Metabolic Syndrome and Impaired Lung Function

To the Editor:

We read with great interest the recent article in CHEST (October 2009) by Watz and colleagues showing an independent association between metabolic syndrome and systemic inflammatory markers in chronic bronchitis and patients with COPD. The authors also demonstrate that the prevalence of metabolic syndrome does not increase for increasing COPD severity, as expressed by the Global Initiative for Chronic Obstructive Lung Disease stage.

Interestingly, metabolic syndrome is also associated with a restrictive ventilatory pattern at spirometry, especially in patients with the highest waist circumference. In this population, visceral fat is known to produce prothrombotic and inflammatory mediators, including C-reactive protein, fibrinogen, interleukin-6, and tumor necrosis factor-α. Since lung restriction is frequently associated with systemic inflammation independent of obesity, the inflammatory burden due to restriction may add to that related to visceral obesity in patients having both diseases. For this reason we believe that the authors should have provided information on the prevalence of a mixed ventilatory pattern in their population instead of classifying patients only on the basis of the FEV1/FVC ratio and FEV1%. This would have required the measurement of total lung capacity. Nonetheless, based on the available data, it would be of interest at least to know how prevalent was a spirometric pattern suggesting a restrictive component, which is known to be associated with systemic inflammation. Indeed, recent evidence is consistent with an FVC based on presumptive diagnosis of lung restriction being comparably accurate in people with and without obstruction. Finally, the authors provide the Charlson index of comorbidity, but they do not list individual comorbidities and their prevalences; selected conditions, such as renal failure, could per se promote systemic inflammation.

Providing such information would allow the authors and the readers to verify whether the inflammatory pattern changes for different combinations of COPD, a restrictive component and visceral obesity. Otherwise, the authors might ascribe to COPD an inflammatory status, which in a relevant proportion of patients likely is multifactorial in origin.

Simone Scarlata, MD
Filippo Luca Finoguari, MD
Leo Moro, MD
Ruggiero Pastorelli, MD
Rome, Italy
Raffaele Antonelli-Incalzi, MD
Taranto, Italy

Affiliations: From the Health Center for Elderly (Centro per la Salute dell’Anziano) (Drs Scarlata, Moro, and Finoguari), Unit of Respiratory Pathophysiology, Università Campus BioMedice; the “Alberto Sordi” Foundation – Olnus (Drs Scarlata and Moro); the Unit of Respiratory Diseases (Drs Finoguari and Pastorelli), Division of Internal Medicine, ASL Roma G Leopoldo Parodi-Delfit no Hospital, Colleferro; and the S. Raffaele – Cittadella della Carità Foundation (Dr Antonelli-Incalzi).

Financial/nonfinancial disclosures: The authors have reported to CHEST that no potential conflicts of interest exist with any companies/organizations whose products or services may be discussed in this article.

Correspondence to: Simone Scarlata, MD, e/o Centro per la Salute dell’Anziano, Via Alvaro del Portillo, 21, 00128 Rome, Italy; e-mail: s.scarlata@unicampus.it

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DOI: 10.1378/chest.09-1539

REFERENCES


Response

To the Editor:

We thank Dr Scarlata and colleagues for their interest in our study results and the comments about possible mechanisms related to systemic inflammation. Dr Scarlata and colleagues argue that a restrictive component of lung function might contribute to the presence of systemic inflammation in our patients with a coexisting metabolic syndrome. We can exclude that such a restrictive component had that effect in our study population because no patient had a total lung capacity < 80%. We did not give the results of body plethysmography previously because they neither contribute to the severity of COPD according to the Global Initiative for Chronic Obstructive Pulmonary Disease nor are they part of the metabolic syndrome.

We appreciate the comment regarding the Charlson index. The Charlson index can indeed only give the information that comorbidities exist at all and it does not discriminate between different entities. However, to date there are 45 studies available that applied the Charlson index in patients with COPD. Therefore, it allows some level of comparison among different study populations.