

A PROSPECTIVE STUDY OF ASSOCIATION OF SERUM CALCIUM AND MAGNESIUM IN HYPERTENSIVES

Srikanth Dubbaka¹, Satheesh Kumar K², C.Yadavendra Reddy³

Received : 24/04/2026
Received in revised form : 20/05/2026
Accepted : 02/06/2026

Keywords:
Hypocalcemia, hypomagnesemia,
hypertension.

Corresponding Author:
Dr. C. Yadavendra Reddy,
Email: chamareddy_07@yahoo.com

DOI: 10.47009/jamp.2026.8.3.291

Source of Support: Nil,
Conflict of Interest: None declared

Int J Acad Med Pharm
2026; 8 (3); 1654-1660



ABSTRACT

Background: Many studies suggest that reduced consumption of calcium or magnesium is associated with an increased risk of developing hypertension and cardiovascular disease. Results of animal studies show that restriction of calcium increases, and supplementation with calcium lowers, the blood pressure of normal and hypertensive rats. The present study aims to study the association between serum calcium and magnesium in hypertensive patients. **Materials and Methods:** The present study was conducted in the Department of General Medicine in a tertiary care hospital. The study was conducted over one year, from June 2022 to June 2023. The inclusion criteria were age more than 18 years, patients who were diagnosed with hypertension, and patients who gave consent for the study. The exclusion criteria were patients with previous hypocalcemic disorders, patients with previous hypomagnesemic disorders, and patients on drugs causing hypertension. A complete and detailed history was taken, and a general physical examination was done for all patients. **Result:** Out of 150 patients, there was a male preponderance, with 62 females and 88 males. There were more patients in the age group of 51–60 years, with 42 patients. Among the hypertensive patients, 108 were normocalcemic, 34 were hypocalcemic, and 8 were hypercalcemic. There were 79 patients with increased magnesium levels, 22 patients with decreased magnesium levels, and the rest had normal magnesium levels. **Conclusion:** We conclude from the study that decreased calcium levels contribute to hypertension. Further role of magnesium in hypertensive patients could not be inferred due to size of the sample.

INTRODUCTION

Dietary potassium, calcium, and magnesium have each been inversely associated with blood pressure in populations.¹⁻¹⁵ Since these cations exist together in commonly eaten foods such as fruits, nuts, vegetables, cereals, and dairy products, their intakes are highly correlated. This collinearity makes it difficult in epidemiological studies to distinguish which among these dietary cations has a causal role in blood pressure regulation.^[1-8] Meta-analysis of clinical trials found a significant blood pressure-lowering effect (–5.9/–3.4 mm Hg) for potassium supplementation in hypertensive but not in normotensive persons.^[9-16] Meta-analysis suggested that calcium supplementation has a small effect (–1.7 mm) on systolic blood pressure in hypertensive but not normotensive patients.^[17,18] Magnesium produced inconsistent results in trials of hypertensives,^[19-28] and no effect in normotensives.^[29,30] The lack of effect of mineral

supplements in clinical trials of normotensives is inconsistent with the significant associations in epidemiological studies, since normotensive persons comprise the vast majority of the population samples.

One possible explanation for the divergence in findings between the observational epidemiologic studies and the clinical trials in normotensive persons is that a subset of the population that is relatively deficient from low intake of minerals may be particularly responsive to supplementation. Epidemiological studies tend to compare those in the lowest category of intake, which serves as a point of reference, with persons having average or high intake. In contrast, most trials have tested the effect of raising an average intake to a high intake. An alternative explanation is that these minerals work in concert to reduce blood pressure and that they would be more effective when given together. To explore both possibilities, we conducted a clinical trial of potassium, magnesium, and calcium supplements, given singly and together, to

participants in the Nurses Health Study II who reported habitually low intakes of these minerals.

Hypertension and calcium: The regulation of blood pressure is complex with several organs being involved. Intracellular calcium plays a crucial role in the regulation of cardiovascular functions: An increased influx of calcium into the vascular smooth muscle cells leads to an augmental muscular tone and therefore to an increased vascular resistance and rise in blood pressure. Parathormone plays a permissive role since it regulates the calcium-influx into the cells and thus increases the vasoconstrictive effect. There is a positive correlation between parathormone and blood pressure, present in primary as well as secondary hyperparathyroidism. Moreover, patients with essential hypertension have high parathormone levels already before hypertension is diagnosed. A calcium-rich diet (> 1000 mg calcium daily) slightly decreases blood pressure. This positive effect is due to parathormone suppression with a subsequently decreased calcium content in the vascular smooth muscle cells. A calcium-rich diet inhibits lipogenesis in the fat tissue; thus additionally improving the cardiovascular risk profile.

Regulation of intracellular calcium plays a key role in hypertension and obesity. Dysregulation of calcium homeostasis appears to be a fundamental factor linking these conditions. Regulation of intracellular calcium in key disease-related target tissues by calcitrophic hormones provides the opportunity to modulate disease risk with dietary calcium. Overall, sub-optimal calcium intakes contribute to the etiology of salt-sensitivity and hypertension. High salt diets exert a calciuretic effect, serving to exacerbate the physiological consequences of sub-optimal calcium diets. Among these are increases in 1,25-dihydroxyvitamin D, which increases vascular smooth muscle intracellular calcium, thereby increasing peripheral vascular resistance and blood pressure. Dietary calcium reduces blood pressure in large part via suppression of 1,25-dihydroxyvitamin D, thereby normalizing intracellular calcium. The practical relevance of this approach has been confirmed in the DASH (Dietary Approaches to Stop Hypertension) trial, which demonstrated that increasing low-fat dairy product and fruit and vegetable consumption exerted profound blood pressure-lowering effects. The magnitude of this effect among hypertensives was comparable to that typically found in pharmacological trials of mild hypertension. 1,25-dihydroxyvitamin D also stimulates calcium influx in human adipocytes, resulting in stimulation of lipogenesis, inhibition of lipolysis and expansion of triglyceride stores. Accordingly, suppression of 1,25-dihydroxyvitamin D by dietary calcium has been identified as a target, which may contribute to the prevention and management of obesity. Indeed, laboratory, clinical and population data all indicate a significant anti-obesity effect of dietary calcium, although large-scale prospective clinical trials have

not yet been conducted to definitively demonstrate the scope of this effect. Thus, available evidence indicates that increasing dietary calcium intakes may result in reductions in fat mass as well as in blood pressure.

Manipulations of dietary calcium have been repeatedly shown to alter blood pressure in animal models of human essential hypertension. Supplemental dietary calcium lowers blood pressure, whereas restricted calcium diets tend to elevate blood pressure. The mechanisms responsible have not been identified, but numerous possibilities have been proposed. Many of the proposals have attempted to relate dietary calcium to calcium metabolism in vascular smooth muscle and altered vascular tone. Other proposals have focused on neural, hormonal, and renal effects of dietary calcium. In this article, mechanisms through which elevations in extracellular calcium levels might influence intracellular calcium levels are explored. Also examined are the potential roles of calcium regulating hormones, sympathetic nervous system, and electrolyte interactions in modifying blood pressure.

Low dietary intake of calcium has been found to be a risk factor for the development of hypertension in a number of epidemiological studies. Moreover, increased dietary calcium intake has been reported to beneficially influence on blood pressure in both humans and animal models of hypertension, suggesting the relative calcium deficiency may indeed be present in these conditions. The underlying mechanisms for the beneficial effect of positive calcium balance on blood pressure have been a subject for extensive research in essential and experimental hypertension. In this article, the possible beneficial effects of increased dietary calcium intake on central blood pressure control are reviewed.

Whether maintenance of normal calcium homeostasis can afford protection against the development of hypertension in humans has emerged as a controversial area of both clinical and basic cardiovascular disease research. The data that have provoked this debate are derived from epidemiological reports, human studies, animal investigations, and cellular research. Ten published reports have identified an association between greater dietary calcium consumption and lower blood pressure in humans. In both humans and experimental animals with hypertension, several end-organ defects have been identified that are consistent with an inability to maintain external calcium balance. With the provision of supplemental dietary calcium, both humans and experimental models with high blood pressure have reduced their blood pressure. A variety of membrane-associated defects of Ca^{2+} -ATPase-dependent calcium transport have been identified in cells derived from multiple organs of both the hypertensive animal and human. These abnormalities of cellular calcium handling could account for the failure of the

hypertensive subject to appropriately defend its calcium balance. More important, they provide a theoretical mechanism by which calcium, interacting with calmodulin, might favorably modify vascular smooth muscle function and, thereby, peripheral vascular resistance.

Hypertension and magnesium: Magnesium intake of 500 mg/d to 1000 mg/d may reduce blood pressure (BP) as much as 5.6/2.8 mm Hg. However, clinical studies have a wide range of BP reduction, with some showing no change in BP. The combination of increased intake of magnesium and potassium coupled with reduced sodium intake is more effective in reducing BP than single mineral intake and is often as effective as one antihypertensive drug in treating hypertension. Reducing intracellular sodium and calcium while increasing intracellular magnesium and potassium improves BP response. Magnesium also increases the effectiveness of all antihypertensive drug classes. It remains to be conclusively proven that cardiovascular disease such as coronary heart disease, ischemic stroke, and cardiac arrhythmias can be prevented or treated with magnesium intake. Preliminary evidence suggests that insulin sensitivity, hyperglycemia, diabetes mellitus, left ventricular hypertrophy, and dyslipidemia may be improved with increased magnesium intake. Various genetic defects in magnesium transport are associated with hypertension and possibly with cardiovascular disease. Oral magnesium acts as a natural calcium channel blocker, increases nitric oxide, improves endothelial dysfunction, and induces direct and indirect vasodilation.

Magnesium,^[32] plays a role in a number of chronic, disease-related conditions. This article reviews current pertinent literature on magnesium, focusing on hypertension and cardiovascular diseases and implications for relationships with diabetes and metabolic syndrome. A major role for magnesium is in the regulation of blood pressure. While data are not entirely consistent, it does appear that an inverse relationship between magnesium intake and blood pressure is strongest for magnesium obtained from food rather than that obtained via supplements. Hypertension associated with preeclampsia appears to be alleviated when magnesium is administered; in addition, women with adequate intakes of magnesium are less likely to be affected by preeclampsia than those with an inadequate intake. A role for magnesium in other cardiovascular diseases has been noted in that increased magnesium intake may improve serum lipid profiles. Dietary magnesium is also recommended to aid in the prevention of stroke and is important for skeletal growth and development. Magnesium may also play a role in the development of diabetes mellitus, obesity, and metabolic syndrome. There are data from some studies, such as the DASH and PREMIER studies, that suggest that lifestyle changes (including adequate magnesium intake) can

benefit blood pressure control, promote weight loss, and improve chronic disease risk.

Blood pressure is affected by many genetic and environmental factors and their complex interactions. The elucidation of the pathophysiological mechanisms of essential hypertension remains one of the most formidable challenges in medicine today. A large number of pathophysiological abnormalities have been described in essential hypertension such as defects in Na, K and Ca metabolism, increased sympathetic activity, vascular supersensitivity to endogenous neurohumoral constricting agents and decreased sensitivity to endogenous vasodilators. Evidence is accumulating which suggests that Mg,^[33] may play an important role in the prevention and control of hypertension. Mg affects the metabolism of Na, K and Ca and the abnormalities in ion transport reported in experimental hypertension are similar to those reported in experimental Mg deficiency. In experimental studies, Mg has been established as an important regulator of vascular tone. Hypertension has been shown to develop in Mg deficient rats. There are a number of epidemiological studies demonstrating a negative correlation between Mg intake and hypertension. Parenteral treatment with magnesium sulphate is well established as successful in reducing hypertension in preeclampsia. Two recent studies indicate that oral supplementation of Mg can lower blood pressure in patients with essential hypertension. Mg may also be important in drug treatment of hypertension and diuretics may result in enhanced losses of magnesium. There is evidence that Mg deficiency may induce resistance to the effects of antihypertensive agents.

Magnesium,^[34] status has a direct effect upon the relaxation capability of vascular smooth muscle cells and the regulation of the cellular placement of other cations important to blood pressure - cellular sodium:potassium (Na:K) ratio and intracellular calcium ($iCa(2+)$). As a result, nutritional magnesium has both direct and indirect impacts on the regulation of blood pressure and therefore on the occurrence of hypertension. Hypertension occurs when cellular Na:K ratios become too high, a consequence of a high sodium, low potassium diet or, indirectly, through a magnesium deficient state which causes a pseudo potassium deficit. Like wise, magnesium deficiency alters calcium metabolism, creating high $iCa(2+)$, low serum calcium and low urinary calcium states even when calcium intake is adequate. High $iCa(2+)$ and high cellular Na:K ratio both occur when cellular magnesium becomes too low and the Mg-ATP driven sodium-potassium pump and calcium pump become functionally impaired. High $iCa(2+)$ has several vasoconstrictive effects which lead to hypertension, an indirect result of low magnesium status. Dietary calcium is directly proportional to dietary magnesium. Serum magnesium does not reflect true magnesium status as do intracellular magnesium measurements.

Several studies on the effect of calcium on blood pressure need these added considerations of magnesium status to fully understand the impact of the Mg:Ca ratio as the primary cause of hypertension and other aspects of Syndrome X. Magnesium supplementation above 15 mmol per day are required to normalize high blood pressure in unmedicated hypertensive patients while 15 mmol per day will lower high blood pressure in patients treated with anti-hypertensive medications. In most humans, healthy blood pressure depends upon a balance of both Na:K and Mg:Ca ratios at both cellular and whole body levels which, in turn, require adequate, long-term intakes of nutritional magnesium. The knowledge that low magnesium causes imbalance in both cellular and physiological calcium widens our view of the studies showing hypertensives have abnormal calcium metabolism.

MATERIALS AND METHODS

The present Prospective observational study was done in Department of general medicine in tertiary care hospital from June 2022 to June 2023. All patients with hypertension above 18 yrs were enrolled. All the necessary investigations like CBC, RFT, thyroid profile, LFT, USG abdomen were done. Special investigations like serum calcium and magnesium were sent.

Inclusion Criteria

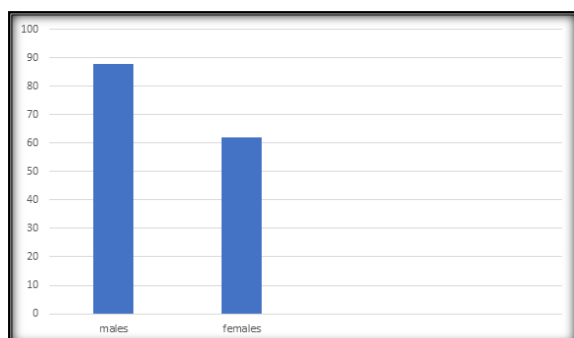
1. Age more than 18 years.
2. Patients who were diagnosed hypertensive.
3. Patients who gave consent for the study.

Exclusion Criteria

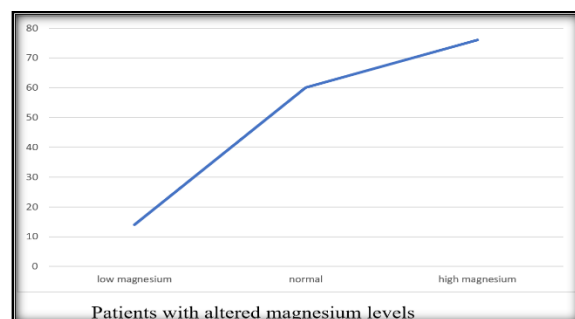
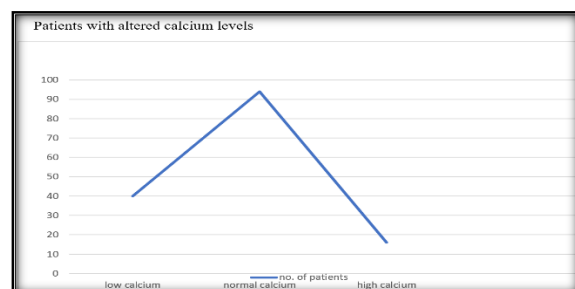
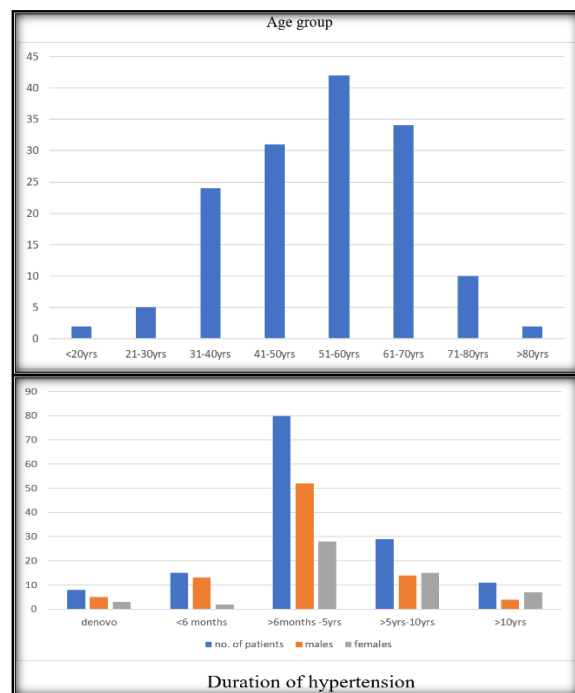
1. Patients with previous hypocalcemic disorders.
2. Patients with previous hypomagnesemic disorders
3. Patients on drugs causing hypertension. Complete detailed history was taken and general physical examination is done for all patients.

The statistical software SPASS was used to analyse the data and Microsoft word and excel have been used to generate graphs, figure etc.

RESULTS

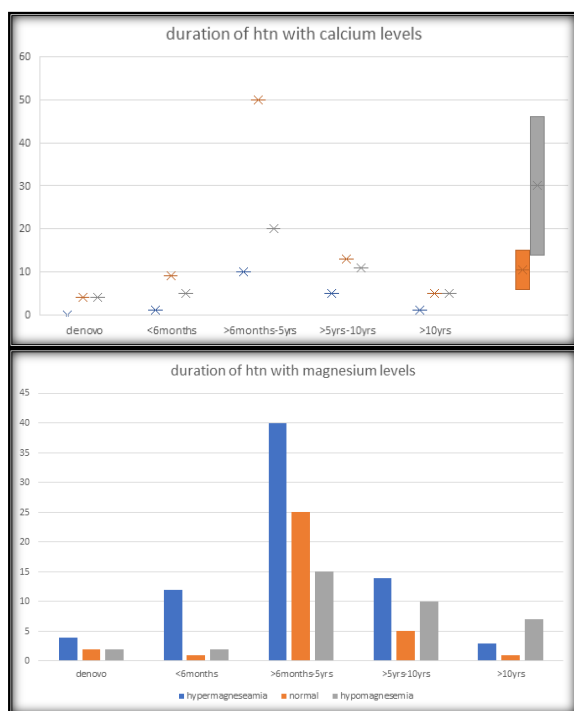


Gender Distribution



SUMMARY OUTPUT									
Regression Statistics									
Multiple R	0.976588								
R Square	0.953724								
Adjusted R	0.938299								
Standard E	7.41165								
Observations	5								
ANOVA									
	df	SS	MS	F	Significance F				
Regression	1	3396.402	3396.402	61.82858	0.004285				
Residual	3	164.7977	54.93256						
Total	4	3561.2							
Coefficients									
	Coefficients	Standard Error	t Stat	P-value	Lower 95%	Upper 95%	Lower 95.0%	Upper 95.0%	
Intercept	4.780347	4.490335	1.064586	0.365149	-9.5099	19.0706	-9.5099	19.0706	
X Variable	7.00578	0.890968	7.863115	0.004285	4.170324	9.841237	4.170324	9.841237	

P value for duration of htn with increased calcium levels is <0.5



P value for duration of htn with decreased magnesium levels <0.5

SUMMARY OUTPUT									
Regression Statistics									
Multiple R	0.882204								
R Square	0.778284								
Adjusted R	0.704379								
Standard E	16.22318								
Observations	5								
ANOVA									
	df	SS	MS	F	Significance F				
Regression	1	2771.625	2771.625	10.53082	0.047665				
Residual	3	789.5749	263.1916						
Total	4	3561.2							
Coefficients									
	Coefficients	Standard Error	t Stat	P-value	Lower 95%	Upper 95%	Lower 95.0%	Upper 95.0%	
Intercept	-5.60586	12.79627	-0.43809	0.69095	-46.3293	35.11759	-46.3293	35.11759	
X Variable	4.750814	1.463986	3.245123	0.047665	0.091758	9.409871	0.091758	9.409871	

DISCUSSION

In our study there was male predominance in the ratio 1.4:1. there were more number of patients in the age group 51-60 years. According to K. Sudhakar et al,^[35] The mean total serum calcium levels were significantly ($p < 0.01$) decreased in males and females in hypertensive group when compared with normotensive controls

In our study there were 45 patients with decreased calcium levels. p value was calculated < 0.001 in correlation with Sudhakar et al study.^[35]

Fu Y, Wang S., et al,^[36] in their study suggested that that the hypertensive group consistently demonstrated a significant decreased activity of ATPase studied, with significantly lower plasma calcium levels $P < 0.01$.

Strazzullo P et al,^[37] studied several of the biochemical abnormalities of calcium metabolism

and were able to detect a significant reduction in total serum calcium levels in hypertensive subjects with essential hypertension.

Possible mechanisms may be that low plasmatic calcium concentration could stimulate the release of parathyroid hormone (PTH),^[38] and parathyroid hypertensive factor,^[39] foster the synthesis of calcitriol,^[40] and activate RAAS system,^[41] which in return increase intracellular calcium concentration and lead to vasoconstriction, vascular resistance, and high blood pressure.^[42] Apart from this, both angiotensin II and PTH could provoke aldosterone secretion from adrenal gland,^[43] which could upregulate epithelial sodium channels (ENaC) in the principal cells of the collecting duct in the kidney, increase apical membrane permeability for Na^+ , facilitate Na^+ , and water reabsorption and thus, increase blood pressure.^[44] Collectively, the calcium deficiency hypothesis of hypertension populated and calcium supplementation was advocated in lowering blood pressure and delaying the onset of hypertension.

The mechanisms underlying the observed association between serum calcium and hypertension remain uncertain. Serum calcium may directly affect vasoconstriction by the influx of calcium into the smooth muscle of the artery, which enhances muscle contracture, increases vascular resistance, and therefore, leads to the development of hypertension.^[45] Additionally, calcium could indirectly promote hypertension by alteration in the extracellular binding of calcium,^[46] inducing insulin resistance,^[47] and interacting with other cations, especially sodium and potassium.⁴⁸ Another possible mechanism lies in that subtle alterations in intracellular calcium may affect the secretion and action of hormones, such as the pressor action of catecholamines, angiotensin II, or aldosterone, which may target the blood pressure control centers and increase blood pressure levels. Moreover, calcium could mediate PTH release and several previous studies have found a positive correlation between PTH concentration and blood pressure.^[49,50] In our study there were 36 patients with decreased magnesium levels. it was observed in our study that magnesium levels decreased in more number of patients as the duration of hypertension increased. p value < 0.04 .

Resnick et al,^[51] reported that in essential hypertension, there is a continuous negative correlation of serum magnesium with plasma renin activity. Ferdousi et al,^[52] reported in their study that the serum magnesium level (mg/dl) was significantly lower in 30 offsprings of essential hypertensive parents when compared to the 30 age- and sex-matched offsprings of normotensive parents $P < 0.01$. In the present study also, there is a negative correlation exists between serum magnesium and blood pressure of hypertensives with longer duration was significantly higher than the normotensive subjects. The results are similar to the findings of the study conducted by Ohira et al.^[53] In

his study, he found that there exists an inverse relationship between serum magnesium with blood pressure.

CONCLUSION

1. Hypertension is influenced by various risk factors.
2. It was observed that serum calcium was low in hypertensives
3. It was observed that serum magnesium was significantly lower in hypertensives
4. It was observed that high serum calcium values varied with the duration of hypertension.
5. It was observed that low serum magnesium values varied with duration of hypertension.

REFERENCES

1. Reed D, McGee D, Yano K, Hankin J. Diet, blood pressure, and multicollinearity. *Hypertension*.1985; 7:405–410.[LinkGoogle Scholar](#)
2. Joffres MR, Reed DM, Yano K. Relationship of magnesium intake and other dietary factors to blood pressure: the Honolulu Heart Study. *Am J Clin Nutr*.1987; 45:469–475.[CrossrefMedlineGoogle Scholar](#)
3. Intersalt Cooperative Research Group. Intersalt: an international study of electrolyte excretion and blood pressure. Results for 24 hour urinary sodium and potassium excretion. *BMJ*.1988; 297:319–328.[CrossrefMedlineGoogle Scholar](#)
4. Kesteloot H, Joossens JV. Relationship of dietary sodium, potassium, calcium, and magnesium with blood pressure: Belgian Interuniversity Research on Nutrition and Health. *Hypertension*.1988; 12:594–599.[LinkGoogle Scholar](#)
5. Sacks FM, Kass EH. Low blood pressure in vegetarians: effects of specific foods and nutrients. *Am J Clin Nutr*.1988; 48:795–800.[CrossrefMedlineGoogle Scholar](#)
6. Khaw K-T, Barrett-Connor E. The association between blood pressure, age, and dietary sodium and potassium: a population study. *Circulation*.1988; 77:53–61.[CrossrefMedlineGoogle Scholar](#)
7. Witteman JC, Willett WC, Stampfer MJ, Colditz GA, Sacks FM, Speizer FE, Rosner B, Hennekens CH. A prospective study of nutritional factors and hypertension among US women. *Circulation*.1989; 80:1320–1327.[CrossrefMedlineGoogle Scholar](#)
8. Iso H, Terao A, Kitamura A, Sato S, Naito Y, Kiyama M, Tanigaki M, Iida M, Konishi M, Shimamoto T, Komachi Y. Calcium intake and blood pressure in seven Japanese populations. *Am J Epidemiol*.1991; 133:776–783.[CrossrefMedlineGoogle Scholar](#)
9. Ascherio A, Rimm EB, Giovannucci EL, Colditz GA, Rosner B, Willett WC, Sacks FM, Stampfer MJ. A prospective study of nutritional factors and hypertension among US men. *Circulation*.1992; 86:1475–1484.[CrossrefMedlineGoogle Scholar](#)
10. Whelton PK. Potassium and blood pressure. In: Izzo JL, Black HR, eds. *Hypertension Primer*. Council on High Blood Pressure Research. Dallas, Tx: American Heart Association; 1993:170–172.[Google Scholar](#)
11. Simon JA, Obarzanek E, Daniels SR, Frederick MM. Dietary cation intake and blood pressure in black girls and white girls. *Am J Epidemiol*.1994; 139:130–140.[CrossrefMedlineGoogle Scholar](#)
12. Cappuccio FP, Elliott P, Allender PS, Follman DA, Cutler JA. Epidemiologic association between dietary calcium intake and blood pressure: a meta-analysis of published data. *Am J Epidemiol*.1995; 142:935–945.[CrossrefMedlineGoogle Scholar](#)
13. Pryer J, Cappuccio FP, Elliott P. Dietary calcium and blood pressure: a review of the observational studies. *J Hum Hypertens*.1995; 9:597–604.[MedlineGoogle Scholar](#)
14. Ma J, Folsom AR, Melnick SL, Eckfeldt JH, Sharrett AR, Nabulsi AA, Hutchinson RG, Metcalf PA, for the Atherosclerosis Risk in Communities study group. Associations of serum and dietary magnesium with cardiovascular disease, hypertension, diabetes, insulin, and carotid arterial wall thickness: the ARIC study. *J Clin Epidemiol*.1995; 48:927–940.[CrossrefMedlineGoogle Scholar](#)
15. Ascherio A, Hennekens CH, Willett WC, Sacks FM, Rosner B, Manson J, Witteman J, Stampfer MJ. A prospective study of nutritional factors, blood pressure, and hypertension among US women. *Hypertension*.1996; 27:1065–1072.[CrossrefMedlineGoogle Scholar](#)
16. Cappuccio FP, MacGregor GA. Does potassium supplementation lower blood pressure? A meta-analysis of published trials. *J Hypertens*.1991; 9:465–473.[CrossrefMedlineGoogle Scholar](#)
17. Allender PS, Cutler JA, Follmann D, Cappuccio FP, Pryer J, Elliott P. Dietary calcium and blood pressure: a meta-analysis of randomized clinical trials. *Ann Intern Med*.1996; 124:825–831.[CrossrefMedlineGoogle Scholar](#)
18. Bucher HC, Cook RJ, Guyatt GH, Lang JD, Cook DJ, Hatala R, Hunt DL. Effects of dietary calcium supplementation on blood pressure: a meta-analysis of randomized controlled trials. *JAMA*.1996; 275:1016–1022.[CrossrefMedlineGoogle Scholar](#)
19. Cappuccio FP, Markandu ND, Beynon GW, Shore AC, Sampson B, MacGregor GA. Lack of effect of oral magnesium on high blood pressure: a double blind study. *BMJ*.1985; 291:235–238.[CrossrefMedlineGoogle Scholar](#)
20. Henderson DG, Schierup J, Schott T. Effect of magnesium supplementation on blood pressure and electrolyte concentrations in hypertensive patients receiving long term diuretic treatment. *BMJ*.1986; 293:664–665.[CrossrefMedlineGoogle Scholar](#)
21. Motoyama T, Sano H, Fukuzaki H. Oral magnesium supplementation in patients with essential hypertension. *Hypertension*.1989; 13:227–232.[LinkGoogle Scholar](#)
22. Zemel PC, Zemel MB, Urberg M, Douglas FL, Geiser R, Sowers JR. Metabolic and hemodynamic effects of magnesium supplementation in patients with essential hypertension. *Am J Clin Nutr*.1990; 51:665–669.[CrossrefMedlineGoogle Scholar](#)
23. Patki PS, Singh J, Gokhale SV, Bulakh PM, Shrotri DS, Patwardhan B. Efficacy of potassium and magnesium in essential hypertension: a double blind, placebo controlled, crossover study. *BMJ*.1990; 301:521–523.[CrossrefMedlineGoogle Scholar](#)
24. Lind L, Lithell H, Pollare T, Ljunghall S. Blood pressure response during long-term treatment with magnesium is dependent on magnesium status: a double-blind, placebo-controlled study in essential hypertension and in subjects with high-normal blood pressure. *Am J Hypertens*.1991; 4:674–679.[CrossrefMedlineGoogle Scholar](#)
25. Widman L, Wester PO, Stegmayr BK, Wirell M. The dose-dependent reduction in blood pressure through administration of magnesium: a double-blind placebo controlled cross-over study. *Am J Hypertens*.1993; 6:41–45.[CrossrefMedlineGoogle Scholar](#)
26. Wirell MP, Wester PO, Stegmayr BG. Nutritional dose of magnesium in hypertensive patients on beta blockers lowers systolic blood pressure: a double-blind, cross-over study. *J Intern Med*.1994; 236:189–195.[CrossrefMedlineGoogle Scholar](#)
27. Witteman JC, Grobbee DE, Derckx FH, Bouillon R, de Bruijn AM, Hofman A. Reduction of blood pressure with oral magnesium supplementation in women with mild to moderate hypertension. *Am J Clin Nutr*.1994; 60:129–135.[CrossrefMedlineGoogle Scholar](#)
28. Sacks FM, Brown LE, Appel L, Borhani NO, Evans D, Whelton P. Combinations of potassium, calcium, and magnesium supplements in hypertension. *Hypertension*.1995; 26:950–956.[CrossrefMedlineGoogle Scholar](#)

29. The Trials of Hypertension Prevention Collaborative Research Group. The effects of nonpharmacologic interventions on blood pressure of persons with high normal levels: results of the Trials of Hypertension Prevention, Phase 1. *JAMA*.1992; 267:1213–1220.CrossrefMedlineGoogle Scholar
30. Yamamoto ME, Applegate WB, Klag MJ, Borhani NO, Cohen JD, Kirchner KA, Lakatos E, Sacks FM, Taylor JO, Hennekens CH, for the TOHP Collaborative Research Group. Lack of blood pressure effect with calcium and magnesium supplementation in adults with high-normal blood pressure: results from Phase I of the Trials of Hypertension Prevention (TOHP). *Ann Epidemiol*.1995; 5:96–107.CrossrefMedlineGoogle Scholar
31. The role of magnesium in hypertension and cardiovascular disease.Houston M.J *Clin Hypertens* (Greenwich). 2011 Nov;13(11):843-7. doi: 10.1111/j.1751-7176.2011.00538.x. Epub 2011 Sep 26.
32. Magnesium in hypertension, cardiovascular disease, metabolic syndrome, and other conditions: a review.Champagne CM.*Nutr Clin Pract*. 2008 Apr-May;23(2):142-51. doi: .1177/0884533608314533.
33. The role of magnesium in the prevention and control of hypertension.Ryan MP, Brady HR.
34. *Ann Clin Res*. 1984;16 Suppl 43:81-8.34.[Magnesium and hypertension].Rosanoff A.*Clin Calcium*. 2005 Feb;15(2):255-60.
35. K. Sudhakar, M. Sujatha, S. Ramesh Babu, P. Padmavathi and P. P. Reddy, serum calcium in patients with essential hypertension and their first degree relatives., *Indian Journal of Clinical Biochemistry*, 2004, 19(1) 21-23.
36. Fu, Y., Wang, S., Lu, Z., Li, H. and Li, S. (1998).Erythrocyte and plasma Ca²⁺, Mg²⁺ and cell membrane adenosine triphosphatase activity in patients with essential hypertension. *Chin. Med. J. (Engl)* 111(2),147-149
37. Strazzullo P, Nunziata V, Cirillo M, Giannattasio R, Mancini M. Abnormalities of calcium metabolism in essential hypertension. *Clin Sci* 1983;65:359-363
38. Goltzman D, Mannstadt M, Marcocci C. Physiology of the calcium-parathyroid hormone-vitamin D axis. *Front Horm Res*. (2018) 50:1–13. 10.1159/000486060 [PubMed] [CrossRef] [Google Scholar]
39. Pang PK, Shan JJ, Lewanczuk RZ, Benishin CG. Parathyroid hypertensive factor and intracellular calcium regulation. *J Hypertens*. (1996) 14:1053–60. 10.1097/00004872-199609000-00002 [PubMed] [CrossRef] [Google Scholar]
40. Areco V, Rivoira MA, Rodriguez V, Marchionatti AM, Carpentieri A, Tolosa de Talamoni N. Dietary and pharmacological compounds altering intestinal calcium absorption in humans and animals. *Nutr Res Rev*. (2015) 28:83–99. 10.1017/S0954422415000050 [PubMed] [CrossRef] [Google Scholar]
41. Resnick LM, Laragh JH, Sealey JE, Alderman MH. Divalent cations in essential hypertension relations between serum ionized calcium, magnesium, and plasma renin activity. *N Engl J Med*. (1983) 309:888–91. 10.1056/NEJM198310133091504 [PubMed] [CrossRef] [Google Scholar]
42. Villa-Etchegoyen C, Lombarte M, Matamoros N, Belizán JM, Cormick G. Mechanisms involved in the relationship between low calcium intake and high blood pressure. *Nutrients*. (2019). 11:1112. 10.3390/nu11051112 [PMC free article] [PubMed] [CrossRef] [Google Scholar]
43. Barkan A, Marilus R, Winkelsberg G, Yeshurun D, Blum I. Primary hyperparathyroidism: possible cause of primary hyperaldosteronism in a 60-year-old woman. *J Clin Endocrinol Metabol*. (1980) 51:144–7. 10.1210/jcem-51-1-144 [PubMed] [CrossRef] [Google Scholar]
44. Maniero C, Fassina A, Guzzardo V, Lenzini L, Amadori G, Pelizzo MR, et al.. Primary hyperparathyroidism with concurrent primary aldosteronism. *Hypertension*. (2011). 58:341–6. 10.1161/HYPERTENSIONAHA.111.173948 [PubMed] [CrossRef] [Google Scholar]
45. Touyz RM, Alves-Lopes R, Rios FJ, Camargo LL, Anagnostopoulou A, Arner A, et al.. Vascular smooth muscle contraction in hypertension. *Cardiovasc Res*. (2018) 114:529–39. 10.1093/cvr/cvy023 [PMC free article] [PubMed] [CrossRef] [Google Scholar]
46. Zidek W, Vetter H. Calcium and primary hypertension. *Nephron*. (1987) 47(Suppl 1):13–20. 10.1159/000184546 [PubMed] [CrossRef] [Google Scholar]
47. Kunutsor SK, Laukkanen JA. Circulating active serum calcium reduces the risk of hypertension. *Eur J Prevent Cardiol*. (2017) 24:239–43. 10.1177/2047487316681174 [PubMed] [CrossRef] [Google Scholar]
48. Ahlström T, Hagström E, Larsson A, Rudberg C, Lind L, Hellman P. Correlation between plasma calcium, parathyroid hormone (PTH) and the metabolic syndrome (MetS) in a community-based cohort of men and women. *Clin Endocrinol*. (2009) 71:673–8. 10.1111/j.1365-2265.2009.03558.x [PubMed] [CrossRef] [Google Scholar]
49. Snijder MB, Lips P, Seidell JC, Visser M, Deeg DJ, Dekker JM, et al.. Vitamin D status and parathyroid hormone levels in relation to blood pressure: a population-based study in older men and women. *J Intern Med*. (2007) 261:558–65. 10.1111/j.1365-2796.2007.01778.x [PubMed] [CrossRef] [Google Scholar]
50. Resnick LM, Laragh JH, Sealey JE, Alderman MH. Divalent cations in essential hypertension. Relations between serum ionized calcium, magnesium, and plasma renin activity. *N Engl J Med* 1983;309:888-91.
51. Ferdousi S, Sultana N, Rahman MH, Khanam R, Howlader M, Arslan M, et al. Serum and erythrocyte magnesium levels in offsprings of essential hypertensive parents. *Bangladesh J Med Biochem* 2013;5:40-3.
52. Ohira T, Peacock JM, Iso H, Chambless LE, Rosamond WD, Folsom AR, et al. Serum and dietary magnesium and risk of ischemic stroke: The atherosclerosis risk in communities study. *Am J Epidemiol* 2009;169:1437-4.