The Role of Aldosterone in Pulmonary Venous Hypertension

To the Editor:

We read with interest the recent article in CHEST (July 2009) by Robbins et al. 1 demonstrating an association between the metabolic syndrome and the existence of pulmonary venous hypertension. Recent research has also demonstrated an association between serum aldosterone, a known cause of left ventricular diastolic dysfunction and remodeling, and the metabolic syndrome. 2-4 Thus, it is possible that increased circulating aldosterone might be independently associated with the risk for pulmonary venous hypertension. Do the authors have data examining aldosterone or renin activity in their subjects?

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REFERENCES

Response

To the Editor:

We thank Drs Farber, Walkey, and Alikhan for bringing to our attention the importance of aldosterone in the development of left ventricular (LV) diastolic dysfunction. The studies noted in their letter highlight the importance of metabolic derangements in the development of LV diastolic dysfunction, which can occur even in the absence of systemic hypertension or LV hypertrophy. 1-3 We did not measure aldosterone levels in our study 4 but plan to do so in future studies.

There is increasing evidence that features of the metabolic syndrome are likely to contribute to the development of pulmonary hypertension in susceptible patients. 5 This is an area that requires further investigation, and we were intrigued by the recent publications of Dr Farher and his colleagues about the potential role of adiponectin deficiency in the development of pulmonary hypertension. 6

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REFERENCES

A Comparative Study of Two Different Metered-Dose Inhaler-Valved Holding Chambers in the Administration of Salbutamol

To the Editor:

Rapid-acting, inhaled β-2 agonists are frequently delivered from a pressurized, metered-dose inhaler (MDI) used with a valved holding chamber (VHC). Several studies have shown that VHCs enhance the efficacy of short-acting β-2 agonists in patients who have poor MDI technique and in children. However, different VHCs are available for inhaled therapy without information in the summaries of product characteristics and patient information leaflets on studies of efficacy and compatibility between drugs and
VHCs. To provide that, we suggest a method already proposed to standardize first-step delivery of aerosolized drugs. 1

We compared the performance of a new VHC (L’Espace; Markos Mefar; Bovezzo Bs, Italy) with the AeroChamber (Trudell Medical International; Plattsburgh, NY) to verify whether they were equally suitable for use in the delivery of a specific drug. Their efficacy was expressed as: (1) the amount of drug within the respirable range (ADRR) according to the formula described by Malone 2 that is, the drug output (measured immediately after treat-

Table 1—Baseline Spirometric Parameters and Related Improvements After Therapy

<table>
<thead>
<tr>
<th>VHCs</th>
<th>FEV1</th>
<th>PEF</th>
<th>FEV50</th>
</tr>
</thead>
<tbody>
<tr>
<td>L’Espace</td>
<td>55.4 ± 5.9</td>
<td>15.4 ± 12.0</td>
<td>60.4 ± 13.8</td>
</tr>
<tr>
<td>AeroChamber</td>
<td>52.5 ± 15.8</td>
<td>16.1 ± 13.0</td>
<td>55.9 ± 16.0</td>
</tr>
</tbody>
</table>

Baseline spirometric parameters and related improvements after therapy are expressed as percentages of predicted values. FEV50 = forced expiratory flow at 25% to 75% of the forced vital capacity; PEF = peak expiratory flow; VHCs = valved holding chambers.

REFERENCES


New Disease—New Terminology

To the Editor:

We read with interest the recent letter to the editor and response 1 in CHEST (June 2009) by Naccache et al and Wynn et al, respectively, in reference to the article by Wynn and colleagues 2 in CHEST (November 2008). They reported anthracofibrosis associated with dust exposure, in particular, coal dust and mixed mineral dust, as a new occupational lung disease. In these writings, anthracofibrosis was defined as “narrowing of the bronchial lumen with overlying anthracotic mucosa” and “a distinct entity of inflammatory bronchial stenosis with overlying anthracotic mucosa,” respectively. We propose a new term, “anthracostenosis,” to further characterize anthracofibrosis due to massive progressive fibrosis in patients with coal worker’s pneumoconiosis (CWP), silicosis, or other forms of pneumoconiosis who present with bronchostenosis to distinguish it from other causes of anthracofibrosis. 3

The diagnosis of anthracofibrosis is established bronchoscopically when bronchial narrowing or obliteration is seen in association with anthracotic pigmentation of the overlying bronchial mucosa. Albeit endobronchial anthracosis is not an uncommon finding on bronchoscopic airway inspection even in individuals with no reported environmental exposure, bronchial stenosis associated with it is rare. Anthracofibrosis is more widely described in Asian and black elderly women who present with respiratory symptoms. In contrast to endobronchial tuberculosis, its involvement of the bronchial wall is typically multifocal without continuity in the distal lobar and/or segmental bronchi bilaterally and often sparing the trachea and main bronchial tree. 4 Although prevalently imputed to TB infection previously, its causality has now been recognized to be linked to environmental air pollution or domestic biomass smoke (including cigarette smoke exposure), in addition to occupational dust inhalation. 5

The pathogenesis of bronchostenosis in CWP and silicosis has been attributed to external luminal constriction from adjacent lymphadenopathy or fibrosis, erosion by enlarged or inflamed lym

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