

Emphysema affects the number and characteristics of solitary pulmonary nodules

To the editor We read with interest the article entitled “Emphysema affects the number and characteristics of solitary pulmonary nodules identified by chest low-dose computed tomography” by Wachuła et al¹ published in the January issue of *Polish Archives of Internal Medicine (Pol Arch Intern Med)*. We would like to discuss 4 issues.

First, the title of this article includes a statement that emphysema affects the number and characteristics of solitary pulmonary nodules. However, the presence of emphysema itself did not appear to affect the number or characteristics of nodules, and we do wonder whether these patients might have such nodules. I would be grateful for the authors’ comments on this issue.

Second, the authors reported that there were differences in nodule size, location, number, and morphology between those with and without emphysema. It would be interesting to know how the authors interpret these differences between groups.

The third point is a query about the patients evaluated in this study. We would like to know whether the study included patients with combined pulmonary fibrosis and emphysema, which is associated with a high incidence of lung cancer.^{2,3} The study included patients with a history of exposure to chemical compounds, such as coal miners. How did this affect the results? Last, the diagnosis of nodules was unclear because the study did not incorporate a full pathological examination. However, we would appreciate the authors’ evaluation of, for example, the intrapulmonary lymph nodes and postinflammatory scars.

ARTICLE INFORMATION

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Authors’ reply Thank you for your interest in our study. Although emphysema has been determined to be an independent predictor of lung cancer development, the underlying phenomenon is not satisfactorily elucidated.¹ The most likely chain of events is structural reshaping of the lung elicited by a chronic inflammatory process as a result of exposure to multiple chemical compounds in tobacco smoke and air pollutants.

Here, we address your doubts and queries in the order they appear in your letter.

First, we demonstrated that emphysema modifies the features of a solitary pulmonary nodule (SPN), as documented in tables 3 and 4. Note that there was a tendency for SPN to have bigger diameter in the emphysema coexisting with nodules (E + N) group in comparison with the group with nodules without emphysema (median [IQR], 12 [3] mm vs 10 [15] mm, respectively; $P = 0.047$). Additionally, nonsolid nodules were more numerous in the E + N group (36.5% vs 21.1%, respectively; $P = 0.01$), whereas solid and part-solid SPNs prevailed in the group with nodules without emphysema. Moreover, the multiple SPNs were more abundant in the E + N group (71.9% vs 56.3%; $P = 0.02$).

Understandably, individuals with emphysema were characterized by older age and higher cumulative tobacco consumption.

Second, query regarding the difference in SPN morphology, size, and number between the 2 groups remains a question open to debate.

From the clinical point of view, our findings confirm the need to incorporate additional factors beyond participants' age and pack-years smoked when considering a target screening cohort. Emphysema is included into the set of risk factors but is not a variable used in risk assessment prediction models like the Bach model, LDCRAT (Lung Cancer Death Risk Assessment Tool), or others. Quite recently, Tammemägi et al² proposed to include the result of the initial low-dose computed tomography into the prediction regression equation. Yong et al,³ with the use of prediction tests, showed that the number needed to screen to detect 1 patient with cancer was lower in the participants with radiological emphysema.

Third, our cohort did not include patients with emphysema and idiopathic pulmonary fibrosis. It is an in-depth question since both diseases are risk factors listed to include a screenee into group 2 according to the guidelines of the National Comprehensive Cancer Network.⁴ Nonetheless, most likely, an individual with concomitant occurrence of these disorders would not be a candidate for screening due to respiratory insufficiency.

Fourth, unfortunately, lack of pathological information pertaining to the nodules is the limitation of our study. This, in turn, is due to the relatively small size of our cohort. This means we know pathological outcome only for those patients with SPNs who underwent diagnostic work-up. In our study, we focused only on the baseline low-dose computed tomography. In further reports we plan to focus on the consecutive low-dose computed tomography rounds.

We hope that this response has, at least to some extent, clarified your queries.

ARTICLE INFORMATION

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