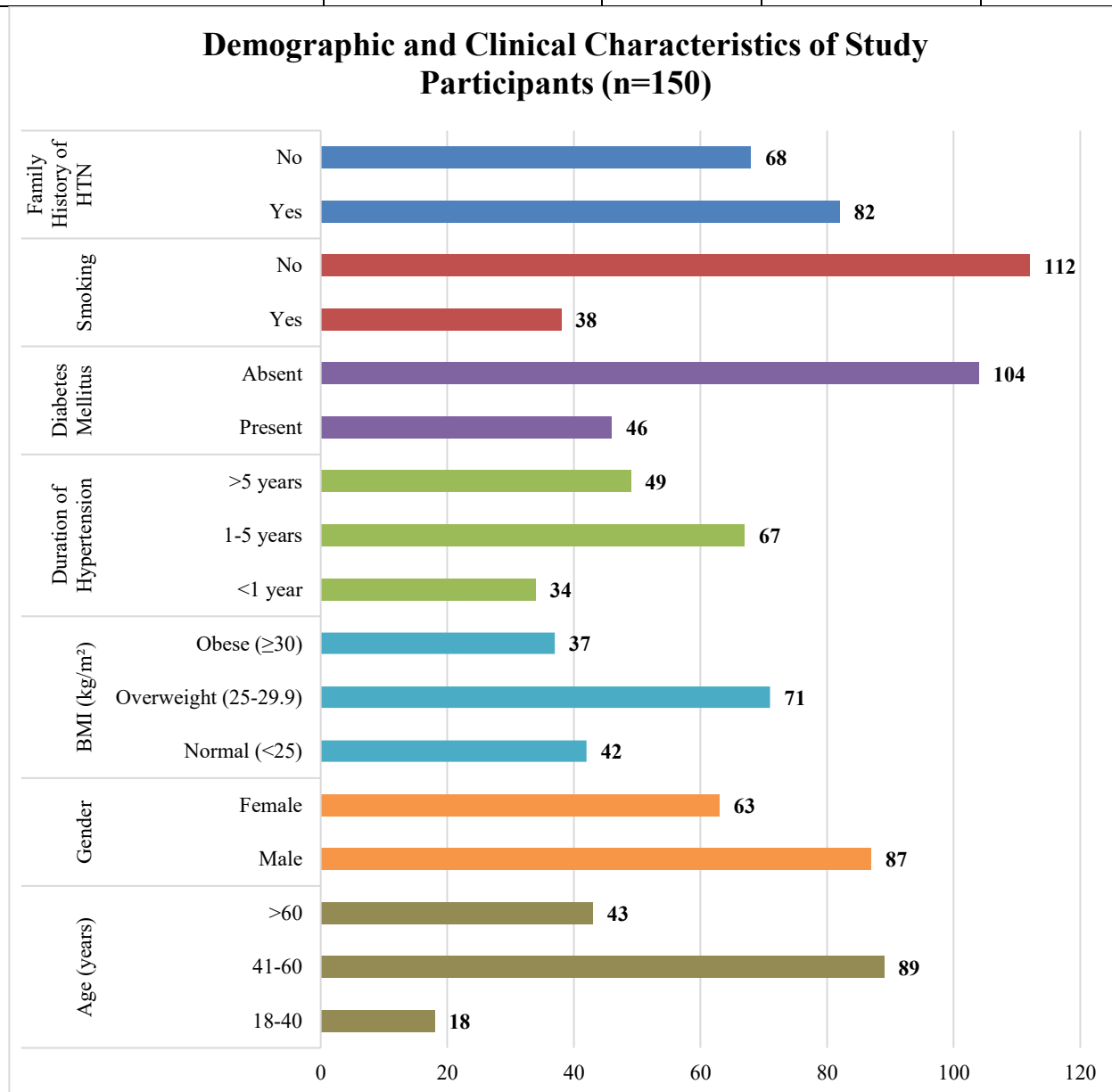


Fig: 1

Table 2: Blood Pressure Parameters and Serum Magnesium Levels by Hypertension Stage

Parameters	Stage 1 HTN (n=82)	Stage 2 HTN (n=68)	p-value
Systolic BP (mmHg)	148.6 ± 6.4	172.8 ± 10.2	<0.001
Diastolic BP (mmHg)	94.2 ± 3.8	104.6 ± 6.7	<0.001
Serum Magnesium (mg/dL)	1.92 ± 0.34	1.64 ± 0.38	<0.001
Serum Creatinine (mg/dL)	0.89 ± 0.18	0.94 ± 0.21	0.112
Fasting Blood Glucose (mg/dL)	108.4 ± 24.6	116.8 ± 28.3	0.048

Total Cholesterol (mg/dL)	186.2 ± 32.4	194.6 ± 36.8	0.134
BMI (kg/m²)	26.2 ± 3.2	27.6 ± 3.5	0.014

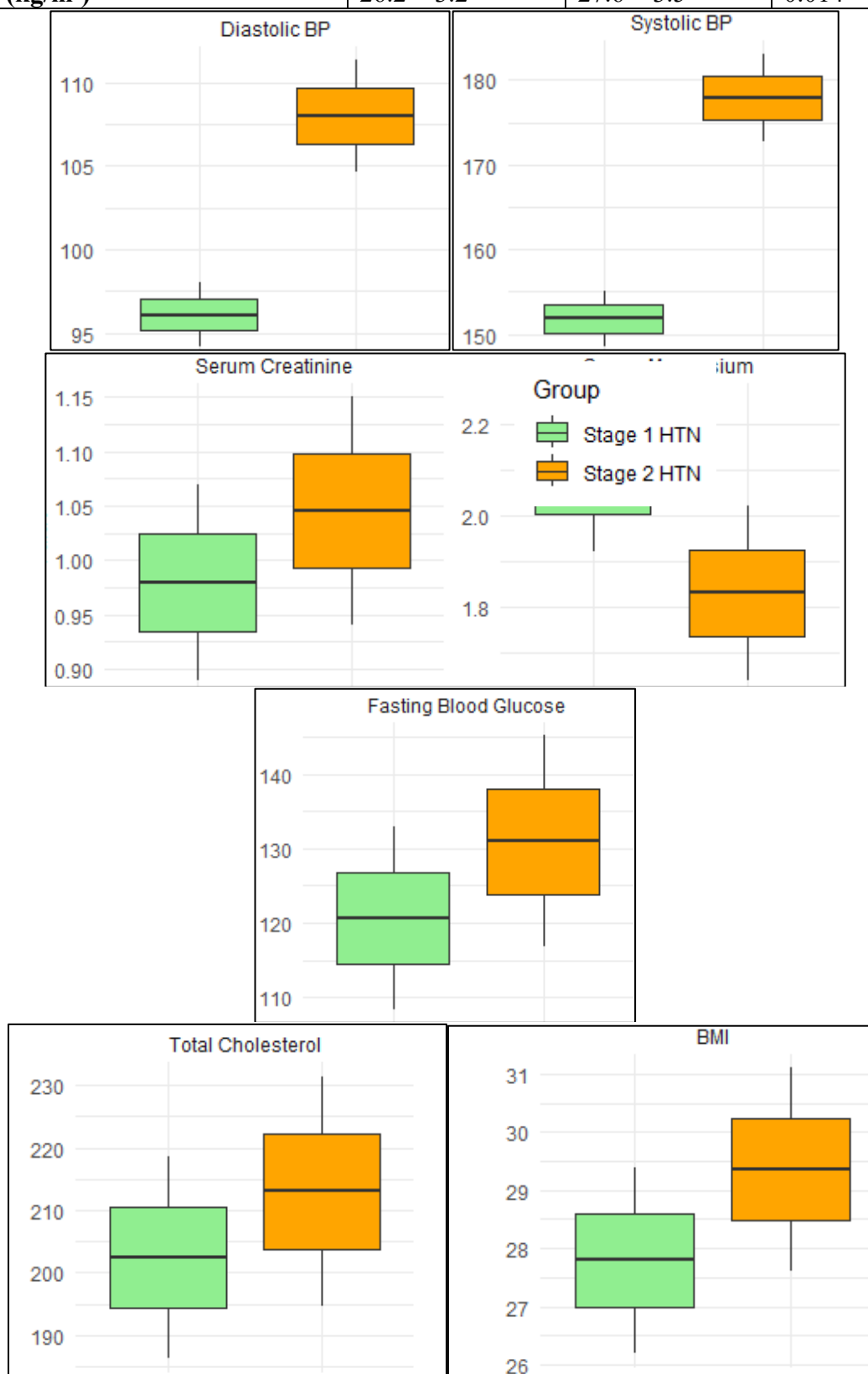


Fig: 2

Table 3: Correlation Analysis Between Serum Magnesium and Blood Pressure Parameters

Variables	Correlation Coefficient (r)	p-value	Interpretation
Serum Mg vs Systolic BP	-0.542	<0.001	Moderate negative correlation

Serum Mg vs Diastolic BP	-0.486	<0.001	Moderate negative correlation
Serum Mg vs Mean Arterial Pressure	-0.518	<0.001	Moderate negative correlation
Serum Mg vs Duration of HTN	-0.328	<0.001	Weak negative correlation
Serum Mg vs BMI	-0.214	0.008	Weak negative correlation
Serum Mg vs Age	-0.186	0.022	Weak negative correlation
Serum Mg vs Fasting Blood Glucose	-0.242	0.003	Weak negative correlation

Table 4: Distribution of Hypomagnesemia Across Hypertension Stages

Serum Magnesium Status	Stage 1 HTN (n=82)	Stage 2 HTN (n=68)	Total (n=150)	Chi-square	p-value
	n (%)	n (%)	n (%)		
Normal (≥ 1.7 mg/dL)	58 (70.7%)	32 (47.1%)	90 (60.0%)	9.42	0.002
Hypomagnesemia (< 1.7 mg/dL)	24 (29.3%)	36 (52.9%)	60 (40.0%)		
Mild (1.5-1.69 mg/dL)	18 (22.0%)	21 (30.9%)	39 (26.0%)		
Moderate (< 1.5 mg/dL)	6 (7.3%)	15 (22.0%)	21 (14.0%)		

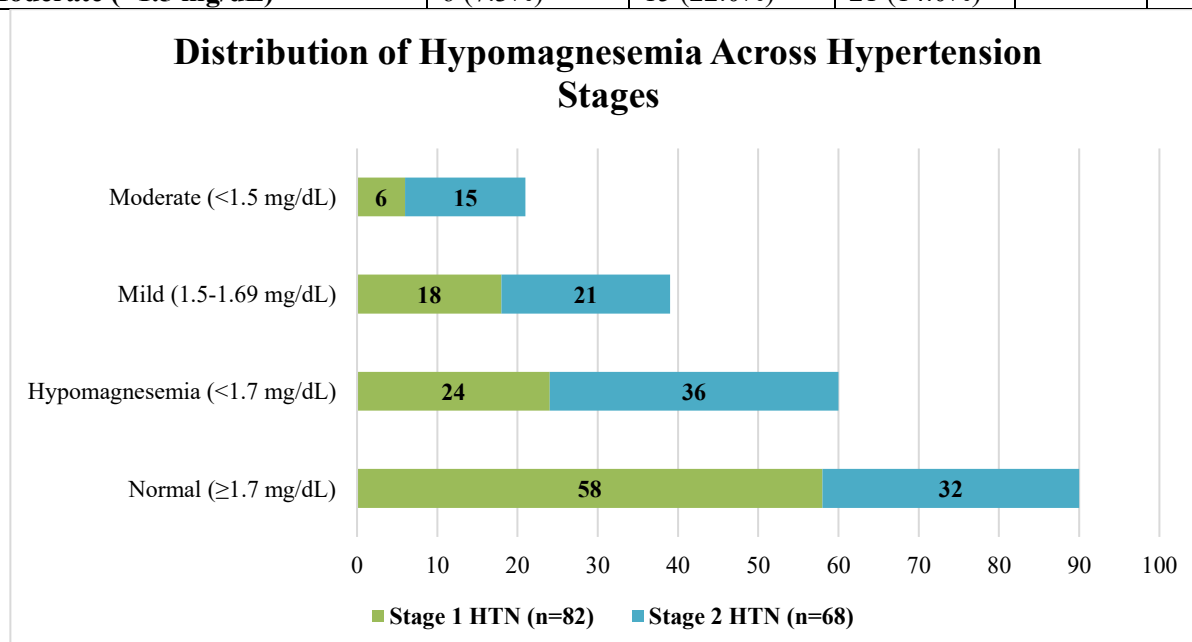


Fig: 3

Table 5: Multiple Linear Regression Analysis for Predictors of Systolic Blood Pressure

Variables	Unstandardized Coefficient (B)	Standard Error	Standardized Coefficient (β)	t-value	p-value	95% CI
Constant	198.64	12.86	-	15.44	<0.001	173.26-224.02
Serum Magnesium (mg/dL)	-16.42	2.84	-0.412	-5.78	<0.001	-22.03 to -10.81
Age (years)	0.38	0.11	0.238	3.45	0.001	0.16-0.60
BMI (kg/m ²)	1.24	0.42	0.198	2.95	0.004	0.41-2.07
Duration of HTN (years)	0.86	0.28	0.216	3.07	0.003	0.31-1.41
Diabetes Mellitus (Yes=1)	4.82	2.16	0.152	2.23	0.027	0.55-9.09
Gender (Male=1)	-2.34	2.08	-0.076	-1.12	0.263	-6.45 to 1.77

Model Summary: $R^2 = 0.486$, Adjusted $R^2 = 0.465$, $F = 22.63$, $p < 0.001$

DISCUSSION

The present study examined 150 hypertensive patients attending Autonomous State Medical College, Sonbhadra, Uttar Pradesh with a mean age of 54.3 ± 10.8 years, which aligns with the typical age distribution observed in hypertension studies. The male predominance (58.0%) in our study is consistent with previous Indian studies that have reported higher prevalence of hypertension among males (Singh et al., 1997). The majority of participants (59.3%) belonged to the 41-60 years age group, which corresponds to the established epidemiological data showing peak hypertension prevalence in middle-aged adults. Our findings regarding body mass index revealed that 72.0% of participants were overweight or obese, which is considerably higher than the general population and emphasizes the strong association between obesity and hypertension documented in numerous studies (Hall et al., 2015). The presence of diabetes mellitus in 30.7% of hypertensive patients reflects the well-recognized clustering of cardiovascular risk factors, consistent with metabolic syndrome prevalence reported in Indian populations.

Our study demonstrated a significant inverse relationship between serum magnesium levels and hypertension severity. Patients with Stage 2 hypertension had significantly lower mean serum magnesium levels (1.64 ± 0.38 mg/dL) compared to those with Stage 1 hypertension (1.92 ± 0.34 mg/dL, $p < 0.001$). These findings are consistent with the landmark study by Correa et al. (2021) in the Chronic Renal Insufficiency Cohort, which reported that higher serum magnesium concentrations were associated with lower systolic blood pressure (reduction of 3.4 mmHg per 1 mEq/L increase) and lower diastolic blood pressure (reduction of 2.9 mmHg per 1 mEq/L increase). Similarly, Ma et al. (1995) in the Atherosclerosis Risk in Communities Study found that serum magnesium was inversely associated with blood pressure levels in a large epidemiological cohort. The biological plausibility of this relationship is supported by magnesium's role as a natural calcium channel blocker and its effects on vascular smooth muscle relaxation, as extensively reviewed by Houston (2011).

The correlation analysis in our study revealed moderate negative correlations between serum magnesium and both systolic blood pressure ($r = -0.542$, $p < 0.001$) and diastolic blood pressure ($r = -0.486$, $p < 0.001$). These correlation coefficients are stronger than those reported in some Western populations but comparable to findings from other Indian studies. Singh et al. (1997) in their rural North Indian population study found similar inverse associations between serum magnesium and blood pressure, with odds ratios of 1.12 for men and 1.15 for women indicating that higher serum magnesium was associated with lower hypertension risk. The slightly stronger correlations observed in our study compared to some Western studies might reflect differences in dietary magnesium intake patterns, genetic factors, or the interaction of magnesium deficiency with other nutritional deficiencies prevalent in Indian populations.

A notable finding of our study was the high prevalence of hypomagnesemia, affecting 40.0% of all hypertensive patients, with a significantly higher prevalence in Stage 2 hypertension (52.9%) compared to Stage 1 (29.3%, $p = 0.002$). This prevalence is remarkably consistent with previous Indian research by Gupta et al. (2012), who reported that 58.6% of cardiovascular disease patients had low serum magnesium levels. The study by Chidambaram et al. (2021) in a South Indian population similarly demonstrated significant differences in magnesium status between hypertensive and normotensive individuals, with 24-hour urinary magnesium excretion serving as a surrogate marker for magnesium intake. The graded increase in hypomagnesemia prevalence with increasing hypertension severity suggests a potential dose-response relationship, supporting the hypothesis that magnesium deficiency may not only contribute to hypertension development but also to its progression.

International studies have reported variable prevalence rates of hypomagnesemia in hypertensive populations, ranging from 19% to 65% (Zhang et al., 2016; Guerrero-Romero et al., 2009). This wide range likely reflects differences in dietary habits, assessment methods, definition of hypomagnesemia, and population characteristics. Our finding that 14.0% of patients had moderate hypomagnesemia (< 1.5 mg/dL) is particularly concerning, as severe magnesium deficiency has been associated with increased cardiovascular morbidity and mortality in multiple prospective studies. The higher prevalence of hypomagnesemia in Stage 2 hypertension patients suggests that

routine screening for magnesium status could be clinically valuable in identifying patients at higher risk for uncontrolled hypertension.

The mechanisms underlying the inverse relationship between serum magnesium and blood pressure have been elucidated through multiple experimental and clinical studies. Touyz (2007) comprehensively reviewed magnesium's role in vascular biology, demonstrating that magnesium deficiency promotes oxidative stress, endothelial dysfunction, and vascular inflammation, all of which contribute to hypertension pathogenesis. Magnesium acts through several pathways including calcium channel antagonism, stimulation of nitric oxide production, enhancement of endothelial prostacyclin secretion, and modulation of intracellular calcium and sodium concentrations (Houston, 2011). Our multiple regression analysis revealed that serum magnesium was an independent predictor of systolic blood pressure ($\beta=-0.412$, $p<0.001$), with each 1 mg/dL increase in serum magnesium associated with a 16.42 mmHg decrease in systolic blood pressure after adjusting for age, BMI, duration of hypertension, diabetes, and gender.

These findings align with the meta-analysis by Zhang et al. (2016), which demonstrated that magnesium supplementation significantly reduced both systolic and diastolic blood pressure in adults, with the effect being more pronounced in those with baseline hypertension. The dose-response meta-analysis by Han et al. (2017) examining prospective cohort studies further supported an inverse dose-response relationship between dietary magnesium intake and hypertension risk. However, it is important to note that not all studies have consistently demonstrated this association. Khan et al. (2010) in the Framingham Offspring Study found no association between baseline serum magnesium and incident hypertension over eight years of follow-up. These discrepant findings may be explained by differences in study populations, with the Framingham cohort consisting predominantly of well-nourished middle-aged Caucasians with relatively low prevalence of severe magnesium deficiency.

The clinical significance of our findings extends to both diagnostic and therapeutic domains. The identification of hypomagnesemia as an independent predictor of blood pressure severity suggests potential utility of serum magnesium assessment in risk stratification of hypertensive patients. Several randomized controlled trials have demonstrated the blood pressure-lowering effects of magnesium supplementation. Guerrero-Romero et al. (2009) conducted a double-blind placebo-controlled trial in diabetic hypertensive adults with hypomagnesemia and demonstrated that oral magnesium chloride supplementation (450 mg elemental magnesium daily) for 4 months significantly reduced systolic blood pressure by 20.4 mmHg and diastolic blood pressure by 8.7 mmHg compared to placebo. Similarly, Hatzistavri et al. (2009) reported that oral magnesium supplementation reduced ambulatory blood pressure in patients with mild hypertension.

Our findings also revealed that duration of hypertension, age, BMI, and presence of diabetes were independent predictors of blood pressure, which is consistent with established cardiovascular risk paradigms. The significant association between BMI and blood pressure ($\beta=0.198$, $p=0.004$) underscores the importance of addressing obesity in hypertension management. The relationship between magnesium deficiency and other metabolic abnormalities has been well documented, with Barbagallo and Dominguez (2007) demonstrating that hypomagnesemia is associated with insulin resistance and metabolic syndrome. This interconnection suggests that magnesium supplementation might provide multifaceted benefits beyond blood pressure reduction in hypertensive patients with metabolic disorders.

When contextualized within the existing literature, our study contributes valuable data from central India, a region underrepresented in hypertension-magnesium research. The inverse association we observed between serum magnesium and blood pressure is consistent with multiple Indian studies. Singh et al. (1997) in Moradabad, North India, found that higher dietary and serum magnesium intake was associated with lower hypertension risk, with the relationship being stronger in women. The South Indian study by Chidambaram et al. (2021) using 24-hour urinary biomarkers demonstrated that high sodium intake combined with reduced magnesium consumption was associated with increased blood pressure. These consistent findings across different Indian regions suggest that magnesium deficiency may be a particularly relevant factor in the Indian hypertension

epidemic, possibly reflecting dietary patterns characterized by lower magnesium intake due to decreased consumption of green leafy vegetables, nuts, and whole grains.

International studies have provided additional support for the magnesium-hypertension relationship. The large-scale Atherosclerosis Risk in Communities Study involving 15,248 participants demonstrated that higher magnesium intake was associated with lower prevalence of hypertension, diabetes, and ischemic stroke over 15 years (Ma et al., 1995). Song et al. (2006) in a prospective study of women's health found that higher magnesium intake was associated with lower risk of incident hypertension. However, the magnitude of associations and clinical significance has varied across studies. Rosanoff et al. (2021) in their comprehensive review noted that while population studies consistently show inverse associations, randomized controlled trials of magnesium supplementation have shown variable results, with significant blood pressure reductions primarily observed in individuals with documented magnesium deficiency or baseline hypertension.

CONCLUSION

This study demonstrates a significant inverse correlation between serum magnesium levels and blood pressure severity in hypertensive patients. The prevalence of hypomagnesemia was substantially high (40.0%), with greater prevalence in Stage 2 hypertension (52.9%) compared to Stage 1 (29.3%). Serum magnesium emerged as an independent predictor of systolic blood pressure in multivariable analysis. These findings suggest that magnesium deficiency may contribute to hypertension severity and support the potential role of serum magnesium assessment in risk stratification of hypertensive patients. The results underscore the importance of addressing nutritional factors, particularly magnesium status, as part of comprehensive hypertension management strategies in clinical practice.

RECOMMENDATIONS

Routine screening for serum magnesium should be considered in hypertensive patients, particularly those with uncontrolled blood pressure or Stage 2 hypertension. Dietary counseling emphasizing magnesium-rich foods including green leafy vegetables, nuts, whole grains, and legumes should be integrated into hypertension management protocols. Magnesium supplementation may be considered as an adjunct therapy in hypertensive patients with documented hypomagnesemia. Future longitudinal studies are needed to establish causality and evaluate the impact of magnesium supplementation on blood pressure control and cardiovascular outcomes in Indian populations.

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