

# Why magnesium level check should be part of standard diabetes care?

Mehmet Uzunlulu<sup>1</sup>, Elif Pala<sup>1</sup>, Aysu Tanrıvermiş<sup>1</sup>, Muhammet Mikdat Akbas<sup>1</sup>, Ender İğneci<sup>2</sup>, Mirac Vural Keskinler<sup>1</sup>

<sup>1</sup>Department of Internal Medicine, Istanbul Medeniyet University, Faculty of Medicine, Istanbul, Turkey

<sup>2</sup>Department of Internal Medicine, Kars Harakani City Hospital, Kars, Turkey

## ABSTRACT

**Objectives:** The aim of this study was to investigate the effectiveness of routine magnesium monitoring in patients with diabetes during follow-up.

**Methods:** A retrospective observational clinical study was conducted, encompassing 387 participants aged 18 years and older, with and without diabetes. The control group comprised patients without diabetes. The group with diabetes consisted of 237 patients (134 women, 103 men), while the control group consisted of 150 patients (85 women, 65 men). Hypomagnesemia was diagnosed at <1.6 mg/dL. The study compared the groups based on the frequency of hypomagnesemia and clinical characteristics.

**Results:** The prevalence of hypomagnesemia was 8.8% (13.8% in patients with diabetes, 1.3% in the control group;  $p=0.001$ ), with a magnesium level of  $1.93\pm 0.24$  mg/dL ( $1.85\pm 0.25$  mg/dL in patients with diabetes,  $2.06\pm 0.16$  mg/dL in the control group;  $p=0.001$ ).

The study found that the prevalence of hypomagnesemia was significantly higher in patients with diabetes, particularly those with advanced age, longer duration of diabetes, impaired glycemic control, and previous hypomagnesemia diagnosis. Moreover, the use of proton pump inhibitors (PPIs) and diuretics were more common in patients with diabetes with hypomagnesemia. Conversely, the frequency of SGLT-2 inhibitor use was lower in patients with hypomagnesemia.

**Discussion:** This study suggests that routine magnesium measurement should be considered as a part of standard evaluation, especially for patients with diabetes exhibiting the aforementioned risk factors, and emphasizes the significance of acknowledging PPI and diuretic usage in such cases.

**Keywords:** Magnesium, diabetes mellitus, risk factors

Magnesium is an important ion involved in almost every mechanism in the cell, including energy homeostasis, protein synthesis, and DNA stability, and hypomagnesemia is defined as a magnesium concentration of 1.6 mg/dL or <2 standard deviations from the general population

mean.<sup>1</sup> Hypomagnesemia may develop due to chronic disease, alcoholism, drugs, gastrointestinal or renal loss and many other reasons. Signs and symptoms can range from mild tremor and malaise to cardiac ischemia and death. It is an important electrolyte disorder that can be observed in 2% of the general

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**Address for correspondence:** Mirac Vural Keskinler; MD, PhD, Assoc Prof., Istanbul Medeniyet University, Faculty of Medicine, Department of Internal Medicine, 34722, Kadıköy, Istanbul, Turkey. E-mail: miracvural@hotmail.com

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population, 12% of hospitalized patients, 60-65% of patients hospitalized in intensive care units, and 14-48% of those with type 2 diabetes.<sup>2-4</sup> It has been reported that magnesium levels are lower in patients with diabetes than in those without diabetes, and that mutations in magnesotropic genes, low dietary Mg intake, autonomic neuropathy or impaired intestinal Mg absorption due to diarrhea triggered by metformin use, drugs such as diuretics, immunosuppressives and proton pump inhibitors, metabolic acidosis and insulin resistance may be related to this.<sup>5</sup> On the other hand, it has been reported that hypomagnesemia is associated with poor glycemic control and micro/macro vascular complications and development of foot ulcers in patients with diabetes.<sup>6</sup> Magnesium is not a routine monitoring parameter in patients with diabetes in daily practice. However, in parallel with the increase in laboratory investigations, asymptomatic or incidental hypomagnesemia cases are frequently encountered. The aim of this study was to determine the frequency of hypomagnesemia and clinical features associated with hypomagnesemia in patients with type 2 diabetes.

## METHODS

Two hundred and thirtyseven patients with a confirmed diagnosis of type 2 diabetes mellitus and 150 controls aged  $\geq 18$  were included in this retrospective, observational, clinical study among consecutive patients attending the outpatient clinics of an university hospital department of Internal Medicine. Patients with advanced kidney and heart failure, chronic liver disease, cancer or malabsorption disorder were excluded from the study. Study protocol was approved by local ethics committee (approval date and number:0581/2022). The study was conducted in accordance with Declaration of Helsinki.

### Study design

Demographic characteristics, comorbid conditions, duration of diabetes, type of diabetes, treatment features, presence of hypomagnesemia detected in previous admissions, use of drugs known to have an effect on magnesium levels (such as diuretic, antibiotic, proton pump inhibitor, immunosuppressive, digitalis) of patients who met the inclusion criteria.

Previous and current laboratory data (glucose, HbA1c, creatinine, alanine aminotransferase, total cholesterol, LDL cholesterol, triglyceride, magnesium) were obtained from medical records. The

diagnosis of hypomagnesemia was accepted as the hospital laboratory lower limit reference value of  $< 1.6$  mg/dL.

Diabetes and control groups were compared according to the frequency and clinical features of hypomagnesemia. The clinical features of patients with hypomagnesemia and normal magnesium who have diabetes were also analyzed. Multivariate logistic regression analysis was performed to identify clinical features associated with hypomagnesemia.

### Diabetes mellitus diagnosis

Patients were diagnosed with diabetes mellitus if they had one of the following: Fasting plasma glucose  $\geq 126$  mg/dL (7.0 mmol/L) or 2 h glucose  $\geq 200$  mg/dL (11.1 mmol/L) during oral glucose tolerance test, or HbA1c  $\geq 6.5\%$  (48 mmol/mol) or a random plasma glucose  $\geq 200$  mg/dL (11.1 mmol/L) in a patient with symptoms of hyperglycemia.<sup>7</sup>

### Laboratory analysis

Fasting glucose concentrations were determined using the hexokinase method. Serum creatinine was assayed using the kinetic Jaffe method. For alanine transaminase concentrations, an enzymatic (without P-50-P, NADH) method was used. Fasting plasma total cholesterol, HDL and LDL cholesterol, and triglyceride concentrations were determined using enzymatic methods (Abbott Architect c16000 and c8000, Abbott). A Tosoh HLC-723 G8 (Tosoh G8) (Tosoh, Japan) (variant-mode) ion exchange high-performance liquid chromatography system was used for HbA1c measurements. Magnesium was studied on Roche/Hitachi cobas 8000 c, Mannheim, Germany.

### Statistical Analyses

NCSS (Number Cruncher Statistical System) 2020 Statistical Software (NCSS LLC, Kaysville, Utah, USA) program was used. Shapiro Wilks test and Box Plot graphs were used to evaluate the conformity of the data to normal distribution. Student t-test was used for the evaluation of two quantitative groups with normal distribution and Mann Whitney U Test was used for the evaluation of two quantitative groups without normal distribution. Pearson Chi-Square Test, Fisher's Exact Test and Fisher Freeman Halton test were used to compare qualitative data. Logistic Regression Analysis was used to determine the risk factors affecting low magnesium. The results were evaluated at 95% confidence interval and significance was evaluated at  $p < 0.05$  level.

## RESULTS

A total of 387 patients (219 females, 168 males) were included in the study. The group with diabetes consisted of 237 individuals (134 female, 103 male, mean age: 60.87±10.86 years), while the control group comprised 150 cases (85 female, 65 male, mean age: 59.56±11.46 years).

The clinical characteristics of the groups are detailed in Table 1. Age and gender distributions were comparable between the groups. Hypomagnesemia was observed in 8.8% of all cases, with a mean magnesium level of 1.93±0.24. The frequency of patients with hypomagnesemia in their previous admissions was 14.2%, and no patients exhibited hypermagnesemia.

The group with diabetes showed a higher prevalence of hypomagnesemia compared to the control group (13.5% vs. 1.3%, p=0.001), with lower mean magnesium levels (1.85±0.25 mg/dL versus

2.06±0.16 mg/dL, p=0.001), and a higher reported incidence of hypomagnesemia in previous records (20.3% vs. 4.7%, p=0.001). Fasting glucose, HbA1c, and triglyceride levels were higher, whereas total cholesterol, LDL cholesterol, and HDL cholesterol levels were lower in the diabetic group (p<0.01 for all). The frequencies of patients using antihypertensive drugs, including diuretics and proton pump inhibitors, were higher in the group with diabetes than in the group without diabetes (p<0.01 for all).

The comparison of clinical and laboratory features of patients with diabetes experiencing hypomagnesemia and those with normal magnesium are given in Tables 2 and 3.

In the diabetic group with hypomagnesemia, mean age (p=0.001), mean duration of diabetes (p= 0.023), frequency of patients with hypomagnesemia at previous admissions (p=0.001), frequency of patients using proton pump inhibitors (p=0.011), patients using mixed insulin frequency (p=0.018) and fasting glucose

**Table 1. Clinical characteristics of the groups**

	Total (n=387)	DM- (n=150)	DM+ (n=237)	p
Age (sd)	60.36±11.1	59.56±11.46	60.87±10.86	0.137
Female (n,%)	219 (56.6)	85 (56.7)	134 (56.5)	
Male (n,%)	168 (43.4)	65 (43.3)	103 (43.5)	0.980
Frequency of previous hypomagnesemia (n,%)	55 (14,2)	7 (4,7)	48 (20,3)	<b>0.001</b>
<b>Laboratory</b>				
Glucose (mg/dL)	128,04±58,48	91,89±12,80	150,92±64,30	<b>0,001</b>
HbA1C (%)	6,86±1,80	5,59±0,36	7,66±1,89	<b>0,001</b>
Creatinine (mg/dL)	0,82±0,22	0,82±0,17	0,83±0,25	0,663
Glomerular filtration rate (mL/dk/1.73 m <sup>2</sup> )	88,5±18,35	89,49±15,72	87,88±19,84	0,779
Alanine aminotransferase	19,8±12,67	20,64±15,27	19,27±10,70	0,505
Total cholesterol (mg/dL)	192,35±46,44	203,64±40,73	185,2±48,45	<b>0,001</b>
LDL cholesterol (mg/dL)	109,56±38,46	120,33±35,07	102,75±39,02	<b>0,001</b>
HDL cholesterol (mg/dL)	51,34±13,71	54,57±13,71	49,29±13,35	<b>0,001</b>
Triglyceride (mg/dL)	159,03±90,9	145,6±86,11	167,52±92,98	<b>0,007</b>
Magnesium (mg/dL)	1,93±0,24	2,06±0,16	1,85±0,25	<b>0,001</b>
Prevalence of hypomagnesemia (n,%)	34 (8,8)	2 (1,3)	32 (13,5)	<b>0,001</b>
<b>Use of hypomagnesemia-related medication</b>				
Diuretics (n,%)	82 (21,2)	18 (12,0)	64 (27,0)	<b>0,001</b>
Proton pump inhibitor (n,%)	81 (20,9)	8 (5,3)	73 (30,8)	<b>0,001</b>
Antibiotics (n,%)	2 (0,5)	2 (1,3)	0 (0,0)	0,150
Immunosuppressive (n,%)	0 (0,0)	0 (0,0)	0 (0,0)	-
Digital (n,%)	2 (0,5)	0 (0,0)	2 (1,8)	0,524
<b>Comorbidities</b>				
Hypertension (n,%)	191 (49,4)	43 (28,7)	148 (62,4)	<b>0,001</b>
Coronary artery disease (n,%)	60 (15,5)	12 (8,0)	48 (20,3)	<b>0,001</b>
Chronic kidney disease (n,%)	16 (4,1)	3 (2,0)	13 (5,5)	0,118
Hypothyroidism (n,%)	58 (15,0)	23 (15,3)	35 (14,8)	0,879

**Table 2. Comparison of clinical characteristics of diabetic patients with and without hypomagnesemia**

	Hypomagnesemia (+) (n=32)	Hypomagnesemia (-) (n=205)	<i>p</i>
Age (mean±SD)	66.56±9.39	59.98±10.82	<b>0.001</b>
Duration of diabetes (years) (mean±SD)	15,19±9,38	11,33±7,73	<b>0.023</b>
Frequency of previous hypomagnesemia (n,%)	14 (43.8)	34 (16,6)	<b>0.001</b>
<b>Diabetes type (n,%)</b>			
Type I	1 (3,1)	6 (2,9)	1.000
Type II	31 (96,9)	199 (97,1)	
<b>Gender (n,%)</b>			
Female	21 (65,6)	113 (55,1)	0.263
Male	11 (34,4)	92 (44,9)	
<b>Duration of diabetes (n, %)</b>			
<10 years	12 (37,5)	113 (55,1)	0.063
≥10 years	20 (62,5)	92 (44,9)	
<b>Diabetes treatment (n, %)</b>			
Insulin	0 (0,0)	13 (6,3)	
Oral antidiabetic	24 (75,0)	134 (65,4)	0,503
Insulin+ oral antidiabetic	8 (25,0)	56 (27,3)	
Lifestyle treatment	0 (0,0)	2 (1,0)	
<b>Comorbidities (n,%)</b>			
Hypertension	24 (75)	124 (60.5)	0,115
Coronary artery disease	5 (15,6)	43 (21)	0,484
Chronic kidney disease	1 (3.1)	12 (5.9)	1.000
Hypothyroidism	3 (9.4)	32 (15,6)	0.434
Diabetic neuropathy	8 (25)	54 (26.3)	0.872
Gastroparesis	0 (0)	1 (0,5)	1.000
Retinopathy	2 (6.3)	8 (3.9)	0.629
<b>Hypomagnesemia-related drug use (n,%)</b>			
Diuretics	9 (28.1)	55 (26.8)	0.878
Proton pump inhibitor	16 (50)	57 (27.8)	<b>0,011</b>
Antibiotics	0 (0)	0 (0)	-
Immunosuppressive	0 (0)	0 (0)	-
Digital	0 (0)	2 (1.0)	1.000

levels ( $p=0.032$ ) were higher than the diabetic group with normal magnesium levels, and the frequency of patients using SGLT-2 inhibitors ( $p=0.001$ ) was lower. In the multivariate logistic regression analysis, age (ODDS: 1.064, 95% CI: 1.020-1.110,  $p=0.004$ ) and fasting glucose levels (ODDS: 1.006, 95% CI: 1.001-1.011,  $p=0.027$ ) were independent risk factors for hypomagnesemia.

## DISCUSSION

The results of this study revealed that the frequency of hypomagnesemia is high in patients with diabetes and that age and fasting glucose levels are the major determinants of hypomagnesemia.

Increasing data reveal that the prevalence of hypomagnesemia is high in patients with diabetes,

although it varies according to study design and population characteristics, and hypomagnesemia is mostly associated with advanced age, diabetes duration, diabetes severity, treatment characteristics, and diabetic micro- and macrovascular complications.<sup>8-10</sup>

For example, in an observational cohort study including 929 patients with type 2 diabetes treated in primary care, the frequency of hypomagnesemia was found to be 9.6%, and it was shown that age, duration of diabetes, body mass index, HbA1c, metformin, sulfonylurea, and DPP-4 inhibitor use were negatively associated with magnesium concentration.<sup>4</sup>

Similar to our study, in an observational study conducted on outpatients with type 2 diabetes, the frequency of hypomagnesemia was found to be 12.9%, and it was stated that hypomagnesemia was associated with poor glycemic control and contributed to the development and progression of micro- and

**Table 3 Comparison of treatment and laboratory characteristics in diabetics with and without hypomagnesemia**

	Hypomagnesemia (+) (n=32)	Hypomagnesemia (-) (n=205)	<i>p</i>
<b>OAD usage (n,%)</b>			
Metformin	28 (87,5)	164 (80,0)	0.314
Sulfonylurea	4 (12,5)	13 (6,3)	0.259
DPP4-i	19 (59,4)	95 (46,3)	0.170
GLP-1 agonist	0 (0,0)	6 (2,9)	1.000
Glitazone	4 (12,5)	25 (12,2)	1.000
SGLT2-i	2 (6,3)	81 (39,5)	<b>0.001</b>
Acarbose	1 (3,1)	6 (2,9)	1.000
No OAD use	0 (0,0)	17 (8,3)	0.139
<b>Insulin usage (n,%)</b>			
Basal insülin	4 (50,0)	57 (82,6)	0.053
Bolus insülin	3 (37,5)	24 (34,8)	1.000
Basal plus	0 (0,0)	4 (5,8)	1.000
Mix insülin	4 (50,0)	8 (11,6)	<b>0.018</b>
<b>Laboratory (mean±SD)</b>			
Fasting glucose (mg/dL)	174,13±74,66	147,30±61,95	<b>0,032</b>
HbA1c (%)	7,82±1,82	7,63±1,91	0.463
Creatinine (mg/dL)	0,81±0,19	0,83±0,25	0.751
GFR	84,11±16,28	88,46±20,32	0.074
Total cholesterol (mg/dL)	183,78±37,03	185,42±50,07	0.859
LDL cholesterol (mg/dL)	96,31±31,55	103,76±40,03	0.317
HDL cholesterol (mg/dL)	51,06±12,18	49,01±13,52	0.321
Triglycerides (mg/dL)	172,53±83,58	166,74±94,53	0.450
Spot urine protein/creatinine ratio	0,42±0,94	0,27±0,69	0.292
<b>Glycemic control (n,%)</b>			
<%7	14 (43,8)	92 (44,9)	0.905
≥%7	18 (56,3)	113 (55,1)	

macrovascular complications of diabetes.<sup>11</sup>

Second; The reason is that the cases using proton pump inhibitors were found to be significantly higher both in the group with diabetes than in the non-diabetic group and in individuals with diabetes and hypomagnesemia compared to those with normal magnesium.

In fact, it is a well-known phenomenon that proton pump inhibitors are associated with the development of hypomagnesemia and is thought to be due to decreased intestinal magnesium absorption, mainly through the transient receptor potential melastatin-6 and -7 (TRPM6/7).<sup>12</sup>

In this respect, it may not be a surprising finding that PPI use is high in individuals with diabetes with dyspeptic complaints such as diabetic gastroparesis. However, the fact that half of the individuals with diabetes and hypomagnesemia were using PPI reveals the necessity of questioning the use of PPI in individuals with diabetes and hypomagnesemia.

Third; The prevalence of SGLT-2 inhibitor users in individuals with diabetes with normal magnesium levels was found to be significantly higher than in individuals with diabetes and hypomagnesemia. This finding raises questions as to whether this is coincidental or whether the use of SGLT-2 inhibitors has a protective effect against hypomagnesemia. In a meta-analysis of randomized controlled trials, it was observed that SGLT-2 inhibitors moderately but significantly increase magnesium levels as a class effect, suggesting that this may be associated with increased magnesium absorption in the renal proximal tubule.<sup>13</sup> Case reports have shown that SGLT-2 inhibitors improve magnesium levels in individuals with diabetes with refractory hypomagnesemia.<sup>14,15</sup>

In this respect, although the cause-effect relationship could not be fully established in our study, considering the literature, this finding supports the idea that the use of SGLT-2 inhibitors may be a therapeutic option, especially in individuals with diabetes with refractory

hypomagnesemia.

Although the use of mixed insulin was found to be higher in the group with diabetes with hypomagnesemia compared to the group with diabetes with normal magnesium levels, although it was found to be statistically significant, it was considered as a coincidental finding due to the low number of cases.

## CONCLUSION

This study suggested that the frequency of hypomagnesemia is high in individuals with diabetes and that magnesium measurement should be a part of routine screening, especially in individuals with diabetes with advanced age, long diabetes duration, impaired glycemic control, and previously diagnosed hypomagnesemia.

On the other hand, it is one of the important results of this study that the use of proton pump inhibitors should be considered in the approach to the individual with diabetes with hypomagnesemia. More comprehensive studies are needed to evaluate the positive effects of SGLT-2 inhibitors on hypomagnesemia.

## Limitations

The retrospective design, the fact that the cases did not receive magnesium treatment at the time of admission or before, alcohol use known to have an effect on magnesium, anthropometric measurements, micro and macrovascular complications, and conditions leading to renal loss such as 24-hour urine magnesium excretion could not be evaluated clearly and accurately.

## Conflict of Interest

The author(s) declared no potential conflicts of interest with respect to the research, authorship, and/or publication of this article.

## Ethical Approval

The protocol of the study was approved by the Medical Ethics Committee of İstanbul Medeniyet University, İstanbul, Turkey. (Decision number: 2022/0581, date: 05.10.2022).

## Authors' Contribution

Study Conception: MU,MVK; Study Design: MU; Supervision: MU,MVK; Materials: EP,AT,Eİ,MMA; Data Collection and/or Processing: P,AT,Eİ,MMA; Analysis and/or Data Interpretation: MU,MVK; Critical Review: MU,MVK; Manuscript preparing:

MU,MVK.

## REFERENCES

1. Pham PC, Pham PM, Pham SV, Miller JM, Pham PT. Hypomagnesemia in patients with type 2 diabetes. *Clin J Am Soc Nephrol*. 2007 Mar;2(2):366-73. doi: 10.2215/CJN.02960906. Epub 2007 Jan 3. PMID: 17699436. Agus ZS. Hypomagnesemia. *J Am Soc Nephrol* 1999; 10:1616.
2. Agus ZS. Hypomagnesemia. *J Am Soc Nephrol*. 1999 Jul;10(7):1616-22. doi: 10.1681/ASN.V1071616. PMID: 10405219.
3. Ahmed F, Mohammed A. Magnesium: The Forgotten Electrolyte-A Review on Hypomagnesemia. *Med Sci (Basel)*. 2019 Apr 4;7(4):56. doi: 10.3390/medsci7040056. PMID: 30987399; PMCID: PMC6524065.
4. Waanders F, Dullaart RPF, Vos MJ, Hendriks SH, van Goor H, Bilo HJG, van Dijk PR. Hypomagnesaemia and its determinants in a contemporary primary care cohort of persons with type 2 diabetes. *Endocrine*. 2020 Jan;67(1):80-86. doi: 10.1007/s12020-019-02116-3. Epub 2019 Oct 24. PMID: 31650393; PMCID: PMC6968975.
5. Kurstjens S, de Baaij JH, Bouras H, Bindels RJ, Tack CJ, Hoenderop JG. Determinants of hypomagnesemia in patients with type 2 diabetes mellitus. *Eur J Endocrinol*. 2017 Jan;176(1):11-19. doi: 10.1530/EJE-16-0517. Epub 2016 Oct 5. PMID: 27707767.
6. Dasgupta A, Sarma D, Saikia UK. Hypomagnesemia in type 2 diabetes mellitus. *Indian J Endocrinol Metab*. 2012 Nov;16(6):1000-3. doi: 10.4103/2230-8210.103020. PMID: 23226651; PMCID: PMC3510925.
7. American Diabetes Association; 2. Classification and Diagnosis of Diabetes: Standards of Medical Care in Diabetes—2019. *Diabetes Care* 1 January 2019; 42 (Supplement\_1): S13–S28. <https://doi.org/10.2337/dc19-S002>
8. Rabeea IS, Al-Gburi K, Adnan I, Hasan B, Mohammed M, Mohammed M. Pattern and Correlates of Hypomagnesemia Among Subset of Diabetes Mellitus. *Curr Diabetes Rev*. 2020;16(4):364-369. doi: 10.2174/1573399814666181026095236. PMID: 30362420.
9. Paladiya R, Pitliya A, Choudhry AA, Kumar D, Ismail S, Abbas M, Naz S, Kumar B, Jamil A, Fatima A. Association of Low Magnesium Level With Duration and Severity of Type 2 Diabetes.

- Cureus. 2021 May 27;13(5):e15279. doi: 10.7759/cureus.15279. PMID: 34194881; PMCID: PMC8235873.
10. Ramadass S, Basu S, Srinivasan AR. SERUM magnesium levels as an indicator of status of Diabetes Mellitus type 2. *Diabetes Metab Syndr*. 2015 Jan-Mar;9(1):42-5. doi: 10.1016/j.dsx.2014.04.024. Epub 2014 May 24. PMID: 25470649.
  11. Zahra H, Berriche O, Mizouri R, Boukhatatia F, Khiari M, Gamoudi A, Lahmar I, Ben Amor N, Mahjoub F, Zayet S, Jamoussi H. Plasmatic Magnesium Deficiency in 101 Outpatients Living with Type 2 Diabetes Mellitus. *Clin Pract*. 2021 Oct 27;11(4):791-800. doi: 10.3390/clinpract11040095. PMID: 34842632; PMCID: PMC8628662.
  12. William JH, Danziger J. Proton-pump inhibitor-induced hypomagnesemia: Current research and proposed mechanisms. *World J Nephrol*. 2016 Mar 6;5(2):152-7. doi: 10.5527/wjn.v5.i2.152. PMID: 26981439; PMCID: PMC4777786.
  13. Tang H, Zhang X, Zhang J, Li Y, Del Gobbo LC, Zhai S, Song Y. Elevated serum magnesium associated with SGLT2 inhibitor use in type 2 diabetes patients: a meta-analysis of randomised controlled trials. *Diabetologia*. 2016 Dec;59(12):2546-2551. doi: 10.1007/s00125-016-4101-6. Epub 2016 Sep 15. PMID: 27628105.
  14. Shah CV, Robbins TS, Sparks MA. Sodium-Glucose Cotransporter 2 Inhibitors and Management of Refractory Hypomagnesemia Without Overt Urinary Magnesium Wasting: A Report of 2 Cases. *Kidney Med*. 2022 Aug 12;4(10):100533. doi: 10.1016/j.xkme.2022.100533. PMID: 36185705; PMCID: PMC9519375.
  15. Ray EC, Boyd-ShiwarSKI CR, Liu P, Novacic D, Cassiman D. SGLT2 Inhibitors for Treatment of Refractory Hypomagnesemia: A Case Report of 3 Patients. *Kidney Med*. 2020 Apr 18;2(3):359-364. doi: 10.1016/j.xkme.2020.01.010. PMID: 32734255; PMCID: PMC7380441.

