Treating Apnea May Help BP

By Chris Kaiser, Cardiology Editor, MedPage Today
Published: May 22, 2012

Reviewed by Zalman S. Agus, MD; Emeritus Professor, Perelman School of Medicine at the University of Pennsylvania and Dorothy Caputo, MA, BSN, RN, Nurse Planner

Treating obstructive sleep apnea in patients who have daytime sleepiness as a symptom may have an additional benefit -- a reduction in the risk of hypertension.

A pair of studies published in the May 23/30 issue of the *Journal of the American Medical Association* point to a possible link between a reduction in hypertension and the use of continuous positive airway pressure (CPAP), but only one of the studies -- an observational one -- found a significant reduction in new onset hypertension.

The second study, a randomized controlled trial, reported an observed treatment effect that did not reach statistical significance.

In the first study patients on CPAP therapy had an average 29% decreased risk of new-onset hypertension versus controls with obstructive sleep apnea who were ineligible for CPAP (HR 1.33), refused CPAP (HR 1.96), or were nonadherent to CPAP (HR 1.78), reported José M. Marin, MD, Hospital Universitario Miguel Servet in Zaragoza, Spain, and colleagues.

Marin's study followed 1,889 patients referred to a sleep center in Spain and followed for a median of 12.2 years. All participants were normotensive at baseline.

Video source: JAMA

Action Points

Note that OSA is recognized by multiple hypertension guidelines as a secondary cause of hypertension and treatment of OSA is recommended in patients with refractory hypertension.

Point out that one observational study reported here found a strong dose-response relationship between OSA and incident hypertension and a strong association between adherent CPAP therapy use and lower incidence of hypertension. In a second study, investigators found a treatment effect of CPAP prescription that did not reach statistical significance.

The patients had been referred to the sleep center by their primary care physicians because of complaints such as snoring and daytime sleepiness.

Mild obstructive sleep apnea was defined as an apnea-hypopnea index from 5 to 14.9, moderate was 15 and 29.9, and severe when was more than 30. Those with an apnea-hypopnea index of less than five events per hour served as controls (n=310).

The mean age of patients was 50 and the majority were men.

Marin and colleagues found that in unadjusted and adjusted analyses all untreated obstructive sleep apnea patients had a greater risk for new-onset hypertension compared with controls.

The risk of developing hypertension for those treated with CPAP was similar to that of controls.

All patients who developed hypertension had a significant increase in body mass index during follow-up, researchers reported.

In the second study, Ferran Barbé, MD, of Hospital Universitari Arnau de Vilanova in Lleida, Spain, and colleagues randomized 725 patients with obstructive sleep apnea to CPAP or no CPAP to assess the impact of treatment on the incidence of hypertension and/or the rate of cardiovascular events.

Patients were from 14 teaching hospitals in Spain and were recruited between 2004 and 2005.

The mean age of patients was 51 and the majority were men. The mean body mass index was 31 and mean blood pressure was 130/80 mm Hg. None of the patients had daytime sleepiness.

Cardiovascular events included hospitalizations for unstable angina or arrhythmia, nonfatal stroke, heart failure, nonfatal myocardial infarction, transient ischemic attack, and cardiovascular death.

Barbé and colleagues found no difference in the incidence of new hypertension or the rate of cardiovascular events at a median follow-up of 4 years between the two groups.

The incidence density rate of hypertension or cardiovascular events in the CPAP group was 11.02 per 100 person-years compared with 9.20 in the control group (*P*=0.20).

The investigators found that those who used CPAP less than 4 hours per night had more new-onset hypertension and cardiovascular events than those who used the therapy longer than 4 hours per night (incident density rate 1.13 versus 0.72) compared with controls. The difference was not significant.

However, during the study period, those with worse oxygen saturation who used CPAP for less 4 hours per night had a greater risk of hypertension and cardiovascular events than those who adhered to the therapy for more than 4 hours per night (incident density rate 1.89 versus 0.71) compared with controls.

The authors concluded that the use of CPAP in obstructive sleep apnea patients without daytime sleepiness may not reduce the incidence of hypertension or cardiovascular events. They suggested, however, that a larger study or longer follow-up might reveal some differences as their study may not have had the statistical power to show differences.

Both the observational study and Barbé's randomized controlled trial had a number of limitations.

Marin and colleagues noted their study was limited by its observational design, and they cautioned that the results cannot be interpreted as causal. Also, those who adhered to CPAP had better adherence to their drug therapy, which might suggest they are more health conscious.

Barbé and colleagues cited higher apnea-hypopnea index scores and worse oxygen saturation in the CPAP group at baseline, the potential "white coat" effect of blood pressure measurements at the clinic, and CPAP titration protocols that may have resulted in elevated residual AHI in CPAP treatment.

In an accompanying editorial, Edward M. Weaver, MD, MPH, and Vishesh K. Kapur, MD, MPH, from the University of Washington in Seattle, agreed that taken together the studies do not provide definitive evidence that CPAP therapy may reduce risk.

That said, they did agree that data "generally support a causal link with hypertension. Treatment may not only reduce blood pressure (although modestly on average), but if confirmed by future studies also may prevent hypertension in at-risk patients."

The study by Marin and colleagues was supported in part by the Instituto Carlos III, Ministry of Health in Madrid, Spain, and the Spanish Society of Respiratory Medicine.

Marin and colleagues reported they had no conflicts of interest.

The study by Barbé and colleagues was supported by Instituto de Salud Carlos III, the Spanish Respiratory Society, Resmed, Air Products–Carburos Metalicos, Respironics, and Breas Medical.

Barbé and colleagues reported they had no conflicts of interest.

Editorialist Weaver reported he had no conflicts of interest. Kapur reported having owned stock within the last 3 years in Merck, Johnson & Johnson, and Bristol-Myers Squibb.

From the American Heart Association:

Sleep Apnea and Cardiovascular Disease

Primary source: Journal of the American Medical Association
Source reference:
Barbé F, et al "Effect of continuous positive airway pressure on the incidence of hypertension and cardiovascular events in nonsleepy patients with obstructive sleep apnea: a randomized controlled trial" *JAMA* 2012; 307(20): 2161-2168.

Additional source: Journal of the American Medical Association
Source reference:
Marin JM, et al "Association between treated and untreated obstructive sleep apnea and risk of hypertension" *JAMA* 2012; 307(20): 2169-2176.

Additional source: Journal of the American Medical Association
Source reference:
Kapur VK, Weaver EM "Filling in the pieces of the sleep apnea-hypertension puzzle" *JAMA* 2012; 307(20): 2197-2198.

Add Your Knowledge ™