Papanikos Ioannis, MD,

Otorhinolaryngologist, Patra, Greece

Dr Dimitrios N. Gelis, MD, Otorhinolaryngologist, DDS, PhD, with special interest in Medical Nutrition,

Damaskinou 46, Korinthos 20100, Greece tel. 00302741026631, 00306944280764

www.gelis.gr, www.orlpedia.gr, www.allergopedia.gr, www.gkelanto.gr, www.pharmagel.gr

Obstructive sleep apnea (OSA) is characterized by repetitive nocturnal upper airway obstructions that are associated with sleep disruption and cyclic intermittent hypoxia (CIH) The cyclic oscillations in oxygen saturation are thought to contribute to cardiovascular and other morbidity but animal and patient studies of the pathogenic link between CIH and these diseases have been complicated by species differences and by the effects of confounding factors such as obesity, hypertension, and impaired glucose metabolism[1].

Sleep apnea has been investigated from various aspects for over 32 years since this concept was first proposed. Attention has been focused on the influence of sleep apnea on the circulatory system. It has been reported that sleep apnea is often associated with circulatory disorders and that it is related to hypertension, pulmonary hypertension, right heart failure, arrhythmias, cerebrovascular disease, and nocturnal sudden death [2].

Obstructive sleep apnoea syndrome (OSAS), due to the collapse of the upper airways, is a common but still underestimated condition. The 'dose-response' type relationship between OSAS and hypertension (HT) has now been clearly proven. therefore, blood pressure must be tested in every apneic patient, if necessary by ambulatory blood pressure monitoring. There are multiple mechanisms explaining this relationship, the main one being an increase in sympathetic activity during the apnea episodes. HT associated with OSAS has several characteristics: high prevalence, diastolic and nocturnal predominance, and frequent non-dipper status; and the HTN tends to be resistant to treatment..

OSAS promotes the formation of arterial lesions (parietal thickening of the carotid artery, increased aortic stiffness, and endothelial dysfunction); the more severe the OSAS, the more severe the lesions [3] .

Furthermore, as OSAS is found in the majority of subjects with refractory HT, it should be systematically investigated in this situation. HT associated with OSAS should be tested for by means of a clinical blood pressure (BP) measurement, to which 24-h ambulatory BP monitoring (ABPM) is often added due to the fact that BP anomalies are frequently present at night.

Hypertensive patients with obstructive sleep apnea syndrome (OSAS) constitute a high-risk group for metabolic syndrome (for example, obesity, dyslipidaemia and insulin resistance). OSAS directly induces negative intrathoracic pressure and decreases pulmonary stretch receptor stimulation, chemoreceptor stimulation, hypoxemia, hypercapnia and microarousal. These changes potentiate various risk factors, including the sympathetic nervous system, renin-angiotensin-aldosterone system and inflammation.

Early detection and treatment of OSAS in asymptomatic hypertensive patients is essentially important to prevent hypertensive target organ damage and subsequent cardiovascular events. Continuous positive airway pressure (CPAP) therapy, a first-line treatment in hypertensive patients with moderate to severe OSAS, reduces ambulatory BP level, particularly during the sleep period, and midnight BP surge. However, individual differences in the BP-lowering effect of CPAP have been observed. OSAS hypertensive patients who do not tolerate CPAP remain at a high risk for cardiovascular disease because of negative intrathoracic pressure and need more aggressive antihypertensive treatment to achieve 24-h BP control with nocturnal BP

The reference treatment for OSAS-nasal continuous positive airway pressure (nCPAP)-seems to be able to lower the BP of hypertensive patients, especially if the HT is severe, untreated or refractory. Moreover, the BP response to nCPAP depends on the severity of the OSAS, in particular the scale of the nocturnal desaturations, and on patient tolerance of the treatment. Optimal treatment for HT associated with OSAS has not been evidenced. Antihypertensive drugs do not change the respiratory parameters during OSAS [4]. Following obstructive apneas there is a transient uncoupling of coronary blood flow (CBF) from myocardial work and an increase in coronary vascular resistance (CVR). This disturbed flow-metabolic coupling may lead to nocturnal myocardial ischemia in patients with both OSA and coronary artery disease [5, 6].

References

1. Tamisier R , Gilmartin GS , Launois SH , PÃ©pin JL , Nespoulet H , Thomas RJ , Levy P , Weiss JW . A New Model of Chronic Intermittent Hypoxia in Humans: Effect on ventilation, sleep and blood pressure.
J Appl Physiol.
2009 Feb 19.

2. Bradley TD , Rutherford R , Grossman RF , Lue F , Zamel N , Moldofsky H , Phillipson EA . Role of daytime hypoxemia in the pathogenesis of right heart failure in the obstructive sleep apnea syndrome.
Am Rev Respir Dis.
1985 Jun;131(6):835-9.

3. Baguet JP , Barone-Rochette G , PÃ©pin JL . Hypertension and obstructive sleep apnoea syndrome: current perspectives. J Hum Hypertens.
2009 Jan 8.

4 Kario K . Obstructive sleep apnea syndrome and hypertension: mechanism of the linkage and 24-h blood pressure control. Hypertens Res. 2009 May 22.

5. Hamilton GS , Meredith IT , Walker AM , Solin P . Obstructive sleep apnea leads to transient uncoupling of coronary blood flow and myocardial work in humans.
Sleep.
2009 Feb 1;32(2):263-70.

6. Gelis N.D. Snoring and its treatment, Vellerophontis Ed, Korinthos, Greece, 1997