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LETTERS TO THE EDITOR

Response to "Sleep apnea and pulmonary hypertension: connecting the dots"

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Sharma et al[1] disagree with the main finding of our study[2] that included 493 patients with OSA who underwent right heart catheterization. We identified that pulmonary hypertension (PH) is associated with the percentage of sleep time spent with a blood oxygen saturation level < 90% but not with AHI.

In our opinion, a direct correlation between AHI and mean pulmonary artery pressure remains unproven. Our study showed no difference in AHI based on the presence/absence of PH, PH type, or cardiopulmonary hemodynamics. In a prospective study of 49 patients with precapillary PH, mean pulmonary artery pressure measured by right heart catheterization did not correlate with the presence of sleep apnea or AHI.[3] In another study of 53 patients with precapillary PH, cardiopulmonary hemodynamics obtained by right heart catheterization were similar in patients with and without sleep apnea—2 groups that by definition have different AHIs.[4]

Two randomized controlled trials were referenced in the Sharma et al letter[1]: Arias et al,[5] which included 23 patients with OSA, 10 of whom had PH, and Sharma, Fox, et al,[6] which included 21 patients with PH accompanying left-sided cardiac dysfunction. These 2 studies share 2 important limitations: (1) the sample size is small, and (2) PH was diagnosed and followed up using echocardiography. Echocardiography is an excellent screening modality but a poor diagnostic modality for PH given its lack of specificity and its inability to accurately estimate pulmonary artery systolic pressure. In one study, echocardiographic estimates of pulmonary artery systolic pressure were unacceptably discordant with the measurements simultaneously obtained by right heart catheterization; the 95% limit of agreement ranged from –28.4 to +44.4 mm Hg.[7]

In Arias et al,[5] the OSA group with PH had a higher pretreatment AHI than the group without PH. Notably, the PH group also had a higher body mass index (33.6 vs 28.9; P = .006) and a higher prevalence of diastolic dysfunction (90% vs 31%; P = .006); these 2 confounders limit the interpretability of the reported association between AHI and PH. Similar to our study, Arias et al[5] reported a 3-fold higher blood oxygen saturation level < 90% in the PH group. Although statistical significance was not reached (P = .08), the trend was strong and likely would have been significant with a larger sample size.[5]

Finally, 30 of the 31 patients with PH included in Arias et al[5] and Sharma, Fox, et al,[6] had left-sided cardiac dysfunction, which is the most common cause of PH.[5,6] Studies, including Sharma, Fox, et al[6], have shown that interventions directed at treating left-sided cardiac dysfunction consequently improve the accompanying PH. CPAP use has proven beneficial in left-sided cardiac dysfunction through multiple mechanisms, irrespective of the presence of PH or OSA. Arias et al[5] and Sharma, Fox, et al[6] observed an improvement in left-sided cardiac performance, pulmonary artery systolic pressure and, as expected, AHI among patients with OSA receiving CPAP.[5,6] However, the presence of left-sided cardiac dysfunction confounds the premise that AHI and pulmonary artery systolic pressure are directly related.

Overall, inconsistencies in the existing literature lead to ambiguities of the interrelationships of sleep-disordered breathing (whether defined by a frequency measure or nocturnal hypoxia) and PH pathophysiology and progression. Larger prospective studies and clinical trials are needed to better elucidate these associations, considering specific PH phenotypes.

CITATION

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DISCLOSURE STATEMENT

The authors have no conflicts to report.