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Letter to the Editor

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Comment on: The relationship between troponin levels in pneumonia and the severity of pneumonia and the oxidant-antioxidant balance

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Dear Editor,

I read with great interest the recent article by Öner and Karalezli[1] evaluating the relationship between troponin levels, oxidative stress markers, and pneumonia severity. The authors should be commended for addressing an important and relatively underexplored aspect of pneumonia pathophysiology, particularly the integration of cardiac biomarkers and redox balance into clinical assessment.

From the perspective of an emergency physician, the identification of readily available biomarkers that may aid in early risk stratification is of substantial clinical value. The observed association between elevated troponin levels and increasing Pneumonia Severity Index (PSI) stages is consistent with prior evidence suggesting that myocardial injury frequently accompanies severe infection and systemic inflammatory states, including sepsis.[2] In this context, troponin elevation may reflect not only primary cardiac pathology but also secondary myocardial stress related to hypoxia, inflammation, and hemodynamic instability.

However, an important consideration in emergency department practice is the limited specificity of troponin elevation. As highlighted in previous literature, cardiac troponins may be elevated in a wide range of non-coronary conditions, including pulmonary and systemic diseases, which can complicate clinical interpretation and decision-making.[3] Therefore, while the findings of the present study are clinically intriguing, the routine use of troponin as a standalone marker for pneumonia severity should be approached with caution, particularly in heterogeneous emergency populations.

The inclusion of oxidative stress parameters, such as thiol–disulfide balance and ischemia-modified albumin, represents a novel and valuable aspect of the study. Increasing evidence suggests that oxidative stress plays a significant role in the pathophysiology and progression of community-acquired pneumonia.[4] The demonstration of altered redox homeostasis in parallel with disease severity provides additional insight into the underlying biological processes and may contribute to a more comprehensive assessment framework.

From a practical standpoint, however, the applicability of these biomarkers in the emergency setting remains uncertain. Unlike troponin, measurements such as thiol–disulfide balance and ischemia-modified albumin are not routinely available in most emergency departments, which may limit their immediate clinical utility. In time-critical environments where rapid decision-making is essential, biomarkers must not only be informative but also accessible and cost-effective.

In addition, established clinical prediction tools such as PSI and CURB-65, although imperfect, remain the cornerstone of risk stratification in pneumonia. Prior studies have demonstrated that the addition of novel biomarkers may improve prognostic accuracy, but this incremental benefit must be weighed against practicality and clinical impact.[5]

In conclusion, this study provides valuable insight into the interplay between myocardial injury, oxidative stress, and pneumonia severity. While the findings are promising, further large-scale, prospective studies are needed to clarify the clinical role and added value of these biomarkers in emergency care settings. I thank the authors for their contribution to this evolving field.

ETHICAL DECLARATIONS

Peer Review Process

This letter was externally peer-reviewed.

Conflict of Interest

The author declares no conflicts of interest.

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Author Contributions

The author is solely responsible for the conception, data collection, analysis, and writing of this manuscript.

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Author reply to "The relationship between troponin levels in pneumonia and the severity of pneumonia and the oxidant-antioxidant balance"

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Dear Editor,

We would like to thank the author for their thoughtful and constructive comments on our study entitled "The relationship between troponin levels in pneumonia and the severity of pneumonia and the oxidant-antioxidant balance." We are pleased that the clinical relevance of integrating cardiac biomarkers and oxidative stress parameters in pneumonia has been recognized.

We fully agree with the comment regarding the limited specificity of cardiac troponins. As emphasized both in our discussion and in previous literature, troponin elevation is not specific to acute coronary syndromes and may occur in a variety of conditions, including systemic infections and pulmonary diseases. In our study, we aimed to minimize this limitation by excluding patients with acute coronary syndrome, renal failure, and other major confounding conditions. Moreover, our primary objective was not to propose troponin as a standalone diagnostic marker, but rather to evaluate its association with disease severity in the context of pneumonia. In this regard, we believe that the observed correlation between troponin levels and PSI stages supports its potential role as an adjunctive biomarker reflecting systemic disease burden and possible myocardial involvement.

We also appreciate the emphasis on the challenges of clinical interpretation in emergency settings. We agree that, due to its limited specificity, troponin should be interpreted cautiously and always in conjunction with clinical findings, imaging, and other laboratory parameters. Our findings should therefore be considered as hypothesis-generating rather than practice-changing.

Regarding oxidative stress markers, we agree that parameters such as thiol–disulfide balance and ischemia-modified albumin are not yet routinely available in most emergency departments. The primary aim of including these markers was to provide a more comprehensive pathophysiological perspective by demonstrating the relationship between oxidative stress and disease severity. While their immediate clinical applicability may be limited, we believe that these findings may contribute to future research exploring more accessible or standardized oxidative stress markers.

We also concur that established clinical scoring systems such as PSI and CURB-65 remain the cornerstone of pneumonia risk stratification. Our intention was not to replace these tools but to explore whether additional biomarkers could complement existing systems. As noted, the incremental clinical value of such biomarkers should be validated in larger, prospective, and multicenter studies.

Finally, we agree that further large-scale studies are needed to clarify the clinical utility, cost-effectiveness, and practical integration of these biomarkers into routine care. Our study should be viewed as an initial step toward better understanding the complex interaction between myocardial injury, oxidative stress, and pneumonia severity.

We thank the author again for their valuable comments and for contributing to the scientific discussion in this evolving field.

Sincerely,