A Possible Role of Visceral Fat-Related Inflammation in Linking Obstructive Sleep Apnea to Left Ventricular Hypertrophy

To the Editor:

We read with great interest the article by Avelar et al. titled “Left Ventricular Hypertrophy in Severe Obesity. Interactions Among Blood Pressure, Nocturnal Hypoxemia, and Body Mass.” Although the contribution of obstructive sleep apnea to the development of left ventricular remodeling is not confirmed by all of the previous studies, the authors pointed out that a reduced oxygen saturation resulted as the strongest independent predictor of left ventricular remodeling, with body mass index and high blood pressure amplifying the effect. In the accompanying editorial commentary, de Simone discussed widely the role of various pathophysiologicals possibly involved in this association. However, it does not make mention of the putative implication of visceral adipose tissue (VAT) and its related inflammatory background.

We have suggested recently, in uncomplicated morbid obesity, that the activation of inflammatory pathways within the myocardium could be responsible for a correlative and causative relationship between abdominal VAT and subclinical echocardiographic abnormalities. This was suggested by the association of both echocardiographic parameters and VAT area with the levels of bioactive proteins involved in ventricular remodeling (eg, monocyte-chemoattractant protein-1, C-reactive protein, and soluble interleukin-6 receptor/interleukin-6 complex). Thus, it is reasonable to consider a role for inflammation in ventricular remodeling, starting before the occurrence of cardiovascular complications (eg, hypertension).

Abdominal fat accumulation also represents the anatomic precondition for an altered nocturnal ventilatory drive. An increased VAT-related inflammatory background was also described for obstructive sleep apnea with its treatment leading to an improvement. However, this reduction in cytokines reaches levels comparable to those of obese controls; thus suggesting a worsening proinflammatory role of obstructive sleep apnea. In this sense, the higher predictive role of hypoxemia may find a further possible explanation. However, the poorer association between left ventricular hypertrophy and body mass index could be ascribed to the high degree of obesity of the population studied by Avelar et al. Although fitting well to population-based studies, body mass index should be regarded as an index of weight excess, because it does not distinguish between VAT from subcutaneous abdominal fat, particularly in morbid obesity. The quantitative assessment of VAT, other than by echography (eg, by computed tomography), is not of clinical use. However, the quantification of VAT surrogates (eg, waist circumference, waist/hip ratio, or epicardial fat by echocardiography) is feasible. Considering the inflammatory background associated with fat distribution, future research might provide further clarifications of the association between obstructive sleep apnea and left ventricular hypertrophy.

Disclosures

None.

Emanuele Cereda
Alexis Elias Malavazos
Endocrinology Unit
Department of Medical and Surgical Sciences
University of Milan
IRCCS Policlinico San Donato
Milano, Italy

References

A Possible Role of Visceral Fat-Related Inflammation in Linking Obstructive Sleep Apnea to Left Ventricular Hypertrophy
Emanuele Cerda and Alexis Elias Malavazos

Hypertension. 2007;49:e23; originally published online February 19, 2007;
doi: 10.1161/01.HYP.0000259820.28092.23
Hypertension is published by the American Heart Association, 7272 Greenville Avenue, Dallas, TX 75231
Copyright © 2007 American Heart Association, Inc. All rights reserved.
Print ISSN: 0194-911X. Online ISSN: 1524-4563

The online version of this article, along with updated information and services, is located on the World Wide Web at:
http://hyper.ahajournals.org/content/49/4/e23

Permissions: Requests for permissions to reproduce figures, tables, or portions of articles originally published in Hypertension can be obtained via RightsLink, a service of the Copyright Clearance Center, not the Editorial Office. Once the online version of the published article for which permission is being requested is located, click Request Permissions in the middle column of the Web page under Services. Further information about this process is available in the Permissions and Rights Question and Answer document.

Reprints: Information about reprints can be found online at:
http://www.lww.com/reprints

Subscriptions: Information about subscribing to Hypertension is online at:
http://hyper.ahajournals.org//subscriptions/