Obstructive Sleep Apnea Syndrome and its Comorbid Medical Conditions

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Obstructive sleep apnea syndrome (OSAS) is a common global health disorder which carries multiple medical and social impacts. OSAS is characterized by periodic complete or partial upper airway obstruction during sleep, causing intermittent cessations of breathing (apnea) or reductions in airflow (hypopnea) despite ongoing respiratory effort. It is associated with sleep disorder symptoms including loud snoring, disturbed and frequent wake-up during sleep and excessive daytime sleepiness. Apnea is a Greek word means "without breath".

The disorder is associated with hypertension [1], cardiovascular diseases [2], decreased libido [3] and emotional problems [4]. Unsurprisingly, this disorder has been linked to heart failure [5], myocardial infarction [6], chronic obstructive pulmonary disease (COPD) [7], stroke [8] and motor vehicle crashes [9,10].

Systemic hypertension is very common among the OSAS patients. It constitutes about 56% of OSAS patient having hypertension [11]. Cyclic intermittent hypoxia as experienced at night by OSAS patient leads to sympathetic activation [12]. This sustained sympathoexcitation through augmentation of peripheral chemoreflex sensitivity and stimulation on central sympathetic regulator causing increases of heart rate, cardiac output, peripheral vascular resistance and systemic arterial pressure thus increases the blood pressure [13,14].

Lavie et al. [15] monitored 24 hour blood pressure (BP) on 38 OSAS patients and their results revealed that diastolic, systolic and mean BP were significantly related to the severity of sleep apnea syndrome. Few hemodynamic studies on OSAS patients showed the systemic arterial pressure increases and remains at higher level during apnea episode compared to wakefulness [16-18]. The blood pressure can rise by 25% of the baseline during apnea episode and systolic and diastolic pressure can exceed 200 mmHg and 120 mmHg respectively [17]. Studies by Lies et al. [19], Sanner et al. [20] and Tun et al. [21] showed significant improvement in blood pressure in OSAS patient treated by CPAP which proves the causal relationship between systemic hypertension and OSAS.

The clinical spectrum of OSAS related cardiovascular disease comprises of systemic arterial hypertension with prevalence of 40-60%, pulmonary hypertension, 20-30%; coronary artery disease, 20-30%; congestive heart failure, 5-10%; and arrhythmia, 50-60% [22]. Sajkov et al. [23] in a pulmonary hemodynamic study on 32 patients with OSA found that pulmonary hypertension is associated with small airways closure during tidal breathing and increased pulmonary press or responses to hypoxia and during increased pulmonary blood flow. These changes are consistent with remodeling of the pulmonary vascular bed in OSAS patients with pulmonary hypertension and unrelated to severity of sleep-disordered breathing.

OSAS is commonly found in patients with coronary heart disease and these patients must be classified as a high risk group because of apnea-associated silent myocardial ischemia and electric instability of the myocardium [24]. The risk of myocardial infarction is increased 20-fold in untreated OSA [25].

The occurrence of arrhythmia in patients with OSAS is closely related to the apnea and hyperventilation events and depends on the sympathovagal balance [24]. Sinus arrhythmia occurs with decreased heart rate during apnea and increased heart rate when breathing resumes. Marked sinus bradycardia is due to hypoxic induced vagal activity. Severe oxygen desaturation can lead to conduction defect such as premature ventricular contraction, second degree heart block and ventricular tachyarrhythmia [26].

The association between OSAS and stroke are much debated, but increasing evidence demonstrates that the OSAS is an independent risk factor for ischemic stroke especially in young patient [27]. Many studies have showed 70 to 95% of acute stroke patients were found to have OSAS [28,29].

Multiple medical conditions are linked to the mechanisms of stroke in OSAS such as arterial hypertension, cardiac arrhythmia, increased atherogenesis, coagulation disorders, and cerebral haemodynamic changes [28]. Presence of OSAS in stroke patients could lead to a poor outcome and increased long-term stroke mortality. Therefore, OSAS should be systematically screened at the moment it is clinically suspected in patients with acute stroke.

Studies worldwide revealed the presence of OSA has been found in 5 to 63% of patients with epilepsy. Patients with neurological disorders seem to have a greater prevalence for sleep disturbance than normal subjects and epilepsy also seemed to have similar preponderance. Miller et al. [30] showed that more than two thirds of patients with epilepsy seen at a university center had complaints regarding sleep. Polysomnographic investigation by Malow et al. [31] showed that nearly one third of patients with medically refractory epilepsy had a respiratory disturbance index of more than 5. Vaughn et al. [32] studied a cohort of 25 patients with intractable epilepsy and found 36% had a respiratory disturbance index of more than 10. Frequency rates were higher in patients with refractory epilepsy or epilepsy patients referred to a sleep center [33,34] and lower [35,36] in unselected populations.

References

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