

ORIGINAL RESEARCH

Investigation of the association between blood pressure and serum magnesium levels in patients with normal and hypertensive conditions

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ABSTRACT

Introduction: Rich in whole grains, legumes, nuts, and leafy green vegetables, magnesium is a necessary mineral. Magnesium functions as a cofactor in a few of our body's enzymes and is involved in the creation of several proteins. Thus, in order to manage the rise in blood pressure despite pharmaceutical treatment, it is important to investigate the magnesium levels in hypertensive individuals in order to understand the link between serum magnesium and blood pressure. This may be done by supplementing with and modifying diet. **Materials and Methods:** The body weight was measured in kilogrammes using a portable weighing machine, and the standing height was measured in centimetres using a stadiometer. To record the blood pressure, a standardised mercury sphygmomanometer was utilised. They were asked to provide a thorough history in order to rule out renal and kidney illnesses, diabetes mellitus, and secondary hypertension causes. **Results:** The serum magnesium level in Group B is comparatively lower than that of Groups A and C. The magnesium level in Group A is lower than in the control group. Group B has the lowest magnesium level among these groups, and Group A has a lower magnesium level as well. This difference is statistically significant. **Conclusion:** By modulating various mechanisms involved in the control of normal blood pressure, magnesium obtains its significance.

Keywords: Magnesium, proteins, hypertension, and normotension

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INTRODUCTION

The finding of safe and effective preventative interventions that may decrease blood pressure (BP) even slightly might have a major influence on public health, because the prevalence and incidence of hypertension are rising. Several fundamental processes have been proposed by a number of lines of evidence from laboratory studies. By directly promoting the production of prostacyclin and nitric oxide, modulating endothelium-dependent and endothelium-independent vasodilation,^{2,3} lowering vascular tone and reactivity,⁴ and preventing vascular injury through its antioxidant and anti-inflammatory properties, magnesium may play a significant role in the regulation of blood pressure.^{5, 6} A multitude of experimental investigations have shown a pathophysiological connection, in a variety of animal models, between reduced blood magnesium levels (hypomagnesemia) or tissue⁷⁻⁹ and hypertension.

Prior to the period of antibiotics, communicable illnesses were prevalent; however, with the development of new, effective antibiotics, communicable diseases have become less of a concern. Non-communicable illnesses are currently seen as a worldwide health emergency due to the rise in the incidence of conditions including cancer, hypertension, diabetes, stroke, and cardiovascular disorders.¹⁰ According to the World Health Organization's (2010) Global Status Report, non-communicable illnesses account for 60% of all deaths globally. The situation in India is dire. Non-communicable illnesses were responsible for 44% of daily adjusted life years lost and 53% of overall mortality in 2005. In India, non-communicable illnesses will account for 67% of all deaths by 2030.¹¹

As per Hippocrates' dictum, "Let food be thy medicine, and medicine be thy food," dietary practices

have a significant role in the aetiology of non-communicable illnesses. Magnesium is consistently the mineral that is least thought of in the diet, apart from carbs, proteins, and fat; minerals also play a significant role in the minerals. Magnesium, however, offers unique medical benefits. Rich in whole grains, legumes, nuts, and leafy green vegetables, magnesium is a necessary mineral. Magnesium functions as a cofactor in a few of our body's enzymes and is involved in the creation of several proteins. Magnesium is essential for hundreds of physiological processes in the human body that maintain equilibrium. Blood pressure (BP) modulation is one of magnesium's primary homeostatic roles.¹² The National Health and Nutrition Examination Survey (1999–2000) indicates that inadequate magnesium intake in the diet will impact the pathways of inflammation and metabolism, which will ultimately result in the clinical manifestation of cardiovascular diseases, metabolic syndrome, Type 2 diabetes mellitus, and hypertension.¹² Given that hypertension is the primary risk factor for stroke and cardiovascular illnesses, a well-thought-out strategy should be used to identify the pathophysiological variables and avoid hypertension. Thus, in order to manage the rise in blood pressure despite pharmaceutical treatment, it is important to investigate the magnesium levels in hypertensive individuals in order to understand the link between serum magnesium and blood pressure. This may be done by supplementing with and modifying diet.

MATERIALS AND METHODS

To gather a thorough medical history, measure the anthropometric indices, and record vital statistics, a comprehensive pro forma was used. The body weight was measured in kilogrammes using a portable weighing machine, and the standing height was measured in centimetres using a stadiometer. To record the blood pressure, a standardised mercury sphygmomanometer was utilised. The Institutional Ethics Committee gave their approval to the project.

told permission was gained when the individuals were told about the technique. They were asked to provide a thorough history in order to rule out renal and kidney illnesses, diabetes mellitus, and secondary hypertension causes. The individual should be placed in an armed chair in a quiet room with their legs spread apart and their back and arms supported. He or she should have the mercury manometer at heart level. Caffeine use must be stopped 30 minutes before to the blood pressure test. After that, a regular sphygmomanometer with a cuff size of 25 cm by 12.5 cm was used to record each subject's blood pressure. Using the auscultatory approach, the BP was recorded twice, and the mean value was taken for analysis. For the purpose of collecting venous blood, the antecubital vein on the front of the forearm was chosen. A cotton swab was used to sterilise the area around the vein. After inserting a single, sterile, disposable needle with a 10-milliliter syringe into the vein, the required volume of blood was drawn. The blood was centrifuged at 3000 revolutions per minute for five minutes in order to extract the serum. The Calmagite technique was used to evaluate serum magnesium levels. To rule out albuminuria, a normal urine examination was performed on all 75 individuals using the lab reader technique.

To test the effects of both blood pressure status (hypertensive versus normotensive subjects) and race (black versus white subjects), all data were analysed using two-way analysis of variance (ANOVA) and statistical significance was determined by post hoc *t* tests for all means (Bonferroni-Dunn) (Super Anova, Abacus Concepts, Berkeley, CA). The means \pm SEM are used to report all data.

RESULTS

In the age- and sex-adjusted groups, the intergroup comparison of serum magnesium was done using analysis of variance. $P < 0.05$ as considered as statistically significant. To study the correlation between serum magnesium and BP, Pearson's correlation was done.

Table 1: Classification of BP for adults ages 18 years and older by Joint National Committee seven

Category	Systolic BP (mm Hg)	Diastolic BP (mm Hg)
Normal	<120 and	<80
Prehypertension	120–140 and	80–90
Hypertension		
Stage 1	140–160 or	90–100
Stage 2	≥ 170	≥ 110

BP: Bloodpressure

When analyzed further, however, it was observed that this difference was only significant among white individuals (NIBP: 0.621 ± 0.007 v HiBP: 0.580 ± 0.022 mmol/L, $P = .0096$), but not among black subjects, in whom Mg-ion levels among normotensive and hypertensive individuals did not significantly differ (NIBP: 0.578 ± 0.008 v 0.554 ± 0.013 mmol/L, $P = .1610$) (Table 2).

Table 2: Serum levels of total mg (mg-tot), ionized mg (mg-ion), and ionized calcium/ionized mg ratios in normotensive (nlbp) and hypertensive (hibp) subjects grouped according to race

Group	Mg-tot (mmol/L)	Mg-ion (mmol/L)	Ca-ion/Mg-ion
White NIBP	0.878 ± 0.007	0.621 ± 0.007	1.955 ± 0.019
HiBP	0.809 ± 0.027	0.579 ± 0.022†	2.104 ± 0.071†
Black NIBP	0.818 ± 0.012*	0.578 ± 0.008*	2.097 ± 0.028*
HiBP	0.781 ± 0.016	0.554 ± 0.013	2.191 ± 0.037

* $P < .0001$ v white subjects; † $P < .01$ v NIBP.

According to Table 3, in Group B, the serum magnesium level is lower when compared to Groups A and C. The Group A is having lower magnesium level than the control group. Among these groups, Group B is having the lowest magnesium level, and Group A is having lower magnesium level, and the difference is statistically significant.

Table 3: Comparison of serum magnesium between the groups			
Groups	Mean ± Standard deviation (serum magnesium)	F value	P value
A	1.5561 ± 0.40321	17.50	0.0001*
B	1.3921 ± 0.4082		
C	2.069 ± 0.4516		

*The parameters is statistically significant as P value is less than 0.01

DISCUSSION

The average blood magnesium level in the control group is 2.068 ± 0.4515 , but in the Stage 1 and Stage 2 hypertensives, it is 1.5560 ± 0.40320 and 1.3920 ± 0.4081 , respectively. There is a significant difference in the serum magnesium levels across the groups, as shown by $P < 0.05$. The blood magnesium level is lower in Stage 1 hypertensives compared to the normotensive patients, and it is lower in Stage 2 hypertensives compared to the Stage 1 hypertension subjects. Therefore, it is clear from the data that as blood pressure rises, serum magnesium levels fall. Serum magnesium levels and systolic and diastolic blood pressure have a negative connection that is statistically significant ($P < 0.01$) between the groups. According to Resnick et al., serum magnesium and plasma renin activity continuously correlate negatively in essential hypertension ($r = -0.60$, $P < 0.001$). Thirteen Ferdousi et al. found that the erythrocyte magnesium level (mg/dl) was lower in cases compared to controls (4.46 ± 0.699 vs. 5.43 ± 0.775 , $P < 0.001$) and that the serum magnesium level (mg/dl) was significantly lower in 30 off springs of essential hypertensive parents when compared to the 30 age- and sex-matched off springs of normotensive parents (1.90 ± 0.210 vs. 2.13 ± 0.366 , $P < 0.01$).¹⁴ There is a negative association between blood pressure and serum magnesium in this research as well. The BMIs of the Stage 1 and Stage 2 hypertensives in the current research (24.2160 ± 0.5225 vs. 24.3680 ± 1.1433 vs. 23.2000 ± 0.8036 , $P < 0.05$) were considerably higher than those of the normotensive patients, although being within normal limits. The results are in line with the research that Ohira et al. undertook. He discovered in his research that the systolic blood pressure and BMI had an inverse connection with serum magnesium.¹⁵

When evaluating this early statistics, a few cautions must be taken into account. First, although black subjects had lower magnesium levels even in the

absence of overt hypertensive disease, magnesium deficiency of this mild kind cannot explain hypertension in and of itself; other, as of yet unidentified concomitant factors are most likely required for the development of elevated blood pressure. This is also in line with reduced intracellular free magnesium levels seen in non-hypertensive diseases such non-insulin-dependent diabetes mellitus.^{16–18} In order to ascertain whether the similarly low serum ionised magnesium values in normotensive and hypertensive black subjects also hold true for the intracellular ionic species, we have not evaluated intracellular free magnesium values in a sufficient number of normotensive and hypertensive black subjects. As a result, reduced intracellular or extracellular magnesium levels may be a prerequisite for the development of hypertension but not a sufficient one. Second, further documentation is required on the use of serum ionised magnesium readings as a reliable indicator of tissue magnesium levels. It is necessary to evaluate intracellular free magnesium levels in organ systems that are important targets for hypertensive and diabetic vascular disease, such as the brain, heart, and kidney, in light of the strong correlation between intracellular free magnesium levels as determined by NMR spectroscopy in peripheral red blood cells and the concurrently measured serum ionised magnesium level.¹⁸ Similar to earlier findings in peripheral blood cells, preliminary results in essential hypertension people point to an intracellular free magnesium deficiency in situ in the brain and skeletal muscle.¹⁹ Third, as was done in this instance, all of these measurements—serum ionised calcium, intracellular free magnesium, and serum ionised magnesium—must be performed when the subject is fasting. This is due to the fact that intracellular free calcium and magnesium levels alter after in vitro glucose and insulin incubation and oral glucose ingestion,²⁰ which seems to be equally and inversely true for serum

calcium and magnesium in the extracellular compartment.²¹ Thus, distinctions between people with hypertension and normotensive subjects may go unnoticed if they are not evaluated while fasting.

CONCLUSION

Elevated peripheral resistance, mostly caused by endothelial dysfunction and vascular tone change, is the primary hemodynamic abnormality in hypertension. By modulating various mechanisms involved in the control of normal blood pressure, magnesium obtains its significance. Many hypothesized pathways exist for how magnesium shortage causes hypertension. Magnesium, together with nitric oxide (NO), modifies the tone of blood vessels via affecting the actions of smooth muscle and endothelium. Any change in the concentration of magnesium induces variations in the synthesis and release of NO, which in turn modifies the concentration of calcium and alters the tone of the smooth muscle in the arteries. We should take action to increase the amount of magnesium in our diets as the current research unequivocally demonstrates the link between low serum magnesium and high blood pressure. In addition to diet, magnesium supplementation may be used to lower blood pressure.

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