Diabetes and Lung Function

Part of a Wider Spectrum

To the Editor:

The metaanalysis by van den Borst et al\(^1\) in a recent issue of CHEST (August 2010) importantly provided further indication that, similar to the cardiovascular system, the lung is a target organ in the systemic inflammatory process among subjects with type 2 diabetes. The International Diabetes Association suggests that 80% of these patients are overweight or obese, and because there is already a well-established relationship between obesity and abnormal lung function, the findings reported are not unexpected.\(^2\) Similarly, the role of inflammation among such patients is already more widely recognized as a part of the metabolic syndrome.\(^4\) The evidence to date on lung function, however, has not been entirely consistent, and although studies have generally established subjects who are obese as being more prone to reduced FEV\(_1\), FVC, and total lung capacity and, therefore, showing a restrictive pattern, there is also contradicting evidence, with the prevalence of diabetes in individuals who are obese shown to be inversely related to lung restriction rather than obstruction.\(^5\) Mechanisms contributing to restriction relate more to chest-wall mechanics, with body deposition of fat, reduced diaphragmatic excursion due to increased abdominal adiposity, or increased weight on the chest wall\(^6\) providing evidence for additional pleural or interstitial lung disease because a more systematic inflammatory process is not as readily evident. Leptin, which is increased in patients who are obese, may be a potential confounder in determining abnormal lung function, as may smoking, and both of these risk factors for morbidity are frequently found among these adult patients. Clarification, possibly using high-resolution chest CT imaging or full-lung-function tests, including lung volume and gas transfer tests, is needed in this population to establish whether restriction is exclusively due to obesity and altered chest mechanics.\(^7\)

REFERENCES


Response

To the Editor:

In response to the letter to Ahmad et al, we would like to clarify how we carefully dealt with BMI in our metaanalysis on pulmonary function in diabetes in our recent article in CHEST (August 2010). All the studies we included in our metaanalysis had a control group, and in almost every study BMI was already closely matched between the diabetes patients and control subjects by the original authors. Still, to exclude the potential influence of a relatively small difference in mean BMI between diabetes patients and control subjects, we analyzed whether the delta of the mean BMI between the groups was a source for between-study heterogeneity, which was proven not to be the case. Therefore, we concluded that the observed differences in pulmonary function indices between diabetes patients and controls were independent of BMI. This finding from our quantitative review is also supported by a recent narrative systematic review on pulmonary function specifically in type 2 diabetes. Ahmad et al directly link the lower pulmonary function in type 2 diabetes patients to systemic inflammation, for which no direct evidence exists. To the contrary, as we discussed, two large studies that sought a systemic inflammatory explanation for lower pulmonary function in diabetes failed to provide significant findings. It has yet to be elucidated whether the subclinically lower lung function associated with diabetes indeed forms a risk of progressive decline.

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