fortune Journals

Case Report

ARCHIVES OF CLINICAL AND MEDICAL CASE REPORTS

ISSN: 2575-9655

Combined Intravenous Selenium, Zinc, and Magnesium Therapy for Refractory Chemotherapy-Induced Peripheral Neuropathy: A Case Report

Li-Kai Wang, MD, MSc[1,2], Shu-Wei Liao, MD[1,3], Wei-Cheng Liu, MD[1,4], Yu-Li Pang, MD[1,5,6], Yao-Tsung Lin, MD, MSc[7], Jen-Yin Chen, MD, PhD[1,8*]

Abstract

Chemotherapy-induced peripheral neuropathy (CIPN) is a dose-limiting toxicity with limited treatment options. We report a 68-year-old female with Anaplastic Lymphoma Kinase (ALK)-mutant non-small cell lung cancer who developed severe, refractory CIPN (numeric rating scale [NRS] 9/10) and sensory ataxia following paclitaxel and carboplatin therapy. Conventional medications, including duloxetine, were ineffective and poorly tolerated. Nutrient analysis revealed low-normal selenium and magnesium alongside zinc deficiency. An intravenous nutrient cocktail of sodium selenite, zinc sulfate, and magnesium sulfate was initiated. Over five months, the patient achieved complete pain relief (NRS 0/10) and near-complete restoration of hand function and proprioception. Mechanistically, selenium stabilizes mitochondria and reduces oxidative stress; zinc inhibits transient receptor potential vanilloid 1 ($TRPV_1$)-mediated nociception; and magnesium acts as an N-methyl-D-aspartate (NMDA) receptor antagonist. This case suggests that intravenous nutrient cocktail therapy is a safe and effective adjunct for managing severe, refractory CIPN, warranting further clinical validation.

Keywords: Chemotherapy; Peripheral neuropathy; Selenium; Zinc; Magnesium

Introduction

Chemotherapy-induced peripheral neuropathy (CIPN) is a common dose-limiting side effect of cancer treatment, resulting from damage to peripheral nerves. This condition manifests as numbness, tingling, and neuropathic pain in the distal extremities, which profoundly impacts the quality of life of cancer survivors. Duloxetine is the only agent currently recommended by the American Society of Clinical Oncology (ASCO) for the treatment of painful CIPN [1]; however, its clinical utility remains limited by modest efficacy and poor patient tolerability. Consequently, the management of established CIPN continues to be a significant therapeutic challenge.

Recent clinical evidence has highlighted the potential of high-dose intravenous selenium (2,000 µg), administered prior to each chemotherapy cycle with platinum-based regimens, in reducing the incidence and severity of CIPN in cancer patients [2,3]. Furthermore, zinc and magnesium sulfate have been investigated as promising adjunctive therapies for neuropathic pain [4-6]. In this report, we describe a case of severe, refractory CIPN following taxane-platinum therapy in a patient with non-small cell lung cancer (NSCLC). The introduction of a multimodal intravenous nutrient cocktail—consisting of selenium, zinc, and magnesium—was associated

Affiliation:

[1]Department of Anesthesiology, Chi Mei Medical Center, Tainan, Taiwan

[2]School of Medicine, Chung Shan Medical University, Taichung, Taiwan

[3]Center of General Education, Chia Nan University of Pharmacy and Science, Tainan, Taiwan

[4]Department of Occupational Therapy, Shu-Zen Junior College of Medicine and Management, Kaohsiung, Taiwan

[5]Department of Nursing, Shu-Zen Junior College of Medicine and Management, Kaohsiung, Taiwan

[6]Institute of Clinical Medicine, College of Medicine, National Cheng Kung University, Tainan, Taiwan

[7]Department of Anesthesiology, Chi Mei Medical Center, Chiali, Tainan, Taiwan

[8]School of Medicine, College of Medicine, National Sun Yat-sen University, Kaohsiung, Taiwan

***Corresponding Author**

Jen-Yin Chen, MD, PhD, Department of Anesthesiology, Chi Mei Medical Center, No. 901, Zhonghua Road, Yongkang District, Tainan 71004, Taiwan, Tel: +886-6-2812811;
Fax: +886-6-2830122

Citation: Li-Kai Wang, Shu-Wei Liao, Wei-Cheng Liu, Yu-Li Pang, Yao-Tsung Lin, Jen-Yin Chen. Combined Intravenous Selenium, Zinc, and Magnesium Therapy for Refractory Chemotherapy-Induced Peripheral Neuropathy: A Case Report. Archives of Clinical and Medical Case Reports. 10 (2026): 96-99.

Received: March 29, 2026
Accepted: April 02, 2026
Published: May 01, 2026

fortune Journals
Li-Kai Wang MD, et al., Arch Clin Med Case Rep 2026
DOI:10.26502/acmcr.96550757

with substantial symptomatic relief and a near-complete restoration of physical function.

Case Presentation

A 68-year-old female (height 154 cm, weight 56 kg, BMI 23.6) with Anaplastic Lymphoma Kinase (ALK)-mutant NSCLC presented with progressive, debilitating CIPN following a complex oncological history. Her previous treatments included surgical resection followed by a four-year regimen involving nivolumab, paclitaxel, carboplatin, and bevacizumab, supplemented by concurrent chemoradiotherapy, pemetrexed, and crizotinib for her ALK-positive disease. During treatment, she developed severe neurotoxicity, manifesting as persistent erythema of the extremities, distal numbness, dysesthesia, hyperesthesia, and excruciating tingling and needling pain. Her pain was rated 9/10 on the numeric rating scale (NRS 0-10) and led to profound sleep disturbances.

The neurological impairment advanced to sensory ataxia, resulting in frequent dropping of objects and a complete inability to perform fine motor tasks such as using chopsticks or spoons, despite having discontinued paclitaxel and carboplatin several months earlier. Her symptoms remained refractory, with pain scores persisting at 7/10 for nearly eight months despite therapeutic trials of duloxetine, pregabalin, and morphine. These conventional agents were poorly tolerated due to significant adverse effects including dry mouth, dizziness, fatigue, and anorexia. She experienced three falls attributed to impaired proprioception and dizziness, necessitating supportive interventions. The patient subsequently presented to our pain clinic for further management.

Her clinical picture was consistent with cumulative neurotoxicity from taxane-platinum therapy, manifesting as a "coasting phenomenon" where neuropathic features persist or worsen after treatment cessation. In contrast, nivolumab, bevacizumab, pemetrexed, and crizotinib are not typically associated with CIPN. She had received nutrient supplementation, including Kentamin (a vitamin B complex preparation containing thiamine [B1] 50 mg, pyridoxine [B6] 50 mg, and cyanocobalamin [B12] 500 µg) at one capsule daily, together with vitamin C 1000 mg and vitamin D3 4000 IU daily for one year. Nutrient analysis demonstrated serum selenium (113 µg/L) and magnesium (1.9 mg/dL) concentrations at the lower limits of the reference range, alongside a definitive zinc deficiency (68 µg/dL). (Reference range: selenium 70-190 µg/L; magnesium 1.9-2.5 mg/dL; zinc 70-120 µg/dL.) An experimental intravenous nutrient cocktail was subsequently initiated, comprising 4 mL of sodium selenite (containing 50 µg/mL elemental selenium), 10 mL of zinc sulfate (containing 5 mg/mL elemental zinc), and 10 mL of magnesium sulfate (containing 98.6 mg/mL elemental magnesium), diluted in 250 mL of normal saline, administered twice weekly as a 2-hour infusion. After four weeks of treatment, pain intensity decreased from NRS 9/10 to 5/10. The area of numbness reduced from the entire palm to approximately half of the distal palm. The regimen was subsequently adjusted to once-weekly infusions of 2 mL of sodium selenite, 5 mL of zinc sulfate, and 10 mL of magnesium sulfate in 100 mL of normal saline, combined with oral supplementation of selenium (200 µg/day) and zinc (10 mg/day) for two months. Following this adjustment, pain intensity further decreased to 3/10, and numbness was localized to the distal metacarpal region. The intravenous infusion, combined with oral supplementation of selenium (100 µg/day) and zinc (10 mg/day), was subsequently reduced to once every two weeks for two months. Over five months, her pain gradually decreased from NRS 9/10 to 0/10, accompanied by a near-complete restoration of hand function and a significant reduction in fall risk. Serum nutrient levels normalized in parallel with clinical recovery: selenium rose to 218 µg/L, zinc to 98 µg/dL, and magnesium to 2.1 mg/dL.

Discussion

This case illustrates that targeted, multimodal intravenous nutrient therapy may effectively reverse established, refractory CIPN, offering a robust therapeutic alternative when conventional pharmacological interventions fail.

The diagnosis of CIPN is established from clinical history and symptom characterization [1]. In this case, the patient's presentation was consistent with the known cumulative neurotoxicity of paclitaxel and carboplatin, further complicated by the coasting phenomenon. Paclitaxel, a taxane, stabilizes microtubules and disrupts axonal transport, resulting in mitochondrial dysfunction, glial activation, and ion channel changes that drive neuroinflammation and neuronal hyperexcitability [7-9]. Meanwhile, platinum agents such as oxaliplatin cause CIPN via DNA adducts formation, mitochondrial dysfunction, oxidative stress, and ion channel dysregulation, resulting in axonal degeneration and apoptosis of dorsal root ganglion neurons [9,10]. These effects of taxane-platinum regimens induce oxidative stress, energy depletion, and sensory dysfunction, leading to CIPN via distinct pathways [9].

Standard management strategies for CIPN remain inadequate. While duloxetine is the only agent recommended by ASCO [1], its benefit is limited in certain cases. Pregabalin was effective in relieving neuropathic pain in some studies [11,12], whereas morphine is not recommended [1]. In our case, the therapeutic effects of duloxetine, pregabalin, and morphine were limited, while their adverse effects proved intolerable. Consequently, non-pharmacological alternative approaches such as nutritional supplements are warranted [6]. Our findings suggest that correcting micronutrient

fortune Journals

Li-Kai Wang MD, et al., Arch Clin Med Case Rep 2026
DOI:10.26502/acmcr.96550757

deficiencies through intravenous administration can bypass gastrointestinal absorption barriers frequently seen in cancer patients, providing rapid therapeutic concentrations of neuroprotective elements. Clinical trials demonstrated efficacy and safety of high-dose intravenous selenium (2,000 µg/day) in CIPN [2,3]. However, transient adverse effects such as nausea and gastrointestinal upset were reported in some patients [13]. Current evidence indicates that multimodal nutritional strategies are more promising than single-agent monotherapy for preventing or mitigating CIPN [14]. To our knowledge, this is the first reported case in which intravenous nutrient cocktail therapy—including selenium, zinc, and magnesium—effectively managed CIPN.

In our case, nutrient analysis revealed low-normal selenium and magnesium with concurrent zinc deficiency. Cancer survivors with CIPN show lower intake of selenium [15] and magnesium [16], while peripheral neuropathy is linked to acute zinc deficiency in guinea pigs [17]. The synergistic effect of selenium, zinc, and magnesium addresses multiple pathogenic pathways. Mechanistically, selenium mitigates CIPN by stabilizing mitochondrial function, reducing apoptosis, and enhancing antioxidant defenses. It downregulates nuclear factor kappa B (NF-κB) and pro-inflammatory cytokines, limits calcium influx mediated by $TRPV_1$ (transient receptor potential vanilloid 1), and modulates ion channel activity, thereby decreasing neuroinflammation, neuronal hyperexcitability, and neuropathic pain [14,18]. Zinc inhibits N-methyl-D-aspartate (NMDA) receptors in the spinal dorsal horn and $TRPV_1$ channels in dorsal root ganglion neurons [4,19], both of which are key mediators of nociceptive signaling and neuropathic pain. Through zinc transporter-mediated inhibition of $TRPV_1$, zinc reduces calcium influx and paclitaxel-induced hypersensitivity [19]. In addition, zinc attenuates pro-inflammatory cytokine release, stabilizes neuronal membranes, and supports antioxidant defenses by serving as a cofactor for antioxidant enzymes, including superoxide dismutase, thereby limiting peripheral sensitization and oxidative stress-related axonal injury [14]. Magnesium mitigates CIPN by acting as a natural calcium antagonist, limiting NMDA receptor and voltage-gated calcium channel influx [20], thereby preserving mitochondrial integrity and preventing neuronal apoptosis. It supports ATP-dependent metabolism, suppresses NF-κB–mediated inflammation, and reduces oxidative stress, ultimately stabilizing sensory neuron excitability and alleviating neuropathic pain [14]. Our outcomes with a multi-nutrient approach are promising for mitigating CIPN, given its complex pathogenesis and the complementary mechanisms of action of these nutrients.

In conclusion, this case demonstrates that a multimodal intravenous nutrient cocktail consisting of selenium, zinc, and magnesium can provide rapid and durable symptomatic relief in patients with severe, refractory CIPN. By simultaneously targeting oxidative stress, mitochondrial stability, and NMDA-mediated excitotoxicity, this regimen successfully reversed sensory ataxia and excruciating pain where conventional pharmacotherapy failed. Given the excellent safety profile and measurable functional recovery observed, further clinical trials are warranted to validate this nutritional intervention as a viable adjunct in oncological supportive care, particularly for patients who are intolerant to guideline-recommended medications [2,3,14].

Conflicts of Interest

The authors declare no conflicts of interest.

References

1. Charles L. Loprinzi, Christina Lacchetti, Jonathan Bleeker, et al. Prevention and Management of Chemotherapy-Induced Peripheral Neuropathy in Survivors of Adult Cancers: ASCO Guideline Update. Journal of Clinical Oncology 38 (2020): 3325-3348.

2. Ga Won Yim, Seungmee Lee, Maria Lee, et al. Efficacy and Safety of Intravenous Administration of High-Dose Selenium for Preventing Chemotherapy-Induced Peripheral Neuropathy in Platinum-Sensitive Recurrent Ovarian, Fallopian or Primary Peritoneal Cancer: Study Protocol for a Phase III, Double-Blind, Randomized Study. International Journal of Gynecological Cancer 35 (2025): 100113.

3. Wedyan Alhazmi, Kyo Young Song. Efficacy and Safety of Intravenous Administration of High-Dose Selenium for Preventing Chemotherapy-Induced Peripheral Neuropathy in Gastric Cancer Patients Receiving Adjuvant Oxaliplatin and Capecitabine After Gastrectomy: A Retrospective Pilot Study. Korean Journal of Clinical Oncology 21 (2025): 81-89.

4. Chihiro Nozaki, Angela Maria Vergnano, Dominique Filliol, et al. Zinc Alleviates Pain Through High-Affinity Binding to the NMDA Receptor NR2A Subunit. Nature Neuroscience 14 (2011): 1017-1022.

5. Yao-Tsung Lin, Kuo-Mao Lan, Li-Kai Wang, et al. Treatment of Postherpetic Neuralgia with Intravenous Administration of Zinc Sulfate: A Case Report. A&A Practice 11 (2018): 8-10.

6. Khaled M. Abdelrahman, Kevin V. Hackshaw. Nutritional Supplements for the Treatment of Neuropathic Pain. Biomedicines 9 (2021): 674.

7. Anukriti Sharma, Ken B. Johnson, Alper Sen, et al. Longitudinal Multi-Omics Analyses of Chemotherapy-Induced Peripheral Neuropathy in Response to Taxanes. BMC Cancer 25 (2025): 1591.

Li-Kai Wang MD, et al., Arch Clin Med Case Rep 2026
DOI:10.26502/acmcr.96550757

8. Renata Zajączkowska, Magdalena Kocot-Kępska, Wojciech Leppert, et al. Mechanisms of Chemotherapy-Induced Peripheral Neuropathy. International Journal of Molecular Sciences 20 (2019): 1451.

9. Xinyu Chen, Yumeng Gan, Nan Pan Bennett Au, et al. Current Understanding of the Molecular Mechanisms of Chemotherapy-Induced Peripheral Neuropathy. Frontiers in Molecular Neuroscience 17 (2024): 1345811.

10. Lumei Kang, Yuyang Tian, Shilin Xu, et al. Oxaliplatin-Induced Peripheral Neuropathy: Clinical Features, Mechanisms, Prevention and Treatment. Journal of Neurology 268 (2021): 3269-3282.

11. Şule Karabulut Gül, Hüseyin Tepetam, et al. Duloxetine and Pregabalin in Neuropathic Pain of Lung Cancer Patients. Brain and Behavior 10 (2020): e01527.

12. Ebrahim Salehifar, Ghasem Janbabaei, Narjes Hendouei, et al. Comparison of the Efficacy and Safety of Pregabalin and Duloxetine in Taxane-Induced Sensory Neuropathy: A Randomized Controlled Trial. Clinical Drug Investigation 40 (2020): 249-257.

13. Matthias W. A. Angstwurm, Juergen Schottdorf, Jochen Schopohl, et al. Selenium Replacement in Patients with Severe Systemic Inflammatory Response Syndrome Improves Clinical Outcome. Critical Care Medicine 27 (1999): 1807-1813.

14. Lin Zhou, Hui Yang, Jing Wang, et al. The Therapeutic Potential of Antioxidants in Chemotherapy-Induced Peripheral Neuropathy: Evidence from Preclinical and Clinical Studies. Neurotherapeutics 20 (2023): 339-358.

15. Karly Miller, Suzanna Zick, Robert Ploutz-Snyder, et al. Food Groups and Micronutrients Associated with Chemotherapy-Induced Peripheral Neuropathy in Survivors of Cancer Post-Neurotoxic Treatment. Supportive Care in Cancer 33 (2025): 806.

16. Evertine Wesselink, Renate M. Winkels, Harm van Baar, et al. Dietary Intake of Magnesium or Calcium and Chemotherapy-Induced Peripheral Neuropathy in Colorectal Cancer Patients. Nutrients 10 (2018): 398.

17. Lizabeth A. Terril-Robb, Donna J. Clemons, Cynthia Besch-Williford, et al. Morphophysiologic Characterization of Peripheral Neuropathy in Zinc-Deficient Guinea Pigs. Proceedings of the Society for Experimental Biology and Medicine 213 (1996): 50-58.

18. Bilal Çiğ. Selenium Reduces Oxaliplatin Induced Neuropathic Pain: Focus on $TRPV_1$. Frontiers in Pharmacology 16 (2025): 1549190.

19. Jialie Luo, Alexis Bavencoffe, Pu Yang, et al. Zinc Inhibits $TRPV_1$ to Alleviate Chemotherapy-Induced Neuropathic Pain. The Journal of Neuroscience 38 (2018): 474-483.

20. Kamila Kulik, Barbara Żyżyńska-Granica, Agnieszka Kowalczyk, et al. Magnesium and Morphine in the Treatment of Chronic Neuropathic Pain–A Biomedical Mechanism of Action. International Journal of Molecular Sciences 22 (2021): 13599.

This article is an open access article distributed under the terms and conditions of the Creative Commons Attribution (CC-BY) license 4.0

Citation: Li-Kai Wang, Shu-Wei Liao, Wei-Cheng Liu, Yu-Li Pang, Yao-Tsung Lin, Jen-Yin Chen. Combined Intravenous Selenium, Zinc, and Magnesium Therapy for Refractory Chemotherapy-Induced Peripheral Neuropathy: A Case Report. Archives of Clinical and Medical Case Reports. 10 (2026): 96-99.