

Correspondence

Increased eosinophil uptake in the lungs of obese patients with asthma—to correct for obesity compare to obese controls

To the Editor:

We read with great interest the study by Farahi et al¹ reporting that single photon emission computed tomography/computed tomography imaging using radiolabeled eosinophils could be used to quantify eosinophil uptake from the blood to the lungs. We agree with the proposal that bronchial eosinophil count and sputum eosinophil count are not correlated. Bronchial biopsies are currently the golden standard to investigate bronchial eosinophilic inflammation. However, a bronchial biopsy is an invasive procedure, which makes the noninvasive method from Farahi et al more interesting. Although Farahi et al did take bronchial biopsies, they did not correlate this eosinophil count to the quantification of eosinophils by single photon emission computed tomography/computed tomography.

Farahi et al also found that eosinophil uptake is enhanced in obese patients with asthma compared with nonobese patients with asthma, and concluded that this finding has important implications for the role of eosinophils in obese asthma and for selecting patients for targeted therapy in the context of type 2 inflammation. However, if one postulates that the obese patients with asthma have more eosinophils because they are obese, one should not compare them with lean patients with asthma, but with obese controls. Or, in other words, the differences Farahi et al found can also be attributed to obesity alone rather than asthma. Particularly because obesity can be seen as a state of low-grade systemic inflammation, one should correct for this systemic inflammation, by comparing obese patients with asthma to obese controls.

We compared (morbidly) obese patients with asthma to (morbidly) obese controls by studying bronchial biopsies.^{2,3} We found no differences between the obese patients with asthma and obese controls, neither before nor after dramatic weight loss induced by bariatric surgery.² Bariatric surgery improves asthma control, asthma-related quality of life, lung function, and medication use. So, weight loss, or bariatric surgery, should be the cornerstone of the treatment of obese patients with asthma.

As mentioned by Farahi et al, the obese patient with asthma is a distinct phenotype, as already characterized by Haldar et al⁴ as a phenotype with low eosinophilic inflammation, but high symptom scores. It is also speculated that the obese asthma phenotype could

be further divided into T_H2-high (high serum IgE) obese patients with asthma having pre-existing allergic asthma that is complicated by obesity, whereas T_H2-low (low serum IgE) obese patients with asthma develop asthma symptoms as a consequence of obesity. This subphenotyping is confirmed by the fact that airway hyperresponsiveness in T_H2-low obese patients with asthma improves after weight loss following bariatric surgery, in contrast to T_H2-high patients with obese asthma.⁵ The latter phenotype might benefit from (inhaled) steroids, whereas steroids might be deleterious in the first T_H2-low phenotype. Farahi et al provide no further information about their subjects to make this subphenotyping.

In conclusion, we question the conclusion of Farahi et al that asthma with obesity, in contrast to previous studies, is associated with eosinophilic inflammation. To make this conclusion, the study should be repeated adding a subgroup of obese controls without asthma, to fully control for the effect of obesity on the inflammation present in bronchial tissue. And perhaps then an answer will come to the remaining question why some obese develop asthma whereas others do not.

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