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Case Report

Severe Hypomagnesemia in a Patient Treated Using Carboplatin Co-Administered with Vonoprazan

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Keywords
Carboplatin · Vonoprazan · Hypomagnesemia · Magnesium oxide · Nab-paclitaxel

Abstract
Introduction: We describe a case of severe hypomagnesemia that occurred during treatment with carboplatin (CBDCA) and nanoparticle albumin-bound paclitaxel (nab-PTX) for lung adenocarcinoma when co-administered with vonoprazan. ***Case Presentation:*** A man in his 70s was diagnosed with stage IIIA lung adenocarcinoma and received CBDCA and nab-PTX as the first-line treatment. The patient had been taking omeprazole 10 mg once daily (for >3 years) for gastroesophageal reflux disease, but it was switched to lansoprazole 15 mg because of hospital's adopted medication. During the first treatment cycle, his serum creatinine levels increased from 1.0 to 1.5 mg/dL, suggesting CBDCA-associated renal impairment. Because of gastric discomfort on day 15 of the second cycle, lansoprazole was switched to vonoprazan 10 mg once daily. On day 23 of the second cycle, he developed torsades de pointes and was hospitalized; severe hypomagnesemia (0.4 mg/dL) was detected to be causing the symptoms. Discontinuation of vonoprazan and a single intravenous infusion of 60 mEq magnesium sulfate raised serum magnesium levels to 3.7 mg/dL, and the arrhythmia disappeared. Mild hypomagnesemia (1.4 mg/dL) reappeared 5 days later, and an additional intravenous infusion of 20 mEq magnesium sulfate with subsequent oral magnesium oxide (1,980 mg/day) resolved the symptoms. CBDCA was discontinued and nab-PTX monotherapy was continued. Vonoprazan was resumed owing to gastric discomfort relapse; however,

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grade ≥2 hypomagnesemia did not reappear later. ***Conclusions:*** This case highlights the risk of severe hypomagnesemia in patients with CBDCA and vonoprazan co-administration; therefore, regular monitoring of serum magnesium levels during the treatment is crucial.

Introduction

Carboplatin (CBDCA) is a widely used platinum drug for treating lung cancer, with fewer adverse effects such as nausea, vomiting, hypomagnesemia, neuropathy, and renal dysfunction compared with those of cisplatin (CDDP) [1, 2]. Hypomagnesemia occurs in 90% of patients administered CDDP, whereas the symptoms are less frequent and severe compared with those associated with CBDCA [2, 3]. Therefore, CBDCA-induced hypomagnesemia is not a widely recognized clinical problem. Vonoprazan, a long-acting antacid used for the treatment of *Helicobacter pylori* and gastroesophageal reflux disease, can also cause severe hypomagnesemia [4]. Severe hypomagnesemia induces muscle cramps, tetany, and arrhythmia, which can be life-threatening [5]. In this report, we describe a case of arrhythmia caused by severe hypomagnesemia during lung adenocarcinoma treatment using CBDCA and nanoparticle albumin-bound paclitaxel (nab-PTX) with vonoprazan co-administration and its treatment process.

Case Presentation

A man in his 70s, with a history of idiopathic interstitial pneumonia, atrial fibrillation, dyslipidemia, reflux esophagitis, and high blood pressure, was diagnosed with stage IIIA lung adenocarcinoma (right upper lobe, T1aN2M0). The patient was taking omeprazole 10 mg once a day (for >3 years), amlodipine 2.5 mg once a day, telmisartan 20 mg once a day, pemafibrate 0.1 mg twice a day, bisoprolol 1.25 mg once a day, disopyramide 100 mg once a day, and omega-3-acid ethyl esters 2 g once a day at baseline, and all symptoms were well managed by the medication. Treatment by CBDCA (area under the concentration-time curve of 6, day 1) and nab-PTX (100 mg/m^2, on days 1, 8, and 15) every 3 weeks was selected as the first-line therapy, considering the patient's history of idiopathic interstitial pneumonia. On the day before the treatment, omeprazole was substituted with lansoprazole 15 mg once daily in accordance with the hospital's adopted medication. The baseline serum laboratory data of the patient are presented in Table 1. Serum magnesium level was 1.3 mg/dL, and serum creatinine level was 1.00 mg/dL (creatinine clearance calculated by Cockcroft-Gault formula 66.1 mL/min), indicating grade 1 hypomagnesemia and mild renal dysfunction at baseline. On day 14 of the first cycle, the patient exhibited a serum creatinine elevation to 1.50 mg/dL (creatinine clearance 44.1 mL/min), which resulted in the suspension of day 15 medication. Additional adverse effects observed during the first cycle included fatigue (grade 1), arthralgia (grade 1), neutropenia (grade 1), increased aspartate aminotransferase level (grade 1), increased alanine aminotransferase level (grade 1), and anemia (grade 1). On day 28 of the first cycle, the second cycle of treatment was administered at the full dose. On day 8 of the second cycle (day 35 from treatment initiation), the patient complained of heartburn symptoms. Substitution of lansoprazole with vonoprazan 10 mg once daily alleviated the symptoms. On day 23 of the second cycle (day 50), the patient developed dizziness, tremors, and hyperventilation, with a heart rate of 120 beats per minute and oxygen saturation of

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Table 1. Laboratory data on day 1 and 51

	Day 1	Day 51
Blood chemistry		
Mg, mg/dL	1.3	0.4
Ca, mg/dL	9.8	7.1
K, mEq/L	4.0	4.2
Na, mEq/L	137	139
Cl, mEq/L	102	105
P, mg/dL	3.4	4.0
Cr, mg/dL	1.00	1.34
BUN, mg/dL	14	19
Free T3, pg/mL	—	2.04
Free T4, ng/dL	—	0.85
TSH, mIU/L	—	1.36
PTH-I, pg/mL	—	19
PRA, ng/mL/h	—	27.7
ALD, pg/mL	—	25.3
TP, g/dL	7.7	6.5
Alb, g/dL	4.6	3.9
AST, U/L	33	39
ALT, U/L	44	46
ALP, U/L	32	24
γ-GT, U/L	64	88
LD, U/L	241	291
CRP, mg/dL	0.74	0.94
Urinalysis		
S.G.	—	1.011
pH	—	5.5
Mg, mg/dL	—	<0.4
Ca, mg/dL	—	<0.3
K, mEq/L	—	28
Na, mEq/L	—	71
Cl, mEq/L	—	76
Cr, mg/dL	—	94
NAG/Cr, U/L	—	4.8
β2-MG/Cr	—	BDL
Protein	—	(−)

Cr, creatinine; BUN, blood urea nitrogen; TSH, thyroid-stimulating hormone; PTH-I, parathyroid hormone intact; PRA, plasma renin activity; ALD, aldosterone; TP, total protein; Alb, albumin; AST, aspartate aminotransferase; ALT, alanine aminotransferase; ALP, alkaline phosphatase; γ-GT, γ-glutamyl transferase; LD, lactic acid dehydrogenase; CRP, C-reactive protein; S.G., specific gravity; pH, potential hydrogen; NAG, N-acetyl-β-D-glucosaminidase; β2-MG, β2 microglobulin; BDL, below detection limit.

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88–90%, which were not observed in the first cycle. The patient was immediately treated with 5 mg of intravenous verapamil and oxygen therapy; however, the symptoms persisted, necessitating hospital admission. Blood tests, echocardiography, and computed tomography revealed no signs of heart failure, ischemic stroke, or pulmonary thromboembolism. The next day (day 51), further laboratory examinations revealed serum magnesium levels to be 0.4 mg/dL and urinary magnesium excretion below the detection limit (Table 1). We hypothesized that the symptoms were caused by torsades de pointes, induced by severe hypomagnesemia. The patient had no history of malnutrition, chronic alcoholism, chronic diarrhea, or inflammatory bowel disease. Primary hyperparathyroidism and aldosteronism were ruled out after a later examination, as the patient's intact parathyroid hormone levels was 19 pg/mL, inorganic phosphorus levels was 4.0 mg/dL, and aldosterone-to-renin ratio was 1, all of which were within the normal range. On the same day, vonoprazan was discontinued as a suspected drug causing the symptoms, and 60 mEq of magnesium sulfate was administered once intravenously, resulting in an improvement in serum magnesium levels to 3.7 mg/dL on the following day. However, on day 56, serum magnesium levels decreased again to 1.4 mg/dL, with a fractional excretion of 6.5% magnesium. On the same day, intravenous magnesium sulfate (20 mEq) was administered once and oral magnesium oxide (MgO) 330 mg twice daily was initiated the next day. On day 57, famotidine 20 mg was administered orally twice daily because of worsened heartburn. On the same day, the serum magnesium levels were elevated to 2.3 mg/dL, and the patient was discharged on the next day. The progression of serum magnesium levels is illustrated in Figure 1. On day 63, the serum magnesium level decreased again to 1.2 mg/dL, and the MgO dose was increased to 660 mg thrice a day. On day 70, the serum magnesium levels were elevated to 1.5 mg/dL and nab-PTX monotherapy (100 mg/m^2, on days 1, 8, and 15, every 3 weeks) was resumed. CBDCA was discontinued because we considered it to be the causative agent of severe hypomagnesemia. On day 8 of the first cycle of nab-PTX (day 77), worsening heartburn led to a switch from famotidine to vonoprazan 10 mg once daily, as needed. However, because his gastrointestinal symptoms did not improve sufficiently, the dose of vonoprazan was adjusted to 10 mg once daily on day 98, leading to symptom improvement without recurrence of severe hypomagnesemia. On day 117, grade 3 diarrhea manifested, leading to the discontinuation of MgO, which subsequently reduced the serum magnesium levels to 1.3 mg/dL on day 1 of the third cycle (day 127). Consequently, MgO was resumed on that day at a dose of either 330 or 660 mg/day, depending on stool characteristics. However, because there was no improvement in the serum magnesium levels, vonoprazan was replaced with famotidine 20 mg twice daily on day 141. Subsequently, the medication was changed back to vonoprazan 10 mg once daily on day 168 owing to worsening heartburn. Thereafter, the patient regularly continued taking both vonoprazan and MgO without grade ≥2 hypomagnesemia. On day 181, interstitial pneumonia worsened, and nab-PTX monotherapy was discontinued (final nab-PTX administration was day 154). Computed tomography on day 280 revealed progressive disease. As described previously, serum creatinine levels increased from 1.00 mg/dL to 1.50 mg/dL on day 14 but improved 7 days later; the levels fluctuated up and down within 1.14–1.47 mg/dL during the treatment. The adjusted calcium levels decreased to 7.1 mg/dL on day 51 but normalized on day 56 without supplementation. Serum potassium levels remained at grade 0–1 throughout the treatment.

Discussion

Magnesium is the fourth most abundant cation in the human body and plays a crucial role in various physiological functions [5]. Magnesium is primarily stored in the bone (60%) and skeletal muscle (20%), with less than 1% stored in extracellular fluid [5].

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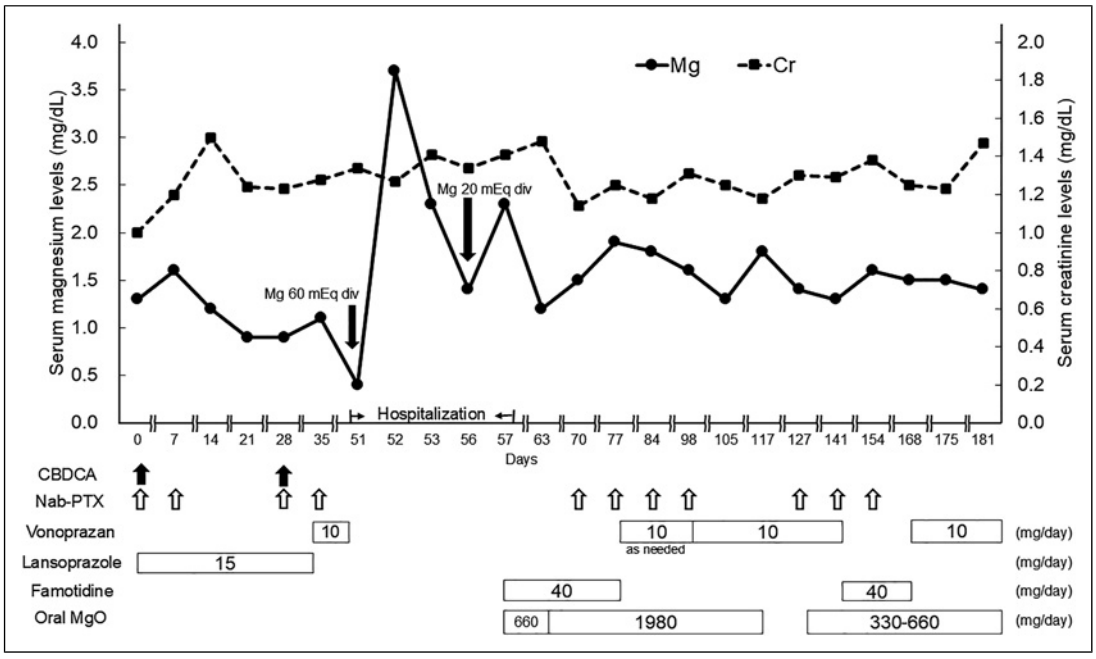

Fig. 1. Variation of serum magnesium and creatinine levels with the administered medication. On admission, the omeprazole was switched to lansoprazole. On day 35, lansoprazole was changed to vonoprazan owing to gastric discomfort. On day 51, after starting combined therapy with CBDCA and nab-PTX, serum magnesium levels decreased to 0.4 mg/dL. Discontinuation of vonoprazan and 60 mEq of intravenous magnesium sulfate increased the serum magnesium levels (day 52), which decreased again on day 56, requiring an additional 20 mEq of intravenous magnesium sulfate. Oral MgO and famotidine treatments were initiated the following day. CBDCA was discontinued and nab-PTX monotherapy was continued between days 70 and 154. Vonoprazan was reintroduced on day 77 owing to gastrointestinal symptoms, without the recurrence of severe hypomagnesemia. Div, drip intravenous injection; CBDCA, carboplatin; nab-PTX, nanoparticle albumin-bound paclitaxel; MgO, magnesium oxide.

Therefore, serum magnesium levels may not accurately reflect total body magnesium stores [6]. Symptoms of hypomagnesemia include muscle cramps, weakness, tremors, tetany, dizziness, ataxia, depression, psychosis, and cardiovascular symptoms, although most cases are asymptomatic [5]. Hypomagnesemia is caused by alcohol dependence, kidney failure, and adverse effects of platinum agents, proton pump inhibitors (PPIs), monoclonal antibody against the epithelial growth factor receptor, and aminoglycoside antibiotics [2, 5, 7].

Although hypomagnesemia is common with CDDP and affects approximately 90% of patients [2], previous reports have shown a lower incidence with CBDCA [3]. In contrast, recent reports suggest that the incidence of hypomagnesemia with CBDCA is approximately 70–80%, which is underestimated, possibly because of inadequate monitoring [8, 9]. CBDCA-induced hypomagnesemia reportedly occurs around the third cycle of CBDCA treatment, as was observed in this case [9]. Gaughran et al. [8] reported that most cases of CBDCA-induced hypomagnesemia are grade 1 or 2, with grade 3 in 8% and grade 4 in 1.9% of cases. Another report described most of the symptoms being grade 1 [9]. These results suggest that the majority of CBDCA-induced hypomagnesemia cases are mild, and severe symptoms are rare. We assessed the causal relationship between severe hypomagnesemia and CBDCA in this case using the Naranjo Adverse Drug Reaction Probability Scale, with a score of 4, indicating "Possible" (online suppl. Table 1; for all online suppl. material, see https://doi.org/10.1159/000542906).

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The kidneys and intestines play significant roles in magnesium regulation; thus, increased renal losses and/or decreased intestinal absorption of magnesium can lead to hypomagnesemia [5]. Approximately 70–80% of the magnesium in the blood is filtered by the renal glomeruli, and more than 96% is reabsorbed by the renal tubules [6]. CDDP-induced hypomagnesemia is primarily attributed to damage to the renal tubular cells or alterations in calcium/magnesium sensing receptors [2]. CDDP downregulates the transient receptor potential subfamily melastatin (TRPM) 6 and epidermal growth factor (EGF) in the distal convoluted tubule, thereby inhibiting magnesium reabsorption [10]. The mechanism of CBDCA-induced hypomagnesemia remains unclear but is hypothesized to be similar to that of CDDP. In this patient, the fractional excretion of magnesium 5 days after a single dose of 60 mEq of magnesium sulfate was 6.5%, suggesting an increased renal magnesium excretion. Moreover, the patient had moderate renal impairment after treatment initiation, and serum magnesium depletion was also confirmed concurrently. Therefore, we speculate that dysregulation of TRPM6 and EGF and renal dysfunction by CBDCA contributed to the initial decrease in magnesium levels before the initiation of vonoprazan.

Long-term use of PPIs for >6 months is also associated with an increased risk of hypomagnesemia, with an incidence rate of 19% [11]. This patient had grade 1 hypomagnesemia at baseline and had been taking omeprazole for gastroesophageal reflux disease for over 3 years, which may have caused the baseline symptoms. In addition, the patient developed severe hypomagnesemia 2 weeks after starting vonoprazan in combination with the second cycle of CBDCA and nab-PTX therapy. A previous report has suggested that vonoprazan can also induce hypomagnesemia [4]. Aiba et al. [4] reported a case in which severe hypomagnesemia occurred 3 weeks after switching from lansoprazole to vonoprazan in a patient who underwent ileocecal resection, suggesting that a change in medication may induce hypomagnesemia. In this case, after the onset of severe hypomagnesemia, CBDCA was discontinued with the continuation of nab-PTX monotherapy. Reintroduction of vonoprazan during nab-PTX monotherapy did not worsen hypomagnesemia beyond grade 1, which is a baseline symptom. Hypomagnesemia caused by a PPI typically resolves rapidly within days of its discontinuation, although it may recur with its resumption [11]. Therefore, it is unlikely that vonoprazan alone induced severe hypomagnesemia. Considering the variation in serum magnesium levels, we believe that the severe hypomagnesemia in this case was induced by the combination of vonoprazan and CBDCA. However, urinary magnesium excretion was lower on day 51. We hypothesized that hypomagnesemia rapidly occurred due to a combination of renal tubular magnesium reabsorption inhibition by CBDCA and intestinal absorption by vonoprazan on day 51. As a result, the patient may have been unable to sufficiently compensate for extracellular magnesium through intracellular stores or absorption processes, as serum magnesium account for only 1% of the total body magnesium levels [5], resulting in decreased urinary magnesium excretion. To the best of our knowledge, this is the first report of severe hypomagnesemia caused by a combination of medications.

Magnesium is actively absorbed by intestinal cells via the intestinal mucosal transporters TRPM6 and 7; elevation of intestinal pH by PPIs reduces TRPM6/7 activity, thus decreasing magnesium absorption [11]. The administration of high-dose PPIs has been reported to increase the risk of severe hypomagnesemia [12]. Vonoprazan is a drug that exerts a more potent acid secretion inhibitory effect in a shorter period than PPIs [13], and it is possible that the further increase in pH by the change in medication might have contributed to severe hypomagnesemia through the decreased activity of TRPM6/7. Therefore, we consider that severe hypomagnesemia in this case was induced by the combined effects of the inhibition of intestinal magnesium absorption by vonoprazan and increased magnesium excretion by

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CBDCA. Consequently, we recommend that serum magnesium levels be routinely monitored in patients treated with CBDCA, as well as CDDP, and further attention should be paid when co-administering PPIs or vonoprazan.

Treatment for hypomagnesemia includes administration of intravenous, oral, or continuous subcutaneous magnesium supplementation; inhibitors of the epithelial sodium channel in the distal nephron; or inhibitors of sodium-glucose transporter type 2 [6]. Intravenous magnesium is usually administered to treat acute and/or severe hypomagnesemia, although its effect lasts for approximately 48 h with a single dose [7]. The patient responded immediately to a single intravenous magnesium sulfate dose of 60 mEq the next day. However, serum magnesium levels dropped again after 5 days, leading to the initiation of oral MgO to prevent symptom recurrence. Oral MgO is frequently administered as the first-line treatment for hypomagnesemia because of its cost-effectiveness [6]. Previous reports have also suggested the prophylactic efficacy of oral MgO for CDDP- or panitumumab-induced hypomagnesemia [14, 15], although another report suggested that oral MgO supplementation does not improve grade 3/4 hypomagnesemia caused by cetuximab [7]. Additionally, hypomagnesemia induced by CBDCA is generally reversible but may take an average of 30 weeks to recover [8]. In this case, the serum magnesium level increased following initiation of oral MgO and was in the grade 1 range even after vonoprazan administration. Although CBDCA and vonoprazan were not co-administered again, we believe that oral MgO may have been effective. Consequently, intravenous magnesium supplementation for severe hypomagnesemia treatment and oral MgO for the prevention of serum magnesium decrease after the elevation may be an effective treatment strategy.

Conclusion

We report a patient who developed severe hypomagnesemia after switching from PPIs to vonoprazan during CBDCA and nab-PTX treatment for non-small cell lung cancer. Regular monitoring of serum magnesium levels is important in patients receiving CBDCA therapy, particularly in combination with PPIs or vonoprazan. Further studies are required to elucidate the clinical conditions and detailed mechanisms for better symptom management.

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Statement of Ethics

This case is reported in compliance with the Declaration of Helsinki. This case report has been granted an exemption from requiring ethics approval at the Hokkaido University Hospital. Written informed consent was obtained from the patient for the publication of the details of the medical case and any accompanying images. The CARE Checklist has been completed by the authors for this case report, attached as online supplementary material.

Conflict of Interest Statement

The authors have no conflicts of interest to declare.

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Author Contributions

Osamu Taniguchi and Yoshitaka Saito contributed to the design of the report, data collected, and drafting of the manuscript. Yuka Yamaguchi, Midori Sakai, Yasuyuki Ikezawa, Jun Sakakibara-Konishi, Mina Eguchi, Yoh Takekuma, and Mitsuru Sugawara revised the manuscript accordingly and approved the final version of the manuscript.

Data Availability Statement

All the data generated during this study are included in this article. Further inquiries can be directed to the corresponding author.

References

1 Ardizzoni A, Boni L, Tiseo M, Fossella FV, Schiller JH, Paesmans M, et al. Cisplatin-versus carboplatin-based chemotherapy in first-line treatment of advanced non-small-cell lung cancer: an individual patient data meta-analysis. J Natl Cancer Inst. 2007;99(11):847–57. https://doi.org/10.1093/jnci/djk196
2 Lajer H, Daugaard G. Cisplatin and hypomagnesemia. Cancer Treat Rev. 1999;25(1):47–58. https://doi.org/10.1053/ctrv.1999.0097
3 Foster BJ, Clagett-Carr K, Leyland-Jones B, Hoth D. Results of NCI-sponsored phase I trials with carboplatin. Cancer Treat Rev. 1985;12(Suppl A):43–9. https://doi.org/10.1016/0305-7372(85)90017-9
4 Aiba M, Tsutsumi Y, Nagai J, Tateno T, Ito S. Convulsive seizure due to hypomagnesemia caused by short-term vonoprazan intake. Intern Med. 2022;61(2):237–40. https://doi.org/10.2169/internalmedicine.7758-21
5 Swaminathan R. Magnesium metabolism and its disorders. Clin Biochem Rev. 2003;24(2):47–66.
6 Ray E, Mohan K, Ahmad S, Wolf MTF. Physiology of a forgotten electrolyte-magnesium disorders. Adv Kidney Dis Health. 2023;30(2):148–63. https://doi.org/10.1053/j.akdh.2022.12.001
7 Fakih MG, Wilding G, Lombardo J. Cetuximab-induced hypomagnesemia in patients with colorectal cancer. Clin Colorectal Cancer. 2006;6(2):152–6. https://doi.org/10.3816/CCC.2006.n.033
8 Gaughran G, Qayyum K, Smyth L, Davis A. Carboplatin and hypomagnesemia: is it really a problem? Asia Pac J Clin Oncol. 2021;17(6):478–85. https://doi.org/10.1111/ajco.13481
9 Liu W, Qdaisat A, Soliman PT, Ramondetta L, Lopez G, Narayanan S, et al. Hypomagnesemia and survival in patients with ovarian cancer who received chemotherapy with carboplatin. Oncologist. 2019;24(6):e312–7. https://doi.org/10.1634/theoncologist.2018-0465
10 Ledeganck KJ, Boulet GA, Bogers JJ, Verpooten GA, De Winter BY. The TRPM6/EGF pathway is downregulated in a rat model of cisplatin nephrotoxicity. PLoS One. 2013;8(2):e57016. https://doi.org/10.1371/journal.pone.0057016
11 Gommers LMM, Hoenderop JGJ, de Baaij JHF. Mechanisms of proton pump inhibitor-induced hypomagnesemia. Acta Physiol (Oxf). 2022;235(4):e13846. https://doi.org/10.1111/apha.13846
12 Seah S, Tan YK, Teh K, Loh WJ, Tan PT, Goh LC, et al. Proton-pump inhibitor use amongst patients with severe hypomagnesemia. Front Pharmacol. 2023;14:1092476. https://doi.org/10.3389/fphar.2023.1092476
13 Sakurai Y, Mori Y, Okamoto H, Nishimura A, Komura E, Araki T, et al. Acid-inhibitory effects of vonoprazan 20 mg compared with esomeprazole 20 mg or rabeprazole 10 mg in healthy adult male subjects: a randomised open-label cross-over study. Aliment Pharmacol Ther. 2015;42(6):719–30. https://doi.org/10.1111/apt.13325
14 Zarif Yeganeh M, Vakili M, Shahriari-Ahmadi A, Nojomi M. Effect of oral magnesium oxide supplementation on cisplatin-induced hypomagnesemia in cancer patients: a randomized controlled trial. Iran J Public Health. 2016;45(1):54–62.
15 Sato J, Ishikawa H, Tanaka R, Sino M. Influence of oral magnesium-containing supplement and antacid administration on hypomagnesemia induced by panitumumab. Cancer Chemother Pharmacol. 2019;83(4):673–9. https://doi.org/10.1007/s00280-019-03772-y