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LETTER

Comment On: "Association Between Admission Prognostic Nutritional Index and Pulmonary Infection Status in Hospitalized Lung Cancer Patients: A Retrospective Cohort Study" [Letter]

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Dear editor

I read with great interest the recent article by Wang et al entitled "Association Between Admission Prognostic Nutritional Index and Pulmonary Infection Status in Hospitalized Lung Cancer Patients: A Retrospective Cohort Study", published in the Journal of Inflammation Research.[1] The authors are to be commended for investigating the potential role of the Prognostic Nutritional Index (PNI), a simple and routinely available immunonutritional marker, in identifying lung cancer patients at risk of concomitant pulmonary infection and in-hospital mortality. Nevertheless, several methodological concerns—particularly regarding constituent bias, confounding by indication, and heterogeneity in infection definition—warrant careful consideration, as they may affect the interpretation of the reported associations.

First, PNI is calculated as serum albumin (g/L) plus 5 × lymphocyte count ($\times 10^9$/L). Its validity as a specific marker of infection risk depends on whether key determinants of its two components have been adequately adjusted for. Serum albumin is influenced by multiple factors beyond nutritional status. These include advanced tumor stage, liver dysfunction, protein-losing enteropathy, heart failure, and systemic inflammation.[2,3] Although the authors adjusted for "cancer stage", detailed TNM staging information was not provided. Notably, the infection group had a significantly lower proportion of lymph node metastasis compared to the control group (39.91% vs 51.26%, P=0.024). This finding is counterintuitive and raises the possibility of stage imbalance. If the infection group had more advanced T stage or higher occult metastatic burden (eg, due to disease complications prompting admission), then the observed lower PNI might partly reflect tumor burden, rather than infection per se. Without detailed T, N, and M category data, this potential confounding cannot be fully excluded. Previous research has shown that cancer dissemination is significantly associated with lower PNI and hypoalbuminemia. Patients with albumin levels below 35 g/L show more than fivefold higher likelihood of disseminated disease.[4]

Second, lymphocyte count is highly sensitive to recent or concurrent chemotherapy. In this study, the control group had a markedly higher chemotherapy rate than the infection group (59.30% vs 26.15%, P<0.001). This raises an important question regarding group comparability. The control group (higher chemotherapy rate) likely consisted of elective admissions for scheduled treatment, with better performance status (eg, Eastern Cooperative Oncology Group [ECOG] 0–1) and stable overall condition. In contrast, the infection group (lower chemotherapy rate) was more likely to present with acute illness, poor general condition (ECOG ≥2), malnutrition, or respiratory complications that precluded chemotherapy. Thus, the lower PNI observed in the infection group may reflect a poorer baseline clinical state. It may not represent a specific and independent association between PNI and infection susceptibility. This scenario is consistent with confounding by indication or selection bias. It cannot be fully resolved by adjusting for "chemotherapy" as a binary covariate, because the timing, intensity, and clinical rationale for (not) receiving chemotherapy are likely qualitatively

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different between groups.[5] A recent meta-analysis has confirmed that lower PNI is associated with worse survival outcomes in lung cancer patients receiving chemotherapy. This underscores the need to disentangle treatment-related effects from infection-related effects.[6]

Third, among the 218 patients classified as having concomitant pulmonary infection, 65 (29.8%) lacked microbiological confirmation. These cases were diagnosed solely on clinical and radiological criteria. This introduces heterogeneity in the infection definition. Some of these "clinical diagnosis" cases may have included non-infectious inflammatory conditions, such as radiation pneumonitis, cryptogenic organizing pneumonia, or cancer-associated lymphangitic spread. These conditions can mimic infection clinically and radiologically. Including such patients could attenuate or bias the observed associations between PNI and infection status.[7]

In summary, Wang et al have provided valuable preliminary evidence suggesting an association between admission PNI and pulmonary infection in hospitalized lung cancer patients. However, due to potential constituent bias, confounding by indication, and heterogeneity in infection definition, the independent nature of this association may be overestimated. Future prospective multicenter studies are warranted. These studies should provide detailed TNM staging, standardized performance status assessment, microbiologically confirmed infection criteria, and careful adjustment for treatment-related factors. Only with these measures can their findings be validated and the true incremental clinical value of PNI be clarified.

I thank the authors for their important contribution and hope these considerations may assist in refining future research in this area.

Sincerely,

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Data Sharing Statement

Data sharing does not apply to this article as no data were created or analyzed in this study.

Author Contributions

Qinqin Dan: Conceptualization, Writing - original draft.

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