Letter to the Editor | Symptom Management in Palliative Medicine and Palliative Care

Assessing lafutidine's potential to protect lung cancer patients from chemotherapy-induced neuropathy

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Upon examination of the article titled "A randomized trial to evaluate the preventive effect of lafutidine on chemotherapy-induced peripheral neuropathy in patients treated with carboplatin and paclitaxel for lung cancer" written by Cho *et al.* (1), we applaud the authors of the study for evaluating the chemoprotective efficacy of lafutidine on chemotherapy-induced peripheral neuropathy (CIPN) in lung cancer patients treated with carboplatin and paclitaxel. It offers details relevant to this issue and suggests further study in this crucial field since the outcomes were not statistically substantial.

Notably, the study would benefit from evaluating the divided doses of paclitaxel since the doses of this medicine are an accepted clinical approach currently. However, in clinical practice, paclitaxel treatment is usually divided into multiple cycles, and this frequent low-level exposure could significantly affect the occurrence of peripheral neuropathy. In a recent article, the intravenous (IV) paclitaxel was given to all patients in this dose of 200 mg/m^2 for 3 hours (2).

Identification of the causative toxic agents that lead to the development of peripheral neuropathy is very important in preventing such complications. Regrettably, obtaining such information from the study at hand was impossible. CIPN is an adverse effect of neurotoxic anticancer drugs like platinum-containing drugs (cisplatin, carboplatin, etc.) which appear 1–2 weeks after the start of the therapy; lasts for several months even after the cessation of the treatment; is proportional to the cumulative dose exposure (3). Bias might have been introduced into the reporting of adverse events in this study because the assessment of adverse events was not blinded. A recent review of clinical research reveals that lafutidine (LAF) is associated with toxic epidermal necrolysis, central nervous system side effects, and hepatotoxicity (4).

Perhaps, it would be more reasonable to share the conclusions of the study with the neurology practitioners, pharmacists, and oncologists. The compound may impact overall tumor status and chemotherapy response in oncologists, and impact peripheral neuropathy and nerve strength in neurologists. Some types of pharmacokinetic and pharmacodynamic effects should be considered by pharmacists between LAF and other drugs used in the CIPN treatment (5). Another area of concern that should be addressed is whether the patient has been compliant with LAF. The failure to achieve the results in this trial might be due to the low compliance with the drug regime believed to cause high variability in the result of the trial.

Finally, a more comprehensive perspective would emerge by placing the conclusions of this study within the broader context of contemporary approaches to managing CIPN. The potential benefits and risks of LAF could be compared to those of other preventative therapies like the use of neurotropic agents like N-acetylcysteine or antidepressants like duloxetine. Besides, it would help to identify the course

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of future CIPN preventive research by raising awareness of the comparative advantages of LAF.

Although LAF did not achieve statistical significance in preventing CIPN, the study by Cho *et al.* has set a much-needed foundation for further studies in this field. In conclusion, even though the authors' work is praiseworthy, a more impartial assessment of competing perspectives would enhance LAF. Addressing possible criticisms would, nevertheless, strengthen the author's position and the quality of the study. Our recommendations are intended to increase the effect and reach of the writers' excellent work in the field, which already has a solid foundation

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