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LETTER

Letter to the Editor Regarding: "Association of Prognostic Nutritional Index with Mortality in High-Risk OSA Individuals and with Disease Severity in Clinical OSA Patients" [Letter]

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Dear editor

We read with interest the study by Li et al investigating the association of prognostic nutritional index (PNI) with mortality in individuals at high risk for obstructive sleep apnea (OSA) and with disease severity in clinically confirmed OSA patients.[1] The authors provide valuable insights into the potential utility of PNI as an integrated marker of nutritional and immune status for risk stratification in OSA populations. While we commend the methodological rigor, we offer several complementary reflections to strengthen the validity and clinical applicability of the findings.

First, regarding participant selection, we believe that individuals with acute infections warrant additional attention. Such conditions can influence both components of PNI. In acute infections, lymphocyte counts may rise transiently due to immune activation, while serum albumin, with a half-life of approximately 20 days, may remain unchanged, leading to a falsely elevated PNI. Conversely, severe or prolonged infections promote immune cell apoptosis and reduce lymphocyte counts, and suppress albumin synthesis as a negative acute-phase protein, resulting in reduced PNI. A narrative review further highlights that serum albumin should not be used as a surrogate for nutritional status as it is heavily influenced by disease factors like inflammation, and is no longer recommended by the American Society for Parenteral and Enteral Nutrition (ASPEN) as a pure nutritional marker.[2] Thus, we suggest excluding individuals with acute infections or high inflammatory markers to ensure PNI reflects the baseline nutritional-immune state, and continuing to explore robust nutritional biomarkers that better characterize malnutrition phenotypes.

Second, with respect to the statistical analysis of the association between PNI and cardiovascular mortality, we would like to raise the issue of competing risks. During follow-up, 1025 all-cause deaths occurred, while only 287 were cardiovascular deaths. This substantial proportion of non-cardiovascular deaths introduces a competing risk that may lead Cox proportional hazards models to overestimate the cumulative incidence of the cause-specific event, which assumes independent censoring. We recommend applying competing risk models, such as the Fine-Gray model, for more precise risk estimation.

Third, while PNI demonstrated higher AUC values (e.g., 0.74 for 12-month all-cause mortality) compared to its individual components, this level of discrimination is generally considered "acceptable" rather than "superior" for clinical decision-making. To more rigorously evaluate the incremental predictive value of PNI, we suggest that future studies calculate the net reclassification improvement (NRI) and integrated discrimination improvement (IDI), and perform decision curve analysis (DCA). Additionally, reporting optimal cut-off values would help translate PNI into a clinically actionable tool for risk stratification.

In conclusion, we commend Li et al for their insightful work. We hope these methodological considerations will aid in further elucidating the predictive value of PNI and improve personalized care for patients with OSA.

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