International Journal of Life Sciences, Biotechnology and Pharma Research Vol. 14, No. 3, March 2025 Online ISSN: 2250-3137
Print ISSN: 2977-0122

DOI: 10.69605/ijlbpr_14.3.2025.232

ORIGINAL RESEARCH

Investigating the Impact of Low Serum Magnesium Levels on Coronary Risk in Indian Patients

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Received: 08 February, 2025 Accepted: 24 February, 2025 Published: 12 March, 2025

ABSTRACT

Introduction: Magnesium is a vital component with a wide range of biological roles in the heart. In order to assess the relationship between dietary and serum magnesium and cardiovascular risk factors, three hundred individuals over the age of 30 who had known cardiovascular disease were examined. **Materials and Methods:** Serum magnesium levels were used to divide the patients into three groups: ≤1.6 (Group 1), >1.6-2.6 (Group 2), and >2.6 mg/dl (Group 3). Dietary magnesium consumption was used to divide the patients into two groups: ≤350 mg/day (Group 1) and >350 mg/day (Group 2), respectively. **Results:** The study included three hundred individuals (M: 226; F: 94, ages 25–92) with known cardiovascular disease. Comparison of cardiovascular risk variables based on serum magnesium levels has been assessed. The patients were 61.8 ± 13.3 years old on average. Males and females did not vary in age (M: 61.94 ± 13.2; F: 62.02 ± 13.8; P = 0.11). **Conclusions:** Among individuals with coronary artery disease, hypomagnesaemia and low magnesium consumption in the diet are closely linked to cardiovascular risk factors. Therefore, taking supplements of magnesium may help lower the risk of cardiovascular disease.

Key words: Cardiovascular disease, dietary magnesium, dyslipidemia, serum magnesium

INTRODUCTION

Magnesium is a vital component with a wide range of biological roles in the heart. Magnesium controls the metabolic regulation of energy-dependent cytoplasmic and mitochondrial pathways, modulates the transmembrane transport of calcium, sodium, and potassium, regulates contractile proteins, controls oxidative-phosphorylation processes, and influences DNA and protein synthesis at the subcellular level. minor modifications to the extracellular magnesium.[1,2] Magnesium deficiency can raise intracellular sodium and calcium concentrations by acting as a cofactor in the sodium-potassium ATPase pump. This can increase peripheral resistance, promote vasoconstriction, attenuate responses to vasodilators, and enhance artery reactivity to vasoconstrictor agents, all of which can raise blood pressure. Additionally, the action of the extracellular enzymes lipoprotein lipase, cholesterol acyl transferase, and lecithin depends on magnesium.[3] Heart failure, hypertension, myocardial infarction, and atherosclerosis have all been connected to serum magnesium levels.[4], but it wasn't a stand-alone risk factor for cardiac patients' deaths.[5] Numerous research have shown a connection between risk factors and magnesium, however the majority of these used animals.[6– 10] Few human research have examined cardiovascular disease in connection to risk variables, suggesting that insufficient magnesium may be a contributing factor to atherosclerosis, hypertension, and diabetes mellitus.[11– 13] Due to a number of confounding dietary variables, the relationship between food consumption and cardiovascular disease is especially uncertain.[11] In order to assess the relationship between dietary and blood magnesium levels and conventional cardiovascular risk factors in individuals with established coronary artery disease, this research was conducted.

MATERIALS AND METHODS

This research comprised 320 participants over 30 with established coronary heart disease. Sequentially, patients who were evaluated for chest discomfort in the cardiology

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department and whose angiography results were positive were chosen for the study. Chronic renal illness, hepatic dysfunction, established rheumatological or endocrine disorders, or persistent infections were all grounds for exclusion. A questionnaire that contained information on physical activity and smoking was used to interview each patient. We measured hip circumference, waist circumference, height, and weight. WHR and BMI were computed. Information on the clinical history of diabetes mellitus, hypertension, and antihypertensive and oral hypoglycemic drugs was also obtained.

Trained personnel conducted interviews to deliver questionnaires and nutrition evaluations. Volume measurements, circular measures, numbers, and linear measures were used to estimate the portion size. A two-day mean recall was calculated. Interviewers supplied frequently used serving plates, bowls, cutlery, cups, and spoons to aid participants in estimating portion sizes. If it was not possible to provide measurements Any anomalous intake was noted on the recall, and we documented in three sizes: small, medium, and big. We reported values per 100 g of edible food product for each nutrient using a standardised unit of measurement. Neither the food habits nor the lifestyle of these people changed much.[14] Following a 14-hour fast, fasting blood samples were taken. LDL and VLDL were computed using Friedewald's formula, whereas total cholesterol, triglycerides, HDL, LDL, and VLDL cholesterol were assessed using CHOD PAP, LIP/GK, and the enzymatic clearance technique, respectively. For every parameter, the intraprecision was 2% and the interassay was 3.84%. The Xylidyl blue technique, which is unaffected by calcium, is used to detect magnesium.[15] Group 1 consisted of patients with serum magnesium levels ≤ 1.6 mg/dl (n = 176, 58.6%), Group 2 of patients with serum magnesium levels > 1.6-2.6 mg/dl (n = 102, 34%), and Group 3 of patients with serum magnesium levels > 2.6 mg/dl (n = 26, 8.6%). Additionally, two groups were created based on the Recommended Dietary Allowance of magnesium: Group 1 had dietary intake of magnesium ≤ 350 mg/day (n = 186, 62%), and Group 2 (n = 114, 38%) had dietary intake of magnesium levels > 350 mg/day. The Deenanath Mangeshkar Hospital's institutional ethics committee gave its approval to the research. Every participant gave their informed permission.

STATISTICAL METHOD

EPI INFO 3.5.3 was used to do statistical analysis (CDC, Atlanta, GA, USA). Unless otherwise noted, the data were shown as a number (%) or mean ± SD. The Student's t-test was used to evaluate all parametric data. The Kruskal--Wallis test was used if the results of Barlett's chi-square test for equality of population variances were less than 0.05. The chi-square test was used to evaluate all nonparametric data. Multiple regression analysis and Pearson's correlation were used to evaluate the degree of association between cardiovascular risk variables and blood magnesium levels. Statistical significance was defined as a P value of less than 0.05.

RESULTS

The study included three hundred individuals (M: 226; F: 94, ages 25– 92) with known cardiovascular disease. Table 1 presents a comparison of cardiovascular risk variables based on serum magnesium levels. The patients were 61.8 ± 13.3 years old on average. Males and females did not vary in age (M: 61.94 ± 13.2; F: 62.02 ± 13.8; P = 0.11).

Table 1: Cardiovascular risk factor in groups according to serum magnesium levels

Parameters	Group 1 (≤ 1.6 mg/dl) median (range)	Group 2 (> 1.6-2.6 mg/dl) median (range)	Group 3 (> 2.6 mg/dl) median (range)	*P* value
Age	64 (26-93), M: 64 (25-92) F: 64 (35-87)	64.4 (26-90) M: 65 (26-86) F: 66 (26-90)	60 (33-76) M:61 (44-76) F: 58 (33-72)	0.6370
P value		0.9844*	0.3351¶, 0.3906#	
Sex	Male:68.5% (123) Female: 33.3% (60)	Male: 78.4% (83) Female: 23.3% (26)	Male: 81.8% (21) Female: 18.1% (7)	0.1204
P value		0.1076*	0.2645¶, 0.8686#	
BMI	28.2 (21.01-40.3) M: 29.1 (21.0-40.3) F: 28.1 (21.2-37.7)	27.7 (20.2-38.6) M: 27.6 (20.2-38.6) F: 28.6 (21.4-38.4)	28.3 (23.17-36.01) M: 28.5 (23.5-36.0) F: 26.8 (23.1-32.3)	0.849
P value		0.477*	0.9171¶, 0.6422#	
Waist hip ratio	0.93 (0.71-2.06) M: 0.93 (0.76-2.06) F: 0.91 (0.71-2.01)	0.93 (0.71-2.17) M: 0.93 (0.71-2.17) F: 0.96 (0.77-2.01)	0.94 (0.82-2.01) M: 0.94 (0.82-2.01) F: 0.94 (0.86-0.96)	0.957
P value		0.77*	0.7819¶, 0.7709#	
Cholesterol	174.4 (92-344)	162 (92-332)	166 (119-235)	<0.0001
P value		<0.00001*	0.0398¶, 0.7594#	
Triglyceride	190 (72-342)	145 (65-299)	146 (98-200)	<0.0001
P value		<0.00001*	<0.00001¶, 0.2347#	
HDL Cholesterol	33 (20-55)	47 (26-59)	49 (32-59)	<0.0001

DOI: 10.69605/ijlbpr_14.3.2025.232

P value		<0.00001*	<0.00001¶, 0.0607#	
LDL Cholesterol	99.8 (13.5-274.7)	87 (15.3-264)	85.4 (40-163.3)	<0.0001
P value		<0.00001*	0.0001¶, 0.8318#	
VLDL Cholesterol	39(15.1-69.1)	30 (13.7-60.5)	30.1 (20.7-39)	<0.0001
P value		<0.00001*	0.0044¶, 0.2059#	
Diabetes	53.7% (95)	20.1% (26)	7.3 %(9)	<0.0001
P value		<0.00001*	0.2183¶, 0.3546#	
Hypertension	75.8% (133)	51% (503)	23.6% (7)	0.0041
P value		<0.0001*	<0.0001¶, 0.0362#	
Diabetes and hypertension	41.2% (73)	17.6% (19)	23.6% (7)	0.0001
P value		<0.00001*	0.1709¶, 0.747#	
Smoking	41.2 % (73)	33.3% (35)	32.7% (9)	0.3606
P value		0.2309*	0.590¶, 0.814#	
Less physical activity	39.2% (69)	39.1 % (41)	46.4 % (12)	0.8012
P value		0.9046*	0.676¶, 0.684#	
**P value between Group-1 and Group-2. ¶P value between Group-1 and Group-3. #P value between Group-2 and Group-3*				

DISCUSSION

This is the first research to examine the effect of dietary and serum magnesium in cardiovascular patients using angiographical evidence from India. In the current research, 62% of patients were consuming dietary magnesium below the recommended dietary intake (350 mg/day), and more than half of patients (58.6%) had low magnesium levels. Others have reported hypomagnesaemia in individuals with acute myocardial infarction and cardiovascular disease ranging from 19% to 53%.[16,17] Few studies have shown the significant impact hypomagnesaemia plays in changing cardiovascular disease risk factors such as hypertension, diabetes, and dyslipidaemia.[11– 13] The fact that there are noticeably more participants with DM, hypertension, and dyslipidaemia in the lowest category in the current research suggests that hypomagnesaemia is prevalent in these disorders. Dietary magnesium showed a similar association. Serum magnesium levels were lower in all of these risk variables, with the exception of those with isolated hypertension. Serum magnesium levels in hypertension individuals have not been shown to vary in other investigations either.[18, 19] After controlling for age, sex, and anthropometric characteristics in multiple regression analysis, serum magnesium continued to have a substantially negative connection with cardiovascular risk variables. Serum magnesium levels may be protective against the development of hypertension, since none of the participants in group three had hypertension. The idea that a higher magnesium intake helps reduce hypertension and cardiovascular disease was backed by several epidemiological and clinical studies.[20– 23] However, there was no significant correlation between dietary magnesium consumption and incident hypertension in either males or females in a six-year follow-up ARIC (atherosclerosis risk in communities) research, which followed up on healthy individuals for incident hypertension.[24] The relationship between hypomagnesaemia and diabetes mellitus has been the subject of several investigations. Despite contradictory findings from small-group patient studies, there is substantial evidence from human and animal research that DM lowers tissue and plasma magnesium levels [25,26].[25, 27] In one investigation, the diabetics' mean plasma magnesium content was much lower than the control group's [26]. According to the "Study of Health in Pomerania," hypomagnesaemia was one of the best indicators of LVM growth during the next five years.[28] Furthermore, experimental hypomagnesaemia suppresses the action of prostacyclin receptors, resulting in an imbalance between the effects of prostacyclin and thromboxane. This imbalance has been implicated as a contributing factor to the development of diabetic vascular disease.[29, 30] In the current investigation, patients with hypomagnesaemia had considerably lower HDL cholesterol and significantly higher total cholesterol, triglycerides, VLDL, and LDL cholesterol. Except for HDL, which found a positive association with blood magnesium levels, other lipid metrics showed a negative correlation with serum magnesium levels. Other research have produced similar findings [11,13], but just as many have not been able to prove this connection.[12, 31] Research involving patients with DM or metabolic syndrome has shown that those with low magnesium levels had greater levels of total cholesterol and triglycerides [33,34] but lower levels of HDL-cholesterol [32– 34]. [34] When compared to the general population, other research that looked at blood magnesium levels found a favourable link with HDL cholesterol, triglycerides, LDL cholesterol, and

DOI: 10.69605/ijlbpr_14.3.2025.232

total cholesterol. [35] A possible link between low ionised magnesium [33,34,36] or total serum magnesium [32,34] and an atherogenic lipid profile, which includes low serum HDL-cholesterol [16,17], high total cholesterol [34,36], and high triglycerides [32– 34], has been documented in individuals with metabolic syndrome, suggesting a possible role for magnesium in the pathophysiology of CVD.[32, 34] Dietary magnesium and total cholesterol, triglycerides, LDL, HDL, and VLDL were shown to be similarly correlated. There has been evidence of a similar correlation between dietary magnesium and lipid levels. Across all magnesium levels, total cholesterol tended to decline with increased consumption [37] and was negatively correlated with HDL cholesterol.[11] In comparison to other groups, patients with hypomagnesaemia also consumed less protein, dietary fibre, calcium, potassium, and folic acid, which may possibly be a contributing factor to other risk factors. The study's primary drawback was the lack of long-term follow-up. Additionally, group 3 had fewer instances and a male preponderance. This gender disparity may be explained by the fact that we have taken consecutive instances and that CVD is a condition that is more common in men.

CONCLUSION

Hypomagnesaemia is seen in half of people with cardiovascular disease. Serum and dietary magnesium levels are highly correlated. Dietary and serum magnesium levels are closely linked to cardiovascular risk factors as hypertension, diabetes, and dyslipidaemia. There aren't many research showing that taking magnesium supplements improves the atherogenic lipid profile. Therefore, taking supplements of magnesium may help lower the prevalence of cardiovascular disease in our society.

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