

Effects of Magnesium Coadministration on Thyroid Function and Autoantibody Titers in Euthyroid Hashimoto's Thyroiditis

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ABSTRACT

Objective: Although magnesium (Mg) is integral to numerous metabolic and immunological processes, its influence in Hashimoto's thyroiditis (HT) has not been fully elucidated. The present study sought to examine the effects of oral Mg supplementation on thyroid function and thyroid autoantibody in hypomagnesemic, euthyroid individuals with HT who were receiving a stable levothyroxine (LT4) regimen.

Methods: In this retrospective observational analysis, adult patients with HT who were euthyroid and had documented hypomagnesemia were evaluated after receiving oral Mg supplementation for a minimum of two months, during which LT4 dosing remained unchanged. All patients received oral Mg oxide equivalent to 365 mg elemental Mg daily. Serum thyroid-stimulating hormone (TSH), free thyroxine (fT4), free triiodothyronine (fT3), anti-thyroid peroxidase (anti-TPO), anti-thyroglobulin (anti-TG), and Mg levels were measured both prior to and following supplementation. Pre- and post-treatment values were compared using paired statistical methods.

Results: A total of 45 patients (82% female, mean age 49.3 ± 15.7 years) were analyzed. After two months of Mg supplementation, serum Mg levels increased significantly (p = 0.04). Serum TSH levels also showed a statistically significant increase (p = 0.03), while remaining within the euthyroid range. No significant changes were observed in fT4, fT3, anti-TPO, or anti-TG titers.

Conclusion: In hypomagnesemic euthyroid patients with HT receiving stable LT4 therapy, short-term Mg supplementation was associated with a modest but statistically significant increase in TSH levels, while thyroid hormone levels and autoantibody titers remained unchanged. This finding may reflect reduced LT4 bioavailability during concomitant Mg administration rather than a direct effect on the hypothalamic-pituitary-thyroid axis.

Keywords: Hashimoto's thyroiditis, Hypomagnesemia, Magnesium supplementation, Thyroid-stimulating hormone, Autoimmune thyroid disease

Ötiroid Hashimoto Tiroiditinde Magnezyum Replasmanının Tiroid Fonksiyonları ve Otoantikör Düzeyleri Üzerine Etkileri

Araştırma Makalesi

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ÖZ

Amaç: Magnezyum (Mg), hücrel metabolizma ve immün düzenleme süreçlerinde önemli bir rol oynamasına karşın, Hashimoto tiroiditinde (HT) tiroid fonksiyonları ve otoimmünite üzerindeki etkileri yeterince bilinmemektedir. Bu çalışmada; sabit levotiroksin (LT4) tedavisi altındaki hipomagnezemik ve ötiroid HT hastalarında oral Mg replasmanının tiroid fonksiyon testleri ve tiroid otoantikör düzeyleri üzerindeki etkilerinin değerlendirilmesi amaçlandı.

Yöntem: Bu retrospektif gözlemsel çalışmaya, en az iki ay süreyle oral Mg desteği alan, LT4 dozu sabit olan, hipomagnezemi olan ötiroid HT tanılı erişkin hastalar dahil edildi. Mg tedavisi öncesi ve sonrası tiroid stimulan hormon (TSH), serbest tiroksin (sT4), serbest triiyodotironin (sT3), anti-tiroid peroksidaz (anti-TPO), anti-tiroglobulin (anti-TG) ve serum Mg düzeyleri değerlendirildi. Tedavi öncesi ve sonrası değerler eşleştirilmiş istatistiksel yöntemler kullanılarak karşılaştırıldı. Tüm hastalara günlük 365 mg elemental Mg'ye eşdeğer olacak şekilde oral magnezyum oksit tedavisi uygulanmıştı.

Bulgular: Toplam 45 hasta (ortalama yaş 49,3 ± 15,7 yıl; %82 kadın) analiz edildi. İki aylık Mg replasmanı sonrasında serum Mg düzeylerinde anlamlı artış saptandı (p = 0,04). Serum TSH düzeylerinde de istatistiksel olarak anlamlı bir artış gözlenmekle birlikte (p = 0,03), tüm değerler ötiroid referans aralığında kaldı. Serbest tiroid hormonları ile anti-TPO ve anti-TG düzeylerinde anlamlı bir değişiklik izlenmedi.

Sonuç: Stabil LT4 tedavisi altında izlenen hipomagnezemik, ötiroid HT hastalarında kısa süreli Mg replasmanı, tiroid hormon düzeyleri ve otoantikör titreri üzerinde belirgin bir etki oluşturmaksızın TSH düzeylerinde hafif ancak anlamlı bir artışla ilişkilidir. Bulgular, Mg replasmanının hipotalamo-hipofizer-tiroid aks üzerinde doğrudan bir etkiden ziyade, eşzamanlı Mg kullanımı sırasında LT4 biyoyararlanımının azalmasını yansıtır olabilir.

Anahtar Kelimeler: Hashimoto tiroiditi, Hipomagnezemi, Magnezyum replasmanı, Tiroid stimulan hormon, Otoimmün tiroid hastalığı

Introduction

Hypothyroidism affects roughly 5% of the population in regions with sufficient iodine intake, and shows a marked predominance in women, with female-to-male ratios approaching 5:1. Within this context, chronic autoimmune thyroiditis—commonly known as Hashimoto's thyroiditis (HT)—represents the leading cause of primary hypothyroidism. The disorder arises from immune-driven injury to thyroid follicular cells, resulting in a progressive decline in the gland's capacity to synthesize thyroid hormones.¹

The diagnosis of HT is established through an integrated assessment of clinical features, thyroid function tests, thyroid autoantibodies, and thyroid ultrasonography. Typical ultrasonographic findings include diffuse parenchymal heterogeneity, reduced echogenicity of the thyroid gland, and a pseudonodular appearance. Among circulating autoantibodies, anti-thyroid peroxidase (anti-TPO) represent the most sensitive serological marker and are detected in approximately 90–95% of affected patients. Anti-thyroglobulin (anti-TG) show lower diagnostic sensitivity but may provide supportive evidence, particularly in patients who are anti-thyroid peroxidase negative.²

The management of Hashimoto's thyroiditis relies on levothyroxine (LT4) replacement therapy, which is individualized according to patient age, serum thyroid-stimulating hormone (TSH) concentrations, and clinical symptom burden.³ The primary objective of LT4 therapy is to maintain serum TSH levels within the reference range, thereby preserving a euthyroid state and alleviating symptoms related to hypothyroidism. LT4 functions as a prohormone and exerts its biological effects following peripheral conversion to triiodothyronine. Orally administered LT4 is absorbed predominantly in the jejunum and ileum, with an estimated bioavailability of 60–80%, a process that requires adequate gastric acidity. Consequently, LT4 absorption may be adversely affected by several dietary components and concomitant medications, including calcium salts, iron preparations, proton pump inhibitors, and bile acid sequestrants, which may reduce therapeutic efficacy despite stable dosing.⁴

Magnesium (Mg), ranking as the fourth most prevalent cation in the human body, participates in numerous enzymatic reactions and fundamental cellular functions. It is indispensable for processes such as neuromuscular activity, nucleic acid synthesis, oxidative phosphorylation, and a variety of intracellular signaling mechanisms, underscoring its central role in cellular energy homeostasis.⁵ Although Mg is primarily obtained through dietary intake, insufficient Mg consumption is common worldwide, and the reported prevalence of hypomagnesemia ranges from approximately 2.5% to 15%, depending on the population.⁶

Reduced Mg availability has been linked to a heightened propensity for inflammatory states. Mg is closely linked to immune system function, and experimental studies have demonstrated that intracellular free Mg acts as an important second messenger in lymphocytes, contributing to lymphocyte activation and immune signaling pathways. In particular, Mg has been shown to be involved in immune cell activation and regulation at the cellular level.⁷

Magnesium supplementation is commonly implemented in routine clinical practice, both to correct deficiency states and as a dietary adjunct. Despite its widespread use, the impact of Mg repletion on thyroid physiology and autoimmune activity has not been clearly established, and current evidence remains limited.⁸ Against this background, the present study was designed to assess

the effects of Mg replacement on thyroid function parameters and thyroid autoantibody titers in patients with hypomagnesemia and HT.

Methods

The study was conducted using a retrospective observational design and received approval from the Ethics Committee of the Faculty of Medicine at Süleyman Demirel University (Meeting Date: February 20, 2025; Meeting No: 92; Decision No: 16).

Adult individuals aged 18 years or older with a diagnosis of HT who were euthyroid at baseline and followed at the endocrinology outpatient clinic of Burdur State Hospital between January 2021 and January 2025 were evaluated for study inclusion. Eligible participants were those with documented hypomagnesemia who had received oral Mg supplementation for at least two months. LT4 dosing remained constant throughout the observation period; however, because of the retrospective nature of the study, the temporal separation between levothyroxine and magnesium administration could not be standardized. All patients received oral Mg oxide equivalent to 365 mg elemental Mg daily.

Study Population: Inclusion and Exclusion Criteria

Participants were considered eligible if they were 18 years of age or older and met the diagnostic criteria for HT, which required the presence of at least one of the following: positivity for anti-TPO, positivity for anti-TG, or characteristic ultrasonographic findings such as heterogeneous parenchymal texture, diffuse hypoechogenicity, or a pseudonodular pattern. All included patients were euthyroid at baseline, with serum thyroid-stimulating hormone (TSH) and free thyroxine (fT4) levels within laboratory reference ranges, and were receiving ongoing levothyroxine therapy at a stable dose, with no dose adjustments before or after the initiation of Mg supplementation. Baseline hypomagnesemia was required for inclusion and was defined as a serum Mg level below the laboratory reference range. Eligible patients had received oral Mg replacement therapy for a minimum duration of two months, using preparations and dosages consistent with current clinical guidelines and routine endocrinology practice.⁹ Availability of thyroid function tests, including at least TSH and optionally fT4 and/or fT3, both before and at two months after Mg supplementation was required, and thyroid autoantibody measurements (anti-TPO and/or anti-TG) were included when available as part of routine clinical follow-up.

Patients were excluded if they had overt hypothyroidism or levothyroxine drug-associated hyperthyroidism at baseline, experienced any change in levothyroxine dosage during the observation period, or used antithyroid medications. Additional exclusion criteria included a diagnosis of Graves' disease, subacute thyroiditis, or postpartum thyroiditis, as well as severe impairment of renal function, characterized by an estimated glomerular filtration rate below 30 mL/min/1.73 m², reliance on renal replacement therapy, or exposure to intravenous Mg treatment. Patients using medications known to significantly interfere with thyroid function or Mg metabolism, including amiodarone or lithium, were also excluded, as were those who were pregnant or in the postpartum period. Moreover, patients receiving vitamin D supplementation were not included in the study population in order to avoid potential confounding effects on thyroid autoantibody titers and thyroid function parameters. Finally, patients with acute systemic illness, active malignancy,

hospitalization during the study period, or incomplete laboratory data—such as missing baseline or follow-up thyroid function tests or an oral magnesium treatment duration shorter than two months—were not included in the analysis.

Laboratory Assessments

Serum concentrations of Mg, anti-TPO, anti-TG, TSH, fT4, and fT3 were assessed in the central biochemistry laboratory using chemiluminescent immunoassays on the Atellica™ IM Analyzer (Siemens Healthineers, Germany). TSH was assessed using a third-generation ultrasensitive assay (Atellica IM TSH3-UL; reference range 0.27–4.20 mIU/L). fT4 and fT3 were assessed by competitive chemiluminescent immunoassays, with reference ranges of 0.93–1.70 ng/dL and 2.0–4.4 pg/mL, respectively. Anti-TPO and anti-TG antibodies were assessed by chemiluminescent immunoassays, with values <34 IU/mL and <115 IU/mL considered negative. Serum Mg concentrations were assessed using routine automated colorimetric methods, and hypomagnesemia was defined as a serum Mg level below the institutional laboratory reference range (1.8–2.5 mg/dL). All laboratory analyses were conducted in accordance with the manufacturer’s recommendations and established quality assurance protocols.

Statistical Analysis

Statistical evaluation was carried out using IBM SPSS Statistics software (version 26.0; IBM Corp., Armonk, NY, USA). The distribution of continuous variables was examined using the Shapiro–Wilk test. Data with a normal distribution were summarized as mean ± standard deviation, while variables showing non-normal distribution were reported as median with

interquartile range.

Comparisons of laboratory measurements obtained at baseline and after two months of magnesium supplementation were conducted using the paired t-test for normally distributed variables and the Wilcoxon signed-rank test for variables with skewed distributions. Statistical significance was defined as a two-sided p value below 0.05.

Results

A total of 45 hypomagnesemic euthyroid patients with HT were included in the study, of whom 37 (82%) were female and 8 (18%) were male. The mean age of the study population was 49.3 ± 15.7 years (range: 22–65 years). All patients were receiving LT4 therapy at a stable dose throughout the study period.

The median daily LT4 dose was 50 µg (interquartile range: 50–100 µg; range: 25–300 µg/day). Anti-TPO and anti-TG positivity was observed in 43 (96%) and 13 (29%) patients, respectively. Both antibodies were positive in 13 patients (29%), whereas 2 patients (4.4%) were negative for both antibodies.

Following a two-month supplementation period, thyroid autoantibody levels as well as fT4 and fT3 concentrations remained unchanged. Conversely, a statistically significant rise was observed in both serum TSH and Mg levels after Mg therapy (p = 0.03 and p = 0.04, respectively). Laboratory parameters obtained at baseline and after treatment are presented in Tables 1 and 2, while the corresponding changes are depicted in Figures 1 and 2.

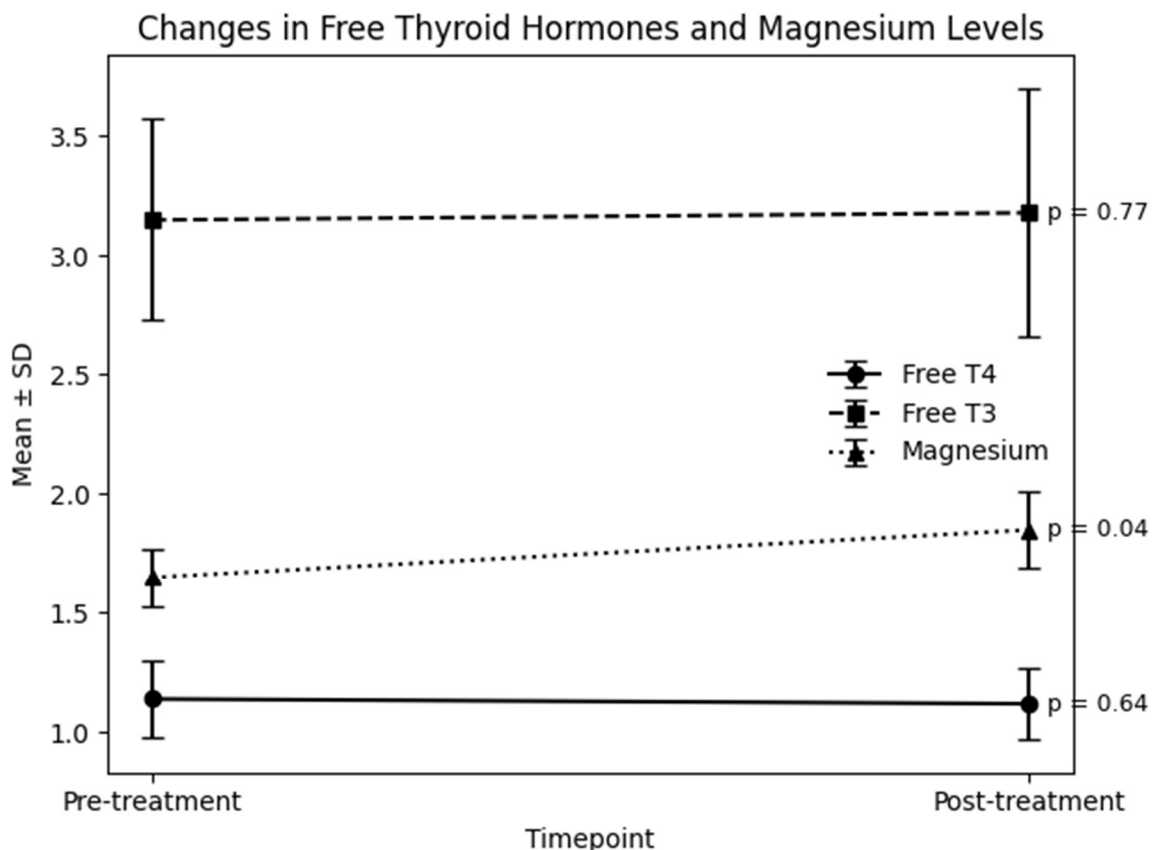


Figure 1. Changes in free thyroid hormones and serum magnesium levels before and after magnesium supplementation. Data are presented as mean ± standard deviation. P values represent paired comparisons between pre- and post-treatment measurements

Changes in TSH and Thyroid Autoantibody Levels Before and After Magnesium Supplementation

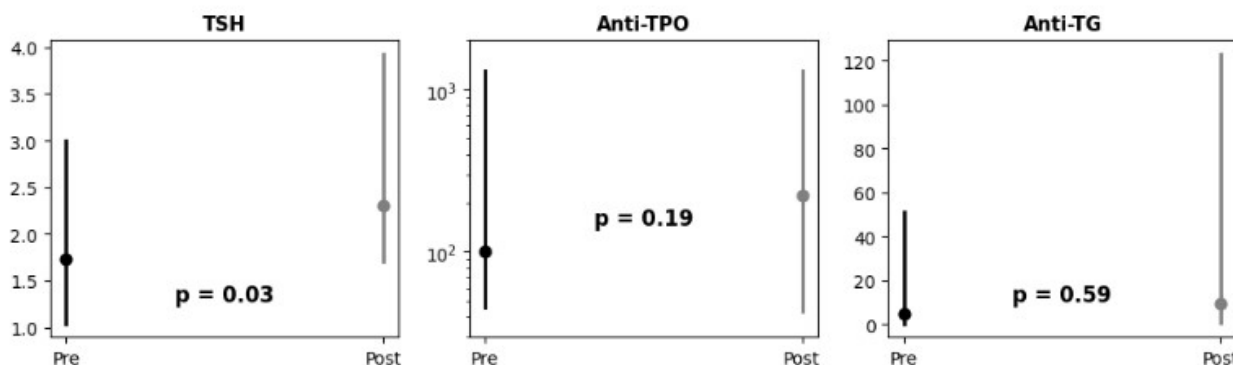


Figure 2. Schematic plots showing median and interquartile range (IQR) for serum TSH, anti-TPO, and anti-TG levels before and after magnesium supplementation. Points indicate median values and vertical lines represent the IQR. P values were derived from Wilcoxon signed-rank tests.

Table 1. Changes in Free Thyroid Hormones and Magnesium Levels Before and After Magnesium Supplementation

Parameter	Pre-treatment	Post-treatment	Mean difference	95% CI	p value
Free T4 (ng/dL)	1.14 ± 0.16	1.12 ± 0.15	-0.02	-0.06 to +0.10	0.64
Free T3 (pg/mL)	3.15 ± 0.42	3.18 ± 0.52	+0.03	-0.26 to +0.19	0.77
Magnesium (mg/dL)	1.65 ± 0.12	1.85 ± 0.16	+0.2	+0.08 to +0.32	0.04

Table 2. Changes in TSH and Thyroid Autoantibody Levels Before and After Magnesium Supplementation

Parameter	Pre-treatment	Post-treatment	p value
TSH (mIU/L)	1.73 (1.04-3)	2.3 (1.7-3.93)	0.03
Anti-TPO (IU/mL)	101 (46-1300)	223 (43-1300)	0.19
Anti-TG (IU/mL)	5 (0.4-51)	9.5 (0.9-123)	0.59

*Data are presented as mean ± standard deviation (SD) for normally distributed variables and as median (25th–75th percentile) for non-normally distributed variables.
 * Non-normally distributed variables were analyzed using the Wilcoxon signed-rank test and are presented as median (25th–75th percentile). Median differences and confidence intervals were not calculated due to the non-parametric nature of the statistical test.

Discussion

The relationship between Mg and thyroid gland physiology remains incompletely understood, and available clinical evidence is limited. To our knowledge, the impact of Mg replacement on thyroid function parameters and autoantibody profiles in hypomagnesemic, euthyroid individuals with HT has not previously been evaluated using a comparable retrospective approach. In the present cohort, Mg supplementation was associated with a statistically significant increase in serum TSH concentrations. However, TSH levels remained within the reference euthyroid range. No significant changes were detected in FT4, FT3, or thyroid autoantibody titers after treatment.

Magnesium exerts immunomodulatory effects and plays a regulatory role in anti-inflammatory and antioxidant pathways. Mg deficiency has been associated with immune dysfunction, increased inflammatory responses, and reduced antioxidant capacity.¹⁰ Experimental and clinical studies have shown that Mg repletion has been suggested to dampen inflammatory signaling cascades, potentially through suppression of nuclear factor kappa B activity and decreased production of pro-inflammatory cytokines, including interleukin-6 and tumor necrosis factor-α, while also supporting mitochondrial performance and cellular energy metabolism.¹¹

In a population-based study including 1,257 participants, serum Mg concentrations were categorized into quartiles. Severe Mg deficiency was identified in approximately 6% of individuals,

while insufficient Mg levels were observed in 28% of the cohort. Participants in the lowest Mg quartile exhibited a significantly higher prevalence of anti-TG positivity and an increased risk of HT compared with those in higher quartiles. Notably, serum Mg concentrations were similar between euthyroid and hypothyroid individuals. The authors suggested that increasing dietary Mg intake and supplementation in individuals with severe Mg deficiency might be beneficial for thyroid health, although causal relationships could not be established.¹² In another cross-sectional analysis involving 1,104 apparently healthy individuals, thyroid function tests, thyroid autoantibodies, and serum metal profiles were evaluated. Serum Mg concentrations were significantly lower in participants with thyroid autoantibody positivity compared with antibody-negative individuals. When serum Mg levels were stratified into quartiles, the highest Mg quartile was associated with a lower prevalence of both anti-TPO and anti-TG compared with the lower quartiles. The authors hypothesized that higher Mg status may exert a protective effect against thyroid autoimmunity.¹³ These epidemiological observations may support a potential link between magnesium status and thyroid autoimmunity. Adequate Mg availability and cellular Mg homeostasis could theoretically contribute to reducing oxidative stress burden within the thyroid gland in HT. In the present study, we did not observe significant changes in thyroid autoantibody titers after a two-month period of Mg replacement, suggesting that short-term Mg supplementation may not substantially modulate established thyroid

autoimmunity. It is conceivable that longer durations of supplementation or greater restoration of Mg status might exert different effects on thyroid autoantibody dynamics, which warrants further investigation in prospective longitudinal studies. Our findings suggest that Mg supplementation may influence biochemical thyroid parameters without significantly altering autoimmune activity over the short term.

Attinger et al. conducted an open-label randomized crossover study in 15 healthy volunteers to evaluate the effects of different Mg salts on LT4 absorption. Participants received three interventions—LT4 alone, LT4 plus Mg aspartate, and LT4 plus Mg citrate—in random sequence with washout periods between treatments, and thyroid function parameters were assessed over a 6-hour post-dose period. Mg aspartate significantly reduced LT4 absorption, with a 12% decrease in the area under the curve (AUC; $p = 0.002$), whereas Mg citrate resulted in a non-significant 7% reduction in AUC. Notably, this study was conducted in healthy individuals, and the magnitude and clinical relevance of such interactions may differ in patients with hypothyroidism receiving chronic LT4 therapy.⁸ In the present retrospective cohort, we observed a modest but statistically significant increase in serum TSH concentrations after two months of Mg replacement. Given the established interactions between LT4 and other divalent cations, such as calcium and iron, the observed TSH increase may reflect subtle changes in LT4 bioavailability or medication adherence rather than a direct endocrine effect of Mg on the hypothalamic–pituitary–thyroid axis. Due to the retrospective design, the timing of Mg and LT4 administration could not be reliably determined. Moreover, there are currently no definitive guideline recommendations regarding the optimal temporal separation between LT4 and Mg supplementation, and evidence in this area remains limited.

In another randomized, double-blind, placebo-controlled study investigated the impact of micronutrient supplementation on thyroid function among individuals with hypothyroidism. Eighty-six patients aged 20–65 years were randomly assigned to one of two parallel groups. The intervention group ($n = 43$) received zinc gluconate (30 mg daily), Mg oxide (250 mg daily), and vitamin A (25,000 IU twice weekly) for 10 weeks, whereas the placebo group ($n = 43$) received matching placebo capsules for the same duration. A significant increase in serum fT4 concentrations was observed in the intervention group, whereas no significant changes were detected in the placebo group. Based on these findings, the authors suggested that combined supplementation with zinc, vitamin A, and Mg may exert beneficial effects on thyroid function in patients with hypothyroidism and potentially in thyroid-related disorders.¹⁴ In this study, minerals were administered in combination; therefore, the independent effect of Mg cannot be isolated or specifically attributed to the observed outcomes.

Several methodological limitations merit consideration. The study was conducted at a single center and employed a retrospective design, which may restrict the external validity of the results. In addition, the retrospective nature of the analysis precluded accurate assessment of the timing between LT4 and Mg administration, thereby limiting control over potential interactions related to dosing intervals. Third, the duration of Mg replacement was relatively short, and longer-term effects on thyroid function and autoimmunity could not be assessed. Finally, Mg is predominantly an intracellular cation, and serum Mg concentrations may not fully reflect total body Mg status, which may have led to misclassification of Mg deficiency severity.

An additional point worth considering is the formulation of Mg used in this study. Mg oxide is known to have lower bioavailability compared with other oral Mg salts such as Mg citrate or Mg glycinate. Although serum Mg levels increased significantly after treatment, they reached only the lower limit of the reference range. It is therefore possible that the modest biochemical changes observed in this study may partly reflect the limited bioavailability of the Mg preparation used. Future studies evaluating Mg formulations with higher bioavailability may help clarify whether different preparations could produce more pronounced effects on thyroid-related biochemical parameters.

In conclusion, based on currently available evidence, the clinical effects of coadministration of LT4 and Mg in patients with hypothyroidism remain incompletely defined, and studies employing a design similar to the present investigation are lacking. Mg coadministration may interfere with LT4 absorption, potentially attenuating its therapeutic effect and leading to deviations from target TSH levels. This interaction may be particularly relevant in vulnerable populations, including pregnant women, older adults, patients with infertility, and those with cardiovascular comorbidities, in whom precise thyroid hormone control is critical. In such settings, temporal separation between LT4 and Mg administration and careful dose titration of LT4 should be considered. Prospective, multicenter studies with larger sample sizes and longer follow-up periods are warranted to better elucidate the clinical significance of Mg–LT4 interactions and to define evidence-based administration strategies.

Declarations

Ethics approval: All procedures involving human participants were carried out in accordance with the ethical standards of the appropriate institutional and national research committees and in compliance with the principles of the Declaration of Helsinki (1964) and its later amendments. Ethical approval was granted by the Ethics Committee of Süleyman Demirel University Faculty of Medicine (Meeting Date: February 20, 2025; Meeting No: 92; Decision No: 16).

Consent to participate: Not applicable

Consent for publication: Not applicable

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Author contribution: EU conceived and designed the study, conducted the data analysis, prepared the manuscript, and oversaw all stages of the research.

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