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SGLT2 Inhibitors in the Management of Hypomagnesemia in Patients Without Diabetes: A Narrative Review

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Abstract

Introduction:

Hypomagnesemia remains an underdiagnosed and undertreated condition, despite its profound adverse health effects. Recently, sodium-glucose cotransporter 2 inhibitors have emerged as effective agents in elevating serum magnesium levels.

Aim of the Study:

The aim of the study is a critical appraisal of the literature regarding the efficacy of sodium-glucose cotransporter 2 inhibitors in elevating serum magnesium levels in hypomagnesemic, non-diabetic patients.

Methods and Materials:

A literature search was conducted across major electronic databases for randomized controlled studies, post-hoc analyses of randomized controlled studies, observational studies and case reports evaluating the effects of sodium-glucose cotransporter 2 inhibitors on serum magnesium levels, with particular attention to the non-diabetic patient population. Seven articles met the eligibility criteria and formed the basis of this review.

Results:

Available data suggest that the use of SGLT2 inhibitors in non-diabetic patients with hypomagnesemia was consistently associated with increased serum magnesium levels. Patients with lower baseline magnesium concentrations appeared to experience more

pronounced improvement of serum magnesium levels, which increased predominantly early in treatment before stabilizing at a plateau. In addition, treatment with SGLT2 inhibitors was frequently associated with a reduced need for magnesium supplementation. In the reviewed literature, data on safety and adverse effects were limited. The included studies had important methodological limitations, including small sample sizes and a predominantly retrospective observational design. Therefore, further well-designed prospective studies are required to confirm the observed effects and better characterize the safety profile of SGLT2 inhibitors in this population.

Keywords:
Hypomagnesemia, SGLT2-inhibitors, Magnesium, Empagliflozin, Dapagliflozin, Non-diabetic

Introduction

Hypomagnesemia is defined as an electrolyte disturbance characterized by a decreased serum level of magnesium. However, thresholds for magnesium deficiency vary significantly depending on the source (Gragossian et al., 2026). Currently, no international consensus regarding the magnesemia reference range exists. Existing reference intervals for the magnesium serum level range between 0.75 mmol/L and 0.96 mmol/L. However, most of the currently utilised lower cut-off values for magnesium serum level in the clinical setting are set too low, resulting in the underdiagnosis of hypomagnesemia. To avoid this, a revised lower limit of 0.85 mmol/L for physiological magnesium levels has been recently proposed (Costello et al., 2016; Rosanoff et al., 2022). Consequently, we decided to adopt this cutoff to determine whether patients could be diagnosed as hypomagnesemic.

Low magnesium status leads to aggravation of numerous chronic diseases, among others, cardiovascular diseases, neoplastic diseases, neurological diseases and diabetes (Fiorentini et al., 2021). The prevalence of hypomagnesemia ranges between 2.5% and 15% in the general population (Gragossian et al., 2026). Considering its high prevalence and associated adverse health consequences, it seems crucial to implement effective diagnosis and treatment of hypomagnesemia. However, hypomagnesemia remains an under-recognised condition (Salinas et al., 2024). Moreover, global consensus on the management of hypomagnesemia remains elusive, particularly for asymptomatic patients (Adomako & Yu, 2024).

Overall, treatment of hypomagnesemia depends on the severity of the condition. According to ERC guidelines, in severe cases of hypomagnesemia (magnesium serum level <0.6 mmol/L)

involving life-threatening arrhythmias, the administration of a 2 g intravenous bolus of magnesium sulfate is recommended (Lott et al., 2025). In cases of severe hypomagnesemia without concurrent arrhythmias, administration of 1-2 g of intravenous magnesium sulfate over 5-60 minutes followed by continuous, 24 hours infusion of 4-8 g of magnesium sulfate is warranted (Adomako & Yu, 2024; Floris et al., 2025).

In mild forms of hypomagnesemia or in asymptomatic, non-hospitalised patients, oral forms of magnesium administration are preferred (Floris et al., 2025; Gragossian et al., 2026; Salinas et al., 2024).

In cases of chronic magnesium depletion, supplementation and limiting of the renal magnesium wasting remain the mainstay of the therapy. To date, blockers of the epithelial sodium channel have been identified as effective agents for reducing renal magnesium excretion, leading to a subsequent increase in serum magnesium concentrations (Tseng et al., 2022).

Recently, the literature has documented the potential of sodium-glucose cotransporter 2 inhibitors (SGLT2 inhibitors, gliflozins, SGLT2i) to mitigate hypomagnesemia through a mechanism of reduced renal magnesium clearance. Interestingly, despite their primary role in diabetes, gliflozins have been found effective in reducing hypomagnesemia regardless of patients' glycemic status.

The aim of this study is to provide a comprehensive summary and critical assessment of the current evidence regarding the efficacy of SGLT2i in the management of hypomagnesemia in non-diabetic subjects.

Methods and materials

An extensive literature search was conducted using the PubMed, Scopus, ScienceDirect, and Google Scholar databases, with a primary focus on studies published within the last five years. The search strategy included the following terms and their combinations: "SGLT2 inhibitors", "flozins", "dapagliflozin", "empagliflozin", "canagliflozin", "hypomagnesemia", "magnesium deficiency", "serum magnesium", and "non-diabetic patients".

Randomized controlled trials, post-hoc analyses of randomized controlled trials, observational studies, case series, and case reports concerning adult patients with a confirmed diagnosis of hypomagnesemia, excluding patients with type 2 diabetes mellitus, were included.

Eligible interventions consisted of treatment with SGLT2 inhibitors, administered either as monotherapy for hypomagnesemia or in combination with intravenous or oral magnesium

supplementation. The primary outcome of interest was improvement in serum magnesium concentration. Secondary outcomes included discontinuation or reduction of magnesium supplementation.
Studies were excluded if only conference abstracts were available, if full-text manuscripts could not be accessed, or if the original article was not available in English. Duplicate publications and studies unrelated to magnesium homeostasis were also excluded.

Due to the limited evidence on the use of SGLT2 inhibitors in non-diabetic patients with hypomagnesemia, studies including mixed patient populations were also considered. These studies were included when they provided high-quality evidence or clinically relevant information on the effects of SGLT2 inhibitors on magnesium homeostasis. Their inclusion allowed for a broader assessment of the impact of SGLT2 inhibitors on serum magnesium levels and enabled comparison between diabetic and non-diabetic populations.

A total of seven studies met the eligibility criteria and were included in this narrative review.

Results

Post-hoc analysis of randomized controlled studies

In a post-hoc analysis of the EMPEROR-Reduced trial, an increase in serum magnesium levels was observed across the study population. EMPEROR-Reduced was a randomized, double-blind, placebo-controlled trial in which 3,730 patients with class II, III, or IV heart failure and an ejection fraction of 40% or less received empagliflozin (10 mg once daily) or placebo, in addition to recommended HFrEF therapy (Packer et al., 2020a). In the post-hoc analysis, patients were stratified into quintiles according to their baseline serum magnesium levels. The study authors noted that only the patients in the lowest quintile fell into the category of hypomagnesemia, with baseline magnesium serum levels of 0.68 ± 0.07 mmol/L. However, applying the hypomagnesemia cutoff postulated by Rosanoff et al. (0.85 mmol/L), patients in the second and the third quintiles with magnesium serum levels of 0.79 ± 0.02 mmol/L and 0.84 ± 0.01 mmol/L, accordingly, could also be classified as hypomagnesemic. Regarding the non-diabetic population, the first quintile consisted of 878 participants (267 [30.4%] non-diabetic), the second of 724 (375 [51.8%] non-diabetic) and the third of 668 (379 [56.7%] non-diabetic).

On average, throughout the follow-up period, empagliflozin increased serum magnesium levels by 0.05 mmol/L. The effects of empagliflozin were more pronounced in the first quintile with magnesium level increase of 0.07 mmol/L compared to 0.04 mmol/L and 0.05

mmol/L in the second and third quintiles, respectively. Notably, randomization to empagliflozin was found to be the strongest independent predictor of a serum magnesium increase with OR = 3.36, 95% CI: 2.91-3.88, $p < 0.001$. Although the authors reported the mean magnesium increase within each quintile, separate statistics for the diabetic and the non-diabetic populations were not provided. However, diabetes status was associated with a higher OR of experiencing a magnesium serum level increase relative to non-diabetic population (OR = 1.35, 95% CI: 1.17-1.56, $p < 0.001$) (Ferreira, Anker, et al., 2026). Such statistical data suggest that empagliflozin was more effective in elevating serum magnesium levels among diabetic patients, nonetheless it potentially retained its efficacy in non-diabetic patients.

In a similarly structured post-hoc analysis derived from the EMPEROR-Preserved trial, the authors reported a consistent effect of empagliflozin on serum magnesium levels. EMPEROR-Preserved was a randomized, double-blind, placebo-controlled trial. A total number of 5,988 patients diagnosed with chronic HF with NYHA functional class II, III and IV, a left ventricular ejection fraction >40% and elevated N-terminal pro-hormone B-type natriuretic peptide levels received empagliflozin (10 mg once daily) or placebo, in addition to their usual therapy (Anker et al., 2021). In a post hoc analysis, similarly to the previous study, patients were stratified into quintiles according to their baseline serum magnesium levels. Mean serum magnesium concentration was 0.67 ± 0.07 mmol/L, 0.78 ± 0.02 mmol/L and 0.83 ± 0.01 mmol/L in the first, second and third quintiles, respectively. Applying the aforementioned lower cutoff for physiological serum magnesium levels, patients in those quintiles would classify as hypomagnesemic. Regarding the non-diabetic population, the first quintile consisted of 1,291 participants (293 [22,7%] non-diabetic), the second of 1,189 (544 [45.7%] non-diabetic), and the third of 1,378 (801 [58.1%] non-diabetic).

The results mirrored those observed in the post-hoc analysis of the EMPEROR-Reduced study. Serum magnesium levels increased by an average of 0.05 mmol/L in patients receiving empagliflozin. Similarly to the EMPEROR-Reduced study, the increase was more substantial in the first quintile than in the second and third quintiles. Randomization to empagliflozin remained the strongest independent predictor of a rise in the serum magnesium level (Ferreira, Packer, et al., 2026).

A notable strength of both studies is the utilization of data from large-scale, high-quality randomized controlled trials. Moreover, a statistically significant rise in serum magnesium was observed across three quintiles comprising patients eligible for a hypomagnesemia diagnosis in the two studies. However, in the context of empagliflozin's effect on serum

magnesium levels within non-diabetic patients, several limitations must be acknowledged. First of all, a primary objective of the studies was to analyse the association of serum magnesium levels with the outcomes of the EMPEROR-Reduced and EMPEROR-Preserved trials and assess the impact of empagliflozin on serum magnesium independently from the glycemic status in the population under study. A lack of data stratification for the diabetic and non-diabetic populations regarding a mean magnesium increase limits the ability to determine the precise magnitude of this increment in non-diabetic patients. Secondly, the EMPEROR-Reduced and EMPEROR-Preserved trials by design evaluated patients with heart failure, thus restricting the extrapolation of their results to the general population. Lastly, since the intervention in the studies was limited to the use of empagliflozin, conclusions regarding its efficacy in elevating the serum magnesium level cannot be directly extended to other SGLT2 inhibitors.

Case studies

Shah et al. reported a case series of four non-diabetic patients with severe refractory hypomagnesemia who were treated with SGLT2 inhibitors. The cohort comprised three women and one man, with three patients in their 50s and one in their 70s. The causes of hypomagnesemia varied across cases. In one patient, hypomagnesemia was associated with calcineurin inhibitor therapy following orthotopic liver transplantation, whereas in the remaining three patients, platinum-based chemotherapy was considered the primary contributing factor. Nevertheless, gastrointestinal losses could not be excluded as an additional mechanism contributing to hypomagnesemia.

Among the included cases, the patient receiving calcineurin inhibitor therapy was treated with empagliflozin at a dose of 25 mg/day, while the remaining patients received dapagliflozin at a dose of 10 mg/day. Initiation of SGLT2 inhibitor therapy was associated with a significant improvement in serum magnesium concentrations in all patients. Mean serum magnesium levels increased, with improvements ranging from 0.08 to 0.19 mmol/L. Specifically, serum magnesium increased by 0.08 mmol/L in case 1 (from 0.66 ± 0.10 to 0.74 ± 0.03 mmol/L; P = 0.02), 0.19 mmol/L in case 2 (from 0.63 ± 0.07 to 0.82 ± 0.12 mmol/L; $p < 0.001$), 0.18 mmol/L in case 3 (from 0.51 ± 0.07 to 0.69 ± 0.07 mmol/L; $p < 0.001$), and 0.17 mmol/L in case 4 (from 0.63 ± 0.03 to 0.80 ± 0.08 mmol/L; $p < 0.001$). Furthermore, initiation of SGLT2 inhibitor therapy resulted in complete discontinuation of oral magnesium supplementation in one patient. Another patient achieved a 33.3% reduction in oral magnesium requirements together with withdrawal of intravenous magnesium sulfate supplementation. Intravenous

magnesium sulfate supplementation was also discontinued in the third patient, while the last patient demonstrated reduced requirements for intravenous supplementation. Despite favorable effects on electrolyte balance, in one of the patients, dapagliflozin was discontinued after one month of therapy because of a severe vaginal fungal infection. Following drug withdrawal, an increased requirement for magnesium supplementation was observed secondary to recurrent hypomagnesemia. The follow-up duration in the remaining patients ranged from 2 to 10 months (Shah et al., 2023).

A major strength of this study is the substantial increase in serum magnesium levels temporally associated with the initiation of SGLT2 inhibitor therapy. In all described patients, marked improvements in laboratory parameters were observed, accompanied by a significant reduction in magnesium supplementation. Another notable observation was the worsening of hypomagnesemia after SGLT2 inhibitor withdrawal in case 4. However, the findings are limited by the uncontrolled study design, the absence of a standardized definition of refractory hypomagnesemia, and the presence of potential confounding factors. The observational nature of the study does not allow the exclusion of spontaneous improvement in magnesium levels, changes in magnesium intake related to diet, alterations in renal function, or modifications in concomitant therapy. The likely medication-induced or gastrointestinal causes of hypomagnesemia in these patients limit the generalizability of the findings.

Ayub et al. in their case series also investigated the effect of SGLT2 inhibitors in treating patients with refractory hypomagnesemia resistant to supplementation. Among the five described patients, only a 67-year-old woman met the inclusion criterion of having a non-diabetic status. The cause of hypomagnesemia in this patient was linked to platinum-based chemotherapy administered for the treatment of triple-negative breast cancer.

The patient's baseline serum magnesium level was 0.37 mmol/L. Following the initiation of both oral and intravenous supplementation, the level increased to approximately 0.53-0.58 mmol/L. The patient received a total of 40 g of intravenous magnesium sulfate in 10 doses and an oral dose of 800 mg of magnesium oxide twice daily. During this period, oral supplementation was discontinued due to the occurrence of watery diarrhea. Empagliflozin (10 mg) was initiated as part of the treatment, after which the serum magnesium concentration reached 0.82 mmol/L. The exact time elapsed from the first dose to the maximum recorded level remains unknown. The patient was discharged after treatment and did not return for follow-up visits. Therefore, the long-term outcomes remain uncertain given the short-term observation period (Ayub et al., 2025).

Despite the promising outcomes observed in this patient, several limitations must be addressed. The retrospective nature of this case study means it can provide only preliminary clinical signals. Furthermore, the concomitant use of intensive magnesium supplementation and the short duration of follow-up make it difficult to determine the long-term efficacy of SGLT2 inhibitors for this specific case.

Further support for this notion comes from the work of Adella et al., who investigated the effect of dapagliflozin on hypomagnesemia in patients with autosomal dominant kidney hypomagnesemia caused by mutations in the Ras-related GTPase D (RRAGD) gene. Out of 13 patients, 6 were selected from the p.(Thr97Pro) *RRAGD* group who received dapagliflozin at a dose of 10 mg for 15 days. Following the treatment period, the mean increase in serum magnesium levels was 0.04 mmol/L. These findings suggest that SGLT2 inhibitors could partially reduce renal magnesium loss. However, a notable exacerbation of hypokalemia was also observed in these patients, representing a potential adverse effect of SGLT2i therapy (Adella et al., 2025).

Furthermore, while the findings offer a promising therapeutic perspective, the study is subject to several limitations. Firstly, the sample size was small (n = 6) and restricted exclusively to patients carrying the p.(Thr97Pro) mutation, which compromises the generalizability of the results. Secondly, the 15-day intervention period was relatively short, leaving the long-term efficacy, safety profile, and potential chronic adverse effects of dapagliflozin in this population to be determined. Lastly, the lack of comprehensive, standardized baseline characteristics for the analyzed cohort complicates direct comparisons with other clinical studies.

Cohort studies

The results reported by Carmine Secondulfo et al. were obtained from a retrospective, single-center observational study conducted as part of the Salerno CKD Cohort Study. The study investigated the impact of SGLT2 inhibitors on magnesium levels in kidney transplant recipients. The analysis included adult kidney transplant recipients (≥18 years) who had undergone transplantation at least one year prior to enrollment. The study cohort consisted predominantly of male patients (82.5%), with a median age of 55 years. The mean eGFR was 56 ± 19 mL/min/1.73 m². Most participants were non-diabetic (43/63, 68.3%). All participants received dapagliflozin at a dose of 10 mg/day as an add-on to their ongoing treatment. Clinical and laboratory evaluations were performed at baseline (T0), after 3 months (T1), and

after 6 months (T2) of dapagliflozin treatment. Hypomagnesemia was defined as a serum magnesium concentration <0.70 mmol/L. The study did not include a control group.

At baseline, magnesium supplementation was administered in 21 patients (33.3%). Low serum magnesium concentrations were found in 24 kidney transplant recipients (38.1%) at study entry. Importantly, half of the individuals with hypomagnesemia were not taking their prescribed oral magnesium supplementation because of gastrointestinal adverse effects or financial burden.

Mean serum magnesium concentration increased significantly over time in the whole cohort, rising by 0.04 mmol/L after 3 months of treatment (from 0.70 ± 0.09 to 0.74 ± 0.09 mmol/L; $p < 0.01$) and by a further 0.02 mmol/L after 6 months (0.77 ± 0.09 mmol/L; $p < 0.05$ vs. 3 months). Similar trends were observed in both patients with and without diabetes. In participants with diabetes, serum magnesium increased by 0.06 mmol/L after 3 months of dapagliflozin therapy (from 0.70 ± 0.08 to 0.76 ± 0.13 mmol/L; $p < 0.05$), whereas the additional increase observed after 6 months (0.77 ± 0.08 mmol/L) did not reach statistical significance. In participants without diabetes, serum magnesium increased significantly by 0.04 mmol/L after 3 months (0.70 ± 0.09 vs. 0.74 ± 0.07; $p < 0.01$) and by a further 0.02 mmol/L between 3 and 6 months (0.76 ± 0.09 mmol/L; $p < 0.05$). Overall, dapagliflozin therapy was associated with a consistent improvement in magnesium homeostasis across the study population. The prevalence of hypomagnesemia progressively decreased during follow-up. In the whole cohort, hypomagnesemia declined from 38.1% at baseline to 20.6% at T1 ($p = 0.019$) and to 15.9% at T2 ($p = 0.01$ vs. T0). A reduction was also observed in both diabetic and non-diabetic participants. Subgroup analysis demonstrated that the increase in serum magnesium from T0 to T1 was significantly greater in patients with baseline hypomagnesemia ($p < 0.05$), while no significant differences were found according to diabetes status, renal function, diuretic therapy, cyclosporine use, or tacrolimus use. A modest decline in eGFR was noted after 3 months of dapagliflozin treatment. However, no further decline in renal function was observed (Secondulfo et al., 2025).

Importantly, this study provides evidence on the effects of SGLT2 inhibitors on magnesium homeostasis in non-diabetic kidney transplant recipients. The findings suggest a potential therapeutic role for this drug class in the management of hypomagnesemia in this population. Although the results are encouraging, the overall level of evidence remains limited because of the retrospective observational design and several methodological limitations, including the absence of a control group and the relatively short observation period. Furthermore, the

generalizability of these findings is limited, as the study population consisted exclusively of kidney transplant recipients.

In a retrospective observational study conducted at Wakayama Medical University in Japan, Osawa et al. assessed the effects of SGLT2 inhibitor therapy on serum magnesium levels in patients with chronic kidney disease. The study included CKD patients treated with SGLT2 inhibitors between 2017 and 2022. After excluding patients with prior exposure to SGLT2 inhibitors, early treatment discontinuation, or incomplete magnesium data, 62 participants were included in the final analysis. Participants were stratified into tertiles according to baseline serum magnesium levels. Hypomagnesemia was defined as a serum magnesium concentration < 0.7 mmol/L. In the study population, the mean baseline serum magnesium concentration was 0.84 mmol/L. Hypomagnesemia was observed in five patients. No cases of hypermagnesemia were reported. Among the 62 participants, 30 (48.4%) had no diagnosis of diabetes mellitus. The prevalence of diabetes mellitus did not differ significantly between the magnesium tertile subgroups. Dapagliflozin and empagliflozin were the most frequently prescribed SGLT2 inhibitors.

Serum magnesium concentrations increased progressively during the 6-month follow-up period, with significant elevations already evident after 1 month of treatment ($p < 0.05$) and sustained increases observed at 3 and 6 months ($p < 0.001$). However, the increase became smaller over time, with only minor changes between months 3 and 6. According to analyses stratified by tertiles, patients with the lowest baseline magnesium concentrations showed the greatest increases in magnesium levels. This association was further supported by multivariable linear regression analysis, which identified baseline serum magnesium concentration as an independent negative predictor of magnesium change after 6 months of SGLT2 inhibitor therapy ($p < 0.001$). In the same model, the presence of diabetes was not a statistically significant factor. The median initial decline in eGFR following SGLT2 inhibitor initiation was modest (−1.1 mL/min/1.73 m²). No significant association was observed between the early reduction in eGFR and changes in serum magnesium concentrations. Among five patients with hypomagnesemia at baseline, four achieved normalization of serum magnesium levels after 6 months of treatment. No new cases of hypomagnesemia were observed after treatment initiation. However, asymptomatic hypermagnesemia was observed in two elderly patients during follow-up (Osawa et al., 2025).

The study suggests that SGLT2 inhibitor therapy may improve magnesium homeostasis in patients with CKD, including those without diabetes mellitus. The largest increases in serum

magnesium concentrations were observed in patients with the lowest baseline magnesium levels. Although the number of hypomagnesemic patients was small, these findings suggest a clinically relevant effect of SGLT2 inhibitors in patients with hypomagnesemia. However, the retrospective observational design, single-center setting, limited sample size and the inclusion of a highly specific patient population with chronic kidney disease should be considered when interpreting the results.

Discussion

To date, SGLT2i have been established in the treatment of type 2 diabetes mellitus, chronic heart failure and chronic kidney disease. However, due to their pleiotropic effects, the potential application of SGLT2i extends beyond these indications, including the management of hypomagnesemia (American Diabetes Association Professional Practice Committee for Diabetes* et al., 2026; McDonagh et al., 2023; Stevens et al., 2024). Although the molecular mechanisms by which SGLT2 inhibitors influence magnesium homeostasis remain elusive, several hypotheses have been proposed. Firstly, SGLT2i enhance transient receptor potential cation channel subfamily M member 6 (TRPM6). TRPM6 mediates luminal magnesium uptake in the distal convoluted tubule (DCT), accounting for 5%-10% of the filtered magnesium load (Schäffers et al., 2018; Shah et al., 2024). Although this fraction seems insignificant, it is of critical importance, as the reabsorption in DCT is a final regulatory step, determining total urinary magnesium loss (Schlingmann et al., 2007). As TRPM6 is also expressed in cecal and colon cells, its enhanced activity results in increased magnesium absorption from the large intestine (Luongo et al., 2018). Notably, TRPM6 expression is stimulated by insulin via phosphoinositide 3-kinase (Nair et al., 2012). Although SGLT2i decrease the secretion of insulin, they simultaneously increase tissue insulin sensitivity, potentially leading to elevated TRPM6 expression (Wu et al., 2025). Moreover, SGLT2 inhibitors upregulate epidermal growth factor (EGF) secretion, which subsequently enhances TRPM6 expression (Ikari et al., 2008; Sen et al., 2023).

Lastly, SGLT2 inhibition in proximal tubules results in an increased luminal sodium concentration and consequently, a higher transmembrane potential gradient (TPMG). Since passive reabsorption of magnesium in the thick ascending limb of the loop of Henle is driven by TPMG, SGLT2 inhibitors enhance magnesium retention (Shah et al., 2024).

The described molecular theory provides a plausible explanation for why SGLT2 inhibitors are effective in elevating serum magnesium levels. However, it explains neither the pronounced efficacy of SGLT2i in cases of severe hypomagnesemia, nor the prominent

response upon treatment initiation followed by a plateau. Assuming a crucial impact of TRPM6 on magnesium homeostasis, an in vivo study assessing the dynamics of TRPM6 upregulation following SGLT2i implementation would be necessary to elucidate the underlying mechanism.

Another important issue is whether the magnesium-enhancing effect of SGLT2i investigated in the reviewed literature can be extrapolated to other SGLT2 inhibitors. In the evaluated studies, only empagliflozin and dapagliflozin were used in the treatment, restricting the generalizability of the findings. However, taking into consideration the proposed molecular mechanism of SGLT2 inhibitors, their impact on the pathways regulating magnesium reabsorption should remain consistent regardless of the chosen agent within this class. This is supported by the literature, suggesting that elevating serum magnesium levels constitutes a class effect of SGLT2 inhibitors (Shah et al., 2024; Zhang et al., 2022).

The benefits of introducing SGLT2i as treatment modality in the non-diabetic, hypomagnesemic population must be weighed against potential side effects in patients without approved indications. The use of SGLT2 inhibitors in non-diabetic patients is associated with a range of adverse effects, with the most serious including extracellular volume depletion leading to hypotension, genital bacterial or fungal infections and a transient decline in the glomerular filtration rate. While the incidence of hypoglycemia with SGLT2 inhibitors is similar to that of placebo, non-diabetic patients experience approximately a 3-fold lower rate of hypoglycemic episodes compared to diabetic cohorts (Heerspink et al., 2020; Packer et al., 2020b). However, compared with patients with type 2 diabetes mellitus, non-diabetic individuals rarely or never experience certain serious adverse events, such as severe ketoacidosis and complicated urinary tract infections (Neal et al., 2017; Zinman et al., 2015). In the analyzed studies, the only reported adverse effects included a severe vaginal fungal infection and the exacerbation of hypokalemia in a specific group of patients carrying the p.(Thr97Pro) mutation in the *RRAGD* gene (Adella et al., 2025; Shah et al., 2023). The remaining studies did not report any registered adverse effects, however, eligible post-hoc analyses by design did not focus on the safety profile of SGLT2i. Upon investigating the primary source trials, adverse effects including the aforementioned effects and hypoglycemic episodes, bone fractures and rare events leading to lower limb amputation are documented. However, only hypoglycemic episodes were stratified by the patients glycemic status, leaving uncertainty regarding the incidence of the remaining adverse effects in a non-diabetic population (Anker et al., 2021; Packer et al., 2020a). At present, the addition of an SGLT2

inhibitor to supplementation may be considered for refractory hypomagnesemia due to its acceptable safety profile in non-diabetic patients.

The clinical management of hypomagnesemia remains a significant therapeutic challenge, largely constrained by the limitations of conventional supplementation. Apart from its accessibility, oral supplementation is characterized by limited efficacy due to several factors. Firstly, magnesium can be administered in various oral forms, including magnesium oxide, citrate, lactate, gluconate, or bisglycinate. The bioavailability of these distinct supplemental forms varies significantly. Frequently utilized formulations containing magnesium oxide exhibit by far the poorest absorption rates, often dropping below 4%-5% (Blancquaert et al., 2019). In contrast, organic salts demonstrate a substantially superior absorption profile of up to 20%-30%, although the required cumulative doses remain high. Secondly, magnesium that is not absorbed in the small intestine may induce osmotic diarrhea. This effect is highly dependent on both the dose and the chemical form used (Pardo et al., 2021). Furthermore, it should be noted that supplemental therapy acts solely as a symptomatic intervention. Consequently, the development of severe side effects frequently compromises long-term compliance. To address this therapeutic challenge, SGLT2 inhibitors emerge as a promising modality that could significantly complement the treatment of hypomagnesemia in patients with heart failure, type 2 diabetes, or chronic kidney disease, but potentially expanding to other clinical contexts as well. A particularly promising application involves the potential use of SGLT2 inhibitors in cases of acute chemotherapy-induced hypomagnesemia. In this clinical scenario, alongside the standard intravenous administration of magnesium sulfate, the use of agents that mitigate renal magnesium wasting caused by nephron damage may prove highly beneficial due to their relatively fast onset of action (Mathavan et al., 2026). The potential application of these agents extends to managing hypomagnesemia in malabsorption syndromes, where SGLT2i may enhance compensatory colonic magnesium absorption by upregulating TRPM6 expression in the large intestine. The pleiotropic effects of SGLT2 inhibitors, characterized by the modulation of renal hemodynamics and the activation of alternative ion transport pathways, position this class of drugs as potential modulators of electrolyte homeostasis across a broad spectrum of magnesium-wasting nephropathies.

Despite the accumulated evidence, several limitations of this review should be noted. To date, there are no randomized controlled studies (RCTs) focusing exclusively on evaluating the efficacy of SGLT2i in the treatment of hypomagnesemia in non-diabetic patients. All of the available evidence stems from post-hoc analyses of RCTs, observational studies and case reports. Furthermore, the baseline characteristics of enrolled patients represent a notable

limitation. Each study evaluated cohorts presenting with specific, major comorbidities including HFpEF, HFrEF, CKD, neoplastic and genetic diseases or included kidney or liver transplant recipients. To our knowledge, currently there are no studies evaluating the efficacy of SGLT2i in isolated hypomagnesemia in non-diabetic settings. However, since hypomagnesemia is typically secondary to other conditions and rarely occurs in an isolated form, recruiting a cohort free of confounding comorbidities would be extremely challenging. Due to the presented limitations, the conclusions of this study should be treated only as hypothesis-generating. Ultimately, we identified a research gap that can be addressed by conducting large-scale, prospective studies, preferably in the form of RCTs.

Conclusions

The primary objective of this study was to compile and analyze the literature regarding the efficacy of SGLT2i in elevating serum magnesium levels in hypomagnesemic, non-diabetic patients. Based on the gathered body of evidence, several relevant conclusions should be highlighted:

1) SGLT2 inhibitors elevate serum magnesium levels by an average of 0.04-0.19 mmol/L in a non-diabetic population.
2) The magnesium-elevating effect of SGLT2i was especially pronounced in cases of severe hypomagnesemia.
3) The magnesium-elevating effect of SGLT2i was especially marked upon treatment initiation, followed by a sustained plateau.
4) The initiation of SGLT2i allowed reduction or discontinuation of magnesium supplementation in some instances.

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