

Chapter: 18

Cardiovascular Complications of Obstructive Sleep Apnea

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The prevalence of Obstructive sleep apnea (OSA) in Wisconsin sleep cohort study is estimated to be 4 % of middle-aged men and 2 % of women in the Western populations. The composite of Wisconsin, Pennsylvania, and Spanish epidemiologic studies revealed the prevalence of mild OSA in one in five adults and moderate OSA in at least one in 15. In older adults three times higher prevalence is estimated than in middle-aged people.

According to some estimates 70 million people suffer from sleep disorders. Up to 50% of these are related to sleep related breathing disorder. It costs millions of dollars in health care every year. At least 2300 sleep studies/100,000 people/year needed to adequately address the demand for diagnosis and treatment.

While Sleep disorders are treatable and sometimes curable, there are serious health consequences if these are left untreated. Significant increase in health care utilization, including both hospitals and providers has been well documented due to these complications. Patients with OSA use health-care resources at higher rates than control subjects for many years prior to diagnosis. Of all co-morbid diagnoses, significant increase in utilization was found for cardiovascular disease, especially hypertension in the OSA patients.

Acute hemodynamic effects of OSA are the result of the occlusion of the upper airway leading to the forceful inspiratory effort against a closed airway resulting increased blood pressure, reflex bradycardia and rebound tachycardia. The hypoxemia, arousal and apnea termination then follow. There is significant increase in myocardial oxygen demand at a time when oxygen saturation is at its lowest. Further, stress release of catecholamines causes increase in the sympathetic nervous system activity adversely affecting the organ functioning.

Data has been evolving over the last decade regarding the consequences of sleep disordered breathing. According to the American heart association 2002 report, cardiovascular disease affects 70 million Americans or 23% of the USA population. Hypertension is the most common association with 65 million affected people followed by Coronary Artery Disease in 13 million and congestive heart failure in 5 million people. The estimated cost of treating Cardio-vascular disorders in 2005 was \$393.5 billion.

Wisconsin Sleep Cohort Study, a prospective trial included 1189 Participants. This study showed a dose-response association between OSA and hypertension. Untreated OSA is associated with a higher prevalence of chronic hypertension over 40% and Idiopathic hypertension is associated with a 30% incidence of OSAS. Blood pressure surges at the

end of the apnea periods. The apneas are associated with arousals, hypoxemia, sympathetic activation and eventually sustained hypertension. Changes in blood pressure with effective Continuous Positive Airway Pressure (CPAP) therapy in patients with OSA suggest improvement with treatment. The therapeutic response to CPAP supports the concept of a casual association between OSA and chronic hypertension. Forty percent patients with untreated sleep disorders breathing have hypertension. On the other hand 80% of drug resistant hypertension patients have sleep disordered breathing.

There is a high prevalence of sleep related breathing disorders (SRBD) in moderate-to-severe congestive heart failure (CHF). Stable CHF with ejection fraction (EF) less than 45% have a 45% incidence of moderate OSA. In symptomatic CHF patients the sleep related breathing disorder is present in 75% of men and 47% of women. In patients presenting with pulmonary edema the incidence of SRBD is 82%. SRBD with CHF signifies a worse prognosis. Treatment of OSA in CHF improves symptoms, cardiac function, the quality of life and survival.

Lanfranchi reported the mortality rates of 12% in the first year and 25% in the second year in CHF patients with OSA. Non-survivors were in a higher New York Heart association (NYHA) class with lower EF, larger atrial and left ventricle size. Non-survivors had a greater percentage of the night in periodic breathing and higher AHI. Multivariate analysis revealed AHI followed by left atrial size as the independent predictors of subsequent cardiac death. No difference was found in the time spent at less than 90% and 85% oxygen saturation levels. Apnea-hypopnea index was the main determinant of survival.

Peker et al prospectively investigated in 97 patients admitted to the ICU. All patients had lower NYHA functional class I to II indicating stable cardiac function. Overnight sleep study was then performed at 4 to 21 months after discharge. OSA was treated with CPAP in 3 out of 19 patients and patients were followed for 60 months. If the study subject died then the death was attributed to a cardiovascular origin in the case of documentation of significant arrhythmias, cardiac arrest, cardiac failure, myocardial infarction, or stroke. The death hazard was calculated as a function of AHI, current age, and time elapsed after the intensive care episode for coronary artery disease. The increase in death rate was noted as function of higher AHI.

Milleron et al in 2004 reported improved risk of coronary artery disease event with CPAP treatment in 54 patients with both OSA and coronary artery disease. In this study the end points included were cardiovascular death, acute coronary syndrome, congestive heart failure, hospitalization and coronary artery bypass grafting. In the CPAP treated group there was an 81% risk reduction noted in the event-free survival.

According to American academy of sleep medicine practice parameters, patients with coronary artery disease should be evaluated for symptoms and signs of sleep apnea. If there is suspicion of sleep apnea, the patients should undergo a sleep study.

The incidence of atrial fibrillation (AF) with OSA is high at 49% compared to 32% without OSA. Atrial fibrillation is difficult to manage in patients with SRBD. Higher percentage of recurrence of AF at 12 months is documented by Kanagala et al comparing treated OSA patients with untreated and noncompliant patients. In a small retrospective review majority of the patients referred for pacemaker evaluation for brady-arrhythmia, had documentation of OSA. A manual review of death certificates from 1987 to 2003 of 112 Minnesota residents who had undergone polysomnography and had died of sudden death revealed increased risk of sudden death in OSA. This study concluded the people with OSA have a shift in the typical pattern of sudden death for cardiac causes to the sleep hours and it appears the risk of sudden death is increased in OSA.

American academy of sleep medicine recommends that patients referred for evaluation of significant tachyarrhythmias or bradyarrhythmias should be questioned about symptoms of sleep apnea. A sleep study is indicated if questioning results in a reasonable suspicion that OSA or CSA are present.

Recommended reading:

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