The Burden of Obstructive Sleep Apnea Along With Masked Hypertension on Elastic Properties

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The ongoing investigation on the detrimental effects of obstructive sleep apnea (OSA) on the cardiovascular system has already identified intermediate pathways leading to overt cardiovascular disease such as sympathetic activation, systemic inflammation, and endothelial dysfunction. Special attention should be paid to increased arterial stiffness, which has been associated with OSA of varying severity in both normotensive and hypertensive populations.¹

Masked hypertension has been associated with target organ damage and increased cardiovascular risk.² In a relatively recent study of OSA-only patients, the high prevalence of masked hypertension was documented, whereas it was found to stand between frank normotension and hypertension, regarding organ damage parameters like ventricular and carotid hypertrophy.³

In the present issue of the American Journal of Hypertension, Drager et al. investigated the interrelation of OSA, masked hypertension, and arterial stiffness in a cross-sectional controlled study of overweight males with moderate-to-severe OSA.⁴ The study confirms the correlation of OSA severity indexes to arterial stiffness, further identifying an independent association of oxygen saturation with pulse wave velocity. It also recognizes increasingly impaired arterial elasticity when comparing individuals without OSA, patients with OSA but without masked hypertension and patients with OSA and masked hypertension.

In this study, a higher proportion of patients with OSA exhibited masked hypertension compared to non-OSA subjects. As there was no adjustment for potential confounders, this higher incidence of masked hypertension should be interpreted with caution in this setting. Another limitation of the study is the exclusion of cases with mild degree of OSA as well as female patients, that both comprise important proportions of individuals with OSA. A larger sample size also including these groups, would have been more appropriate. Especially, mild OSA is largely underdiagnosed, and its clinical implications and the need for therapy have often been a matter of dispute.

Because patients with OSA often exhibit a disturbed 24-h blood pressure (BP) pattern, evaluation of BP levels should not be limited to office measurements alone but specifically include ambulatory monitoring. This study further strengthens the need for ambulatory BP monitoring for identifying patients with masked hypertension, who account for up to 30% of apparently normotensive apneics. It is notable, however, that in an earlier study, an office BP of 125/83 mm Hg has a 90% positive predictive value for masked hypertension.⁵

Along these lines, application of continuous positive airway pressure (CPAP) has been found to improve arterial elasticity in patients with OSA.⁵ Evidence of its effect on BP levels, though not consistent, seems to favor its use especially regarding alleviation of the disturbed night time patterns, at least in the setting of frank hypertension. Future research may reveal the effect of CPAP on target organ damage in the setting of masked hypertension in OSA patients.

In conclusion, this work suggests that when OSA is present, normal office BP measurements may not reflect the true 24-h hemodynamic load compared to non-apneic individuals, and therefore masked hypertension should always be in mind. Whether increased arterial stiffness is primarily related to apnea severity or rather to the effect of OSA on BP per se in this setting needs further clarification.

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