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Reply to the letter to the editor: The role of diaphragm thickness and mobility in chronic obstructive pulmonary disease classification and exacerbations

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We sincerely thank you for the opportunity to respond to the Letter to the Editor^[1] regarding our article, "The Role of Diaphragm Thickness and Mobility in Chronic Obstructive Pulmonary Disease Classification and Exacerbations."^[2] We are also grateful to the author(s) for their careful reading of our work and for their thoughtful and constructive comments.

We appreciate the positive remarks concerning the clinical relevance of diaphragmatic excursion and its association with the GOLD classification and the frequency of exacerbations. As emphasized both in the letter and in our study, diaphragmatic dysfunction represents an important yet often underrecognized component

of COPD pathophysiology. Our findings demonstrated that deep inspiratory diaphragmatic excursion differed significantly between COPD patients with and without exacerbations in univariate analysis. These results are consistent with previous literature and support the value of diaphragm ultrasonography as a practical tool for functional assessment and clinical stratification in COPD.

Regarding diaphragm thickness, we acknowledge the important point raised that there is no significant relationship with disease severity. The literature indeed contains both concordant and conflicting findings.^[3-5] In our study, diaphragm thickness showed a gradual increase from GOLD A to C and then

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decreased in GOLD D, although the differences were not statistically significant. This pattern may suggest early adaptive hypertrophy of inspiratory muscles in response to increased respiratory load, with possible atrophy in advanced stages due to oxidative stress and muscle remodeling. While this observation was not statistically significant, it may reflect the complex structural adaptations occurring across disease stages.

Diaphragm thickening fraction, considered an indirect indicator of muscle fiber contractility, has been proposed as a potentially more sensitive parameter than thickness alone. Experimental data demonstrate alterations in diaphragm fiber structure and contractile proteins in COPD.^[6] However, in our cohort, the thickening fraction was not associated with GOLD classification or exacerbation outcomes. Similar findings were reported by Baria *et al.*,^[7] who suggested that diaphragmatic dysfunction in COPD may primarily reflect mechanical limitation related to hyperinflation rather than intrinsic contractile impairment. These findings highlight the complex interplay between structural remodeling and mechanical disadvantage in COPD. Functional parameters such as diaphragmatic excursion may better capture the diaphragm's integrated mechanical performance.

We fully agree that standardization of ultrasonographic techniques and of thickening-fraction calculation methods is important for improving comparability across studies. Further longitudinal investigations incorporating both structural and functional assessments of the diaphragm may help clarify these relationships.

We again thank the author(s) for their valuable comments and for contributing to a constructive scientific discussion.

Sincerely,

On behalf of the authors

Conflicts of Interest

The authors have no conflicts of interest to declare.

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