



Serum Magnesium Level and Dietary Magnesium Intake in Patients with Hypertension-Related Complications

Buse Bakır¹ , Habibe Şahin² , Ali Doğan³

ABSTRACT

Cite this article as:
Bakır B, Şahin H, Doğan A. Serum Magnesium Level and Dietary Magnesium Intake in Patients with Hypertension-Related Complications. Erciyes Med J 2021; 43(5): 482-6.

A summary of the study was presented as a 'poster presentation' at the ESPEN Congress in Madrid, Spain on 1-4 September 2018

Objective: The objective of this study is to evaluate serum magnesium levels, dietary magnesium intake, and their relationships with hypertensive complications in adults with hypertension (HT).

Materials and Methods: Patients with HT for ≥ 10 years were divided into two groups for evaluation. Although the case group comprised 34 patients having at least one of the hypertensive complications of atherosclerosis, heart failure, and renal failure, the control group comprised 34 patients with HT without complications. In addition to demographic information, anthropometric measurements (body weight, height, waist circumference [WC]), and medical information of the patients were collected, dietary information was acquired via a three-day food record.

Results: Dietary magnesium intake was similar in both the groups; however, both values were below the recommended daily intake. The case group had significantly lower serum magnesium level than the control group. We found no correlations between serum magnesium and dietary magnesium intake level. Low serum magnesium level was a possible risk factor for complications (OR: 9.02, 95% CI: 3.00–27.10). Serum magnesium concentration was negatively correlated with the duration of HT, WC, fasting blood glucose; however, it positively correlated with the estimated glomerular filtration rate. Although the mean body mass index levels were similar, the mean WC was higher in the case group than in the control group.

Conclusion: In conclusion, this study's results clearly showed insufficient dietary magnesium intake and lower serum magnesium levels in patients with hypertensive complications. Serum magnesium level was also found to be correlated with some parameters closely related to the complications of HT.

Keywords: Hypertension, complication, diet, magnesium, waist circumference

INTRODUCTION

Hypertension (HT) is an important public health challenge and one of the leading causes of cardiovascular deaths worldwide (1). In addition to antihypertensive medications, nutritional habits also play an important role in the management of HT. The nutrition plan "Dietary Approaches to Stop Hypertension (DASH)" has been shown to be effective in controlling blood pressure in adults. It is an eating plan in which simple sugar, sugar-sweetened drinks, and red meat are consumed rarely, whereas whole grains, fruits and vegetables, low-fat dairy products, nuts, fish, and poultry are consumed more often. Although it is rich in protein, fiber, calcium, magnesium, and potassium, the DASH diet is poor in cholesterol, saturated and total fat (2).

Magnesium is an alkaline mineral that is fourth most abundantly found in the human body and second most abundantly found in the intracellular area (3). Magnesium deficiency has been associated with many chronic diseases. The first indicator of magnesium deficiency is believed to be serum magnesium level. Shils et al. (4) demonstrated that the first change in the body in experimentally induced magnesium deficiency was a decrease in the serum level. After this study (4), it was accepted that serum magnesium level was a reliable marker in clinical studies for determining the magnesium status of the body because bone and serum magnesium levels were correlated to each other in hypo/hypermagnesemia (3).

Inconsistent data about the relationship between magnesium and HT are present in the literature. Although some data showed no relationships between HT and serum magnesium level (5), some suggest that low serum magnesium concentration could play an important role in the etiology of HT (6). In contrast, serum magnesium level has been found to be lower in patients with HT (7); in addition, it was associated with an increased risk of atherosclerosis, heart failure, and kidney disease (8, 9). Similarly, low dietary magnesium intake could increase the risk of HT (10) and heart failure (11) and cause decline in glomerular filtration rate (GFR) (12).

The primary role of magnesium in the regulation of blood pressure has been suggested to be the role as a calcium (the main determinant of the vascular contraction) antagonist by inhibiting the calcium transport into the smooth muscle cells. Likewise, it promotes vasodilation and protects the vascular function by exerting antioxidant and anti-inflammatory effects (13).

Submitted
05.10.2020

Accepted
28.01.2021

Available Online
14.07.2021

Correspondence
Buse Bakır,
İzmir Katip Çelebi University
Faculty of Health Sciences,
Department of Nutrition and
Dietetics, İzmir, Turkey
Phone: +90 232 329 35 35/
4715
e-mail:
buse.bkr.92@gmail.com

©Copyright 2021 by Erciyes
University Faculty of Medicine -
Available online at
www.erciyesmedj.com

Considering the data in the literature, the objective of this study is to evaluate the dietary magnesium intake, serum magnesium concentrations of patients with HT, and their relationships with hypertensive complications. This study is the first known research in Turkey on the relationship of serum magnesium and dietary magnesium with hypertensive complications. This study obtained significant results to underline the importance of monitoring serum magnesium levels in patients with HT.

MATERIALS and METHODS

Study Design and Subjects

This case–control study was conducted among patients with HT enrolling cardiology outpatient clinics at the Erciyes University Heart Hospital in Kayseri. Eligibility criteria were: age >40 years, HT diagnosis ≥ 10 years, administration of angiotensin-converting enzyme (ACE) inhibitors, and being able to willfully attend to the study. Exclusion criteria were: other chronic diseases, resistant HT, diabetes mellitus, and administration of diuretics, oral antidiabetic medications, and vitamin–mineral supplementations. To evaluate the correlation among serum magnesium concentration, dietary magnesium intake, and hypertensive complications, patients were divided into two groups. There were 35 patients who agreed to participate in the study for each group, but one patient from both groups left the study by stating that they did not want to fill in the food consumption record during the three days. As the patients applied to the outpatient clinic, they were grouped according to their complication status. The case group ($n=34$) comprised patients with hypertensive complications including atherosclerosis, heart failure, and renal failure. The control group ($n=34$) comprised patients with HT and without complications. The complication status was decided by a physician. To ensure that these complications occurred after the diagnosis of HT, medical records of each patient were reviewed and the patients were not included in the study if they were diagnosed with complication before HT. Cardiac functions measures were evaluated by echocardiography and angiography; the renal function was assessed by eGFR via Cockcroft–Gault estimation.

Ethical Considerations

This study was reviewed and approved by the Erciyes University Ethics Committee of Clinical Research (Decision Number: 2016/521, 07.10.2016). Written informed consent was also obtained from all the patients.

Data Collection

The questionnaire containing individual, sociodemographic, and medical information was filled out in face-to-face interviews with the patients. The latest biochemical findings were recorded from the patients' medical folder; moreover, researchers measured body weight, height, and waist circumference (WC). Body mass index (BMI) was calculated by dividing the measured weight with the squared height. In addition, dietary information was obtained via a three-day food record. These records were evaluated with a computer program (BeBiS, Stuttgart, Germany), and daily average energy and nutrient intake were calculated.

Statistical Analysis

Data were analyzed using SPSS 24.0 (SPSS Inc., Chicago, IL, USA), and a p -value < 0.05 was acknowledged as statistically sig-

Table 1. Characteristics of the patients

	Case group		Control group		p
	n	%	n	%	
Gender					0.625
Males	20	58.8	18	52.9	
Females	14	41.2	16	47.1	
Complications					
Atherosclerosis	23	67.6	–	–	
Heart failure	10	29.4	–	–	
Kidney failure	14	41.2	–	–	
Number of complications					0.001
None	–	–	34	100	
1	21	61.8	–	–	
2	13	38.2	–	–	

Table 2. The mean age, anthropometric measurements, and duration of HT of patients

	Case group (Mean \pm SD)	Control group (Mean \pm SD)	p
Age (years)	70.02 \pm 9.87	70.20 \pm 9.92	0.672
Body weight (kg)	77.79 \pm 15.62	77.73 \pm 14.28	0.667
Height (m)	1.62 \pm 0.10	1.64 \pm 0.08	0.420
BMI (kg/m ²)	29.39 \pm 6.13	28.75 \pm 5.52	0.602
WC (cm)	103.14 \pm 15.88	92.76 \pm 14.32	0.006
Duration of HT (years)	17.11 \pm 5.23	15.05 \pm 4.44	0.102

SD: Standard deviation; BMI: Body mass index; WC: Waist circumference; HT: Hypertension

nificant for this study. The normality of the data was assessed using Shapiro–Wilk test. Independent-samples t -test was used to compare normally distributed data in the case and the control groups, and Pearson chi-square test was performed for the categorical variables. The correlations between variables were analyzed by the Spearman's correlation test. Mean and the standard deviation (SD) values for the numerical variables, and the number (n) and percent (%) values for the categorical variables were presented.

RESULTS

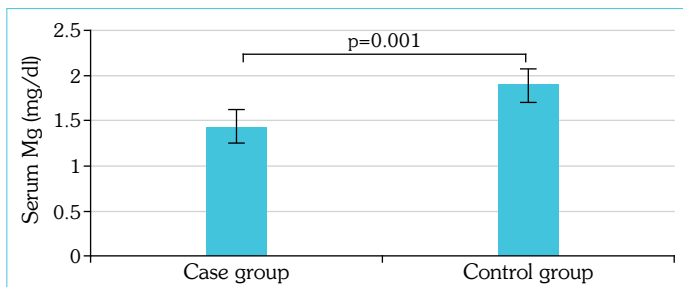
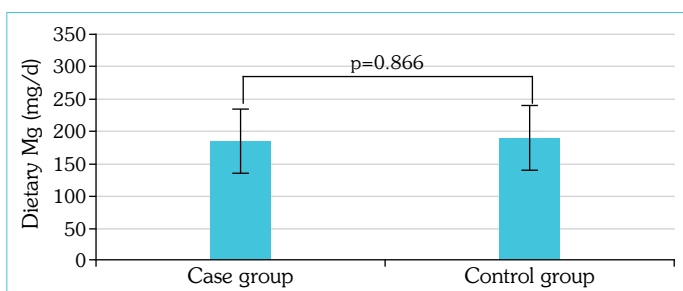
Patient characteristics, anthropometric measurements, biochemical findings, serum magnesium levels, and dietary magnesium intake were compared separately between the case and the control groups (Table 1–3; Fig. 1, 2). The correlations of some selected variables with serum magnesium level and dietary magnesium intake were evaluated in all the patients (Table 4).

Table 1 shows the patient characteristics and complication status. There were no differences between the groups with respect to gender. Atherosclerosis (67.6%) was the highest among the complications in the case group followed by kidney failure (41.2%) and heart failure (29.4%). In addition, 61.8% of the patients with HT

Table 3. The biochemical findings and the blood pressure level of the patients

	Case group (Mean±SD)	Control group (Mean±SD)	p
Fasting blood glucose (FBG) (mg/dl)	132.35±48.31	104.01±15.79	0.006
Total cholesterol (mg/dl)	149.79±45.64	118.4±34.97	0.005
HDL (mg/dl)	42.91±13.54	48.06±10.33	0.082
LDL (mg/dl)	90.99±28.93	106.69±9.94	0.004
Triglycerides (mg/dl)	131.46±51.48	109.97±36.25	0.121
BUN (mg/dl)	34.29±16.47	18.41±7.49	0.001
Creatinine (mg/dl)	2.13±2.16	0.75±0.23	0.001
SBP (mmHg)	126.15±16.71	134.71±16.37	0.019
DBP (mmHg)	73.59±12.42	85.29±9.61	0.001
eGFR (ml/min)	53.57±36.58	96.01±20.32	0.006

SD: Standard deviation; HDL: High-density cholesterol; LDL: Low-density cholesterol; BUN: Blood urea nitrogen; SBP: Systolic blood pressure; DBP: Diastolic blood pressure; GFR: Glomerular filtration rate

**Figure 1.** Serum Mg levels in the case and control groups. The normal range is 1.6–2.4 mg/dl**Figure 2.** Dietary Mg intake levels in the case and the control groups. The recommended level is 300–350 mg/d

in the case group had only one complication and 38.2% had two complications. In the analysis performed according to the complications and serum magnesium status, those having low serum magnesium levels were nine times more likely to have the HT-related complications (OR: 9.02, 95%CI: 3.00–27.10).

According to the results shown in Table 2, there were no significant differences in age, body weight, height, BMI levels, and duration of HT between the groups; only WC was significantly higher in the case group ($p < 0.05$).

Table 4. The correlations between some selected variables and serum Mg, dietary Mg levels

Variables	Serum Mg		Dietary Mg	
	r	p	r	p
Duration of HT	-0.242	0.047	0.127	0.303
Body weight	-0.157	0.201	0.040	0.749
WC	-0.299	0.013	-0.207	0.090
BMI	-0.120	0.331	0.010	0.934
SBP	0.126	0.304	0.053	0.670
DBP	0.223	0.067	0.202	0.098
FBG	-0.267	0.028	-0.229	0.060
Total cholesterol	-0.166	0.175	-0.359	0.003
HDL	0.243	0.046	0.156	0.205
LDL	0.381	0.001	-0.118	0.338
Triglycerides	-0.036	0.770	-0.005	0.970
eGFR	0.581	0.000	0.018	0.881
Creatinine	-0.574	0.000	-0.104	0.400
BUN	-0.412	0.000	-0.155	0.208

HT: Hypertension; WC: Weight circumference; BMI: Body mass index; SBP: Systolic blood pressure; DBP: Diastolic blood pressure; FBG: Fasting blood glucose; HDL: High-density cholesterol; LDL: Low-density cholesterol; GFR: Glomerular filtration rate; BUN: Blood urea nitrogen

Table 3 presents the biochemical findings and the mean blood pressure levels. The results demonstrate significantly higher levels of fasting blood glucose (FBG), total cholesterol, blood urea nitrogen (BUN), and creatinine while significantly lower levels of LDL cholesterol, systolic blood pressure (SBP), diastolic blood pressure (DBP), and eGFR in the case group ($p < 0.05$). Besides, as compared to the control group, serum magnesium level was significantly lower in the case group (1.89 ± 0.18 mg/dl and 1.43 ± 0.19 mg/dl, respectively) (Fig. 1). In contrast, dietary magnesium intake was similar between the groups (case group: 186.28 ± 63.38 mg/d, control group: 188.69 ± 53.39 mg/d; $p > 0.05$), but it was below the recommended level in both groups (Fig. 2).

Table 4 shows the correlations of serum magnesium concentration and dietary magnesium intake with some variables such as biochemical findings and anthropometric measurements. Serum magnesium concentration was found to be significantly and negatively correlated with the duration of HT, WC, FBG, creatinine, and BUN, and positively correlated with HDL, LDL, and eGFR levels ($p < 0.05$). Dietary magnesium intake was negatively correlated with only total cholesterol levels among these variables ($p < 0.05$).

DISCUSSION

This case-control study including 68 patients was conducted for evaluating serum magnesium concentration and dietary magnesium intake in patients with HT and with/without complications. To our knowledge, this is the first report searching the correlations of serum magnesium concentration and dietary magnesium intake with hypertensive complications in Turkey.

Because magnesium exerts vasodilator, antioxidant, anti-inflammatory, and vascular resistant reducer effects, insufficient magnesium levels in the body have been related to the risk of cardiovascular diseases, including HT (13). Yasmin et al. (7) found serum magnesium level to be lower in patients with HT as compared to the individuals with normal blood pressure. We did not include a healthy group to our study, but the mean serum magnesium concentration of all patients was 1.66 ± 0.29 mg/dl (data not included in the tables) and patients with complications had significantly lower serum magnesium levels. The risk analysis in this study revealed that low serum magnesium level may be a risk factor for the HT-related complications.

Because WC is one of the components of metabolic syndrome, it maintains its importance as the major risk factor for cardiovascular diseases (14). In addition to HT (15), WC has been shown to be associated with HT-related complications such as atherosclerosis (16), heart failure (17), and renal failure (18). These findings are consistent with this study where higher WC measurements were found in patients with hypertensive complications. Similar to the study conducted by Shamnani et al. (19), serum magnesium concentration was weakly correlated with WC.

Because HT is a chronic disease, it may affect the clinical findings and quality of life of patients. It will also facilitate the emergence of complications causing organ damage. Yang et al. (20) reported that the duration of HT is correlated with atherosclerosis. In our study, HT year was similar between the groups and it was inversely correlated with serum magnesium level in all the patients.

Cardiovascular diseases are complex disorders coexisting with high blood pressure, lipid and glucose abnormalities, and obesity (21). Atherogenic lipid aggregation has great importance as it has been emphasized that HT can independently trigger atherogenesis (22). In this study, the mean blood lipid levels of the patients were in the normal range. Serum magnesium was found to be positively correlated with HDL (23) and LDL (5). We also found positive correlations of serum magnesium with HDL and LDL cholesterol levels. In contrast, magnesium deficiency can be considered as a risk factor for cardiovascular diseases because of increased endothelial dysfunction, platelet aggregation, oxidative stress, and proinflammatory response (6). Bain et al. (10) reported that an increased dietary magnesium intake may have a favorable effect on total cholesterol levels. This finding is supported by the results of correlation analysis in which we found an inverse correlation between dietary magnesium intake and total cholesterol.

Renal damage caused by HT has been known as the second most important risk factor for end-stage renal failure following diabetic nephropathy (24). Small vessel damages caused by the thinning of the media layer due to high blood pressure affects glomeruli and results in renal damage by causing podocyte loss. Elevated BUN and creatinine levels along with decreased GFR are the most prominent symptoms of kidney injury (25). In contrast, the kidneys play an essential role in the transport and balance of electrolytes in the body, including magnesium. Magnesium deficiency can also promote the progression to kidney injury and subsequent renal failure due to the detrimental effects on tubular epithelium (26). As expected, BUN, creatinine levels were found to be higher and eGFR was lower in the case group involving patients with HT-related kidney injury.

Additionally, serum magnesium concentration was negatively correlated with BUN and creatinine levels. Nevertheless, in contrast to the study conducted by Kunutsor et al. (27), we found a positive correlation between serum magnesium level and eGFR.

Limitations of the Study

There are some limitations that need to be mentioned in this study. First, we were able to evaluate the magnesium status of the patients only by serum magnesium concentration. It has been known that magnesium level in blood represents less than 1% of the total magnesium in the body and magnesium measurement in 24-h urine is more reliable (28). Shils et al. (4) showed that decreased serum magnesium is the first change in the case of magnesium deficiency. Besides, serum magnesium is sufficient to confirm the diagnosis in individuals with suspected magnesium deficiency (29). Considering all these conditions and the old age group in the study, we only evaluated the serum magnesium, as 24-h urine collection could cause patients to refuse to participate in the study. Additionally, dietary magnesium intake was calculated via three-day food records, but meals were recorded with the statements of the patients. For this reason, it is believed that there may be over/under-reporters among the participants. Second, this study was conducted in a single center; therefore, the number of patients may not be enough to make a sweeping statement. In addition, left ventricular hypertrophy and left ventricular mass index increase evaluation and 24-h ambulatory blood pressure monitoring of the participants could not be performed. Finally, there were no patients who have all three complications determined in the method. Atherosclerosis was the most prevalent among complications and complications were not evenly distributed among patients.

CONCLUSION

As a result of this research, the dietary magnesium intake of hypertensive patients was found to be significantly inadequate. Serum magnesium concentration was lower in patients with HT-related complications, and it was inversely associated with the duration of HT, WC, and eGFR in all the patients. Considering these results, it should be considered that magnesium level may decrease in parallel with the duration of HT and patients should be monitored closely with regard to the presence of complications. To prevent or delay the development of hypertensive complications, patients should be encouraged to see the dietitian frequently and must be encouraged to comply with the DASH diet which is rich in magnesium-rich foods such as whole grains, legumes, nuts, and green leafy vegetables. The relationship between hypertensive complications and nutritional status should be evaluated with a larger sample size and long-term follow-up studies.

Acknowledgements: We would like to thank Assistant Professor İffet İpek Boşgelmez from Erciyes University Faculty of Pharmacy, for her valuable support.

Ethics Committee Approval: The Erciyes University Clinical Research Ethics Committee granted approval for this study (date: 07.10.2016, number: 2016/521).

Informed Consent: Written informed consent was obtained from patients who participated in this study.

Peer-review: Externally peer-reviewed.

Author Contributions: Concept – BB, HŞ, AD; Design – BB, HŞ, AD; Supervision – BB, HŞ, AD; Materials – AD; Data Collection and/or Processing – BB; Analysis and/or Interpretation – BB, HŞ, AD; Literature Search – BB; Writing – BB; Critical Reviews – HŞ, AD.

Conflict of Interest: The authors have no conflict of interest to declare.

Financial Disclosure: The authors declared that this study has received no financial support.

REFERENCES

- Mills KT, Stefanescu A, He J. The global epidemiology of hypertension. *Nat Rev Nephrol* 2020; 16(4): 223–37. [\[CrossRef\]](#)
- Challa HJ, Ameer MA, Uppaluri KR. DASH diet to stop hypertension. In: *StatPearls*. Treasure Island (FL): StatPearls Publishing; May 19, 2021.
- Chaudhary DP, Sharma R, Bansal DD. Implications of magnesium deficiency in type 2 diabetes: a review. *Biol Trace Elem Res* 2010; 134(2): 119–29. [\[CrossRef\]](#)
- Shils ME. Experimental human magnesium depletion. *Medicine (Baltimore)* 1969; 48(1): 61–85. [\[CrossRef\]](#)
- Barragán R, Llopis J, Portolés O, Sorlí JV, Coltell O, Rivas-García L, et al. Influence of demographic and lifestyle variables on plasma magnesium concentrations and their associations with cardiovascular risk factors in a mediterranean population. *Nutrients* 2020; 12(4): 1018.
- Kostov K, Halacheva L. Role of magnesium deficiency in promoting atherosclerosis, endothelial dysfunction, and arterial stiffening as risk factors for hypertension. *Int J Mol Sci* 2018; 19(6): 1724. [\[CrossRef\]](#)
- Yasmin F, Samad N. REPORT- Association between serum electrolytes and erythrocytes Na⁺, K⁺ in hypertensive and normotensive male compared to female. *Pak J Pharm Sci* 2020; 33(1): 207–14.
- Tangvoraphonkchai K, Davenport A. Magnesium and cardiovascular disease. *Adv Chronic Kidney Dis* 2018; 25(3): 251–60. [\[CrossRef\]](#)
- Pendón-Ruiz de Mier MV, Rodelo-Haad C, Diaz-Tocados JM, Muñoz-Castañeda JR, Rodríguez M. Magnesium: An old player revisited in the context of CKD-MBD. *Clin Chim Acta* 2020; 501: 53–9.
- Bain LK, Myint PK, Jennings A, Lentjes MA, Luben RN, Khaw KT, et al. The relationship between dietary magnesium intake, stroke and its major risk factors, blood pressure and cholesterol, in the EPIC-Norfolk cohort. *Int J Cardiol* 2015; 196: 108–14. [\[CrossRef\]](#)
- Zhang W, Iso H, Ohira T, Date C, Tamakoshi A; JACC Study Group. Associations of dietary magnesium intake with mortality from cardiovascular disease: the JACC study. *Atherosclerosis* 2012; 221(2): 587–95. [\[CrossRef\]](#)
- Rebholz CM, Tin A, Liu Y, Kuczmarski MF, Evans MK, Zonderman AB, et al. Dietary magnesium and kidney function decline: The healthy aging in neighborhoods of diversity across the life span study. *Am J Nephrol* 2016; 44(5): 381–7. [\[CrossRef\]](#)
- Schutten JC, Joosten MM, de Borst MH, Bakker SJL. Magnesium and blood pressure: A physiology-based approach. *Adv Chronic Kidney Dis* 2018; 25(3): 244–50. [\[CrossRef\]](#)
- Chang KT, Chen CH, Chuang HH, Tsao YC, Lin YA, Lin P, et al. Which obesity index is the best predictor for high cardiovascular disease risk in middle-aged and elderly population? *Arch Gerontol Geriatr* 2018; 78: 165–70. [\[CrossRef\]](#)
- Haberka M, Stolarz-Skrzypek K, Biedroń M, Szóstak-Janiak K, Partyka M, Olszanecka-Glinianowicz M, et al. Obesity, visceral fat, and hypertension-related complications. *Metab Syndr Relat Disord* 2018; 16(10): 521–9. [\[CrossRef\]](#)
- Al-Domi H, Al-Shorman A. Increased waist circumference is associated with subclinical atherosclerosis in schoolchildren. *Diabetes Metab Syndr* 2019; 13(1): 264–9. [\[CrossRef\]](#)
- Fernandes-Silva MM, Shah AM, Claggett B, Cheng S, Tanaka H, Silvestre OM, et al. Adiposity, body composition and ventricular-arterial stiffness in the elderly: The atherosclerosis risk in communities study. *Eur J Heart Fail* 2018; 20(8): 1191–201. [\[CrossRef\]](#)
- Dai H, Lu S, Tang X, Lu M, Chen R, Chen Z, et al. Combined association of serum uric acid and metabolic syndrome with chronic kidney disease in hypertensive patients. *Kidney Blood Press Res* 2016; 41(4): 413–23. [\[CrossRef\]](#)
- Shammani G, Rukadikar CA, Gupta V, Singh S, Tiwari S, Bharti SS, et al. Serum magnesium in relation with obesity. *Natl J Physiol Pharm Pharmacol* 2018; 8(7): 1074–7. [\[CrossRef\]](#)
- Yang Y, Wu QH, Li Y, Gao PJ. Association of SLRPs with carotid artery atherosclerosis in essential hypertensive patients. *J Hum Hypertens* 2018; 32(8-9): 564–71. [\[CrossRef\]](#)
- Catapano AL, Graham I, De Backer G, Wiklund O, Chapman MJ, Drexel H, et al; ESC Scientific Document Group. 2016 ESC/EAS Guidelines for the management of dyslipidaemias. *Eur Heart J* 2016; 37(39): 2999–3058. [\[CrossRef\]](#)
- Hurtubise J, McLellan K, Durr K, Onasanya O, Nwabuko D, Ndisang JF. The different facets of dyslipidemia and hypertension in atherosclerosis. *Curr Atheroscler Rep* 2016; 18(12): 82. [\[CrossRef\]](#)
- Ansari MR, Maheshwari N, Shaikh MA, Laghari MS, Darshana, Lal K, et al. Correlation of serum magnesium with dyslipidemia in patients on maintenance hemodialysis. *Saudi J Kidney Dis Transpl* 2012; 23(1): 21–5.
- Levey AS, Coresh J. Chronic kidney disease. *Lancet* 2012; 379(9811): 165–80. [\[CrossRef\]](#)
- Seccia TM, Caroccia B, Calò LA. Hypertensive nephropathy. Moving from classic to emerging pathogenetic mechanisms. *J Hypertens* 2017; 35(2): 205–12. [\[CrossRef\]](#)
- DiNicolantonio JJ, O'Keefe JH, Wilson W. Subclinical magnesium deficiency: a principal driver of cardiovascular disease and a public health crisis. *Open Heart* 2018; 5(1): e000668. [\[CrossRef\]](#)
- Kunutsor SK, Khan H, Laukkanen JA. Serum magnesium and risk of new onset heart failure in men: the Kuopio Ischemic Heart Disease Study. *Eur J Epidemiol* 2016; 31(10): 1035–43. [\[CrossRef\]](#)
- Elin RJ. Assessment of magnesium status for diagnosis and therapy. *Magn Res* 2010; 23(4): S194–8.
- Pham PC, Pham PM, Pham SV, Miller JM, Pham PT. Hypomagnesemia in patients with type 2 diabetes. *Clin J Am Soc Nephrol* 2007; 2(2): 366–73. [\[CrossRef\]](#)