Original Research

Comparison of Abnormal Heart Rate and Nocturnal Arrhythmia in Patients with Obstructive Sleep Apnea and Normal Subjects

Besharat Rahimi, Anahita Tavoosi, Toktam Sadeghi, Romina Arjmand, Maryam Edalatifard*

Advanced Thoracic Research Center, Tehran University of Medical Sciences, Tehran, Iran

Received: 06 Dec. 2018 Accepted: 30 Aug. 2019

Abstract

Background and Objective: Obstructive sleep apnea (OSA) is a common sleep-related breathing disorder and an important risk factor for cardiovascular diseases (CVDs). This study aimed to compare abnormal heart rate and arrhythmias among patients with OSA and normal subjects.

Materials and Methods: This case-control study was conducted on patients with OSA who were attending Imam Khomeini Hospital Sleep Lab, Tehran, Iran, to undergo a full-night polysomnography (PSG). Apnea-Hypopnea Index (AHI) was measured for study participants. Those with AHI < 5/h were considered as control (normal) and those with AHI > 15/h were considered as case (OSA) group. We compared abnormal heart rate and nocturnal arrhythmia during PSG between the two study groups.

Results: A total of 60 patients (30 cases and 30 controls) were included in this study. Abnormal heart rate (bradycardia and tachycardia) and nocturnal arrhythmia were compared between the two study groups. Twenty-eight percent of patients with moderate to severe OSA developed arrhythmia including sinus arrest, premature atrial contraction (PAC), atrial fibrillation (AF), and bigeminy premature ventricular contraction (PVC). Multiple PVC episodes were observed in 6.7% of subjects without OSA. Nocturnal arrhythmia was more common in cases; however, there was no significant difference. Mean heart rate in cases was 45 beat/minute in comparison with 68/minute in controls (P = 0.0001).

Conclusion: Different arrhythmias are observed in patients with OSA due to autonomic disorders. Bradycardia was more prevalent among patients with OSA. Regarding the association between OSA and nocturnal arrhythmias, treatment of OSA is important to prevent cardiac mortality.

Keywords: Thermography; Thyroid gland; Sleep

Citation: Rahimi B, Tavoosi A, Sadeghi T, Arjmand R, Edalatifard M. **Comparison of Abnormal Heart Rate and Nocturnal Arrhythmia in Patients with Obstructive Sleep Apnea and Normal Subjects.** J Sleep Sci 2019; 4(3-4): 59-62.

Introduction

Sleep breathing disorders have different types and causes which among them obstructive sleep apneas (OSAs) are more common (1).

OSA is a result of repeated respiratory arrests usually due to obesity or craniopharyngeal abnormalities. It usually leads to repeated nocturnal hypoxemia, autonomic and sympathetic stimulations, increased night-time blood pressure, somnolence and diurnal inattention, and decreased quality of life (1).

**** Corresponding author**: M. Edalatifard, Advanced Thoracic Research Center, Tehran University of Medical Sciences, Tehran, Iran*
Tel: +98 21 61192646, Fax: +98 21 66581594
Email: maryam_edalatifard@yahoo.com

During normal sleep, benign arrhythmias may develop such as sinus arrhythmia due to effect of respiration on heart rate (2), physiological bradyarrhythmia resulting from increased vagus activity in non-rapid eye movement (NREM) sleep (3), premature ventricular contraction (PVC) in normal sleep (4), and sympathetic predominance at the end of sleep.

During sleep apnea, the hypoxemia activates cardiac parasympathetic activity leading to bradyarrhythmia and likely increased peripheral sympathetic activity, peripheral vasoconstriction, and vascular resistance (5). After termination of apnea event, the sympathetic activity may increase with a rising blood pressure and heart rate (6). OSA prob-

ably increases chronic sympathetic activity during sleep that may be extended to wake state too. Atrial fibrillation (AF) is the most common type of arrhythmia in OSA. In AF cases, due to OSA, lack of post cardioversion treatment would result in 82% one-year recurrence rate (7). The other arrhythmias include sinus arrest, ventricular tachycardia (VT), and second-degree atrioventricular (AV) block. Limited data are available at the current time regarding frequency of nocturnal arrhythmias among patients with OSA in the region. Thus, we conducted this study to compare abnormal heart rate among patients with OSA and normal subjects.

Materials and Methods

This case-control study was done among 60 patients without cardiac disease in two groups of 30 participants categorized in terms of OSA at Imam Khomeini Hospital Sleep Lab, Tehran, Iran.

Polysomnography (PSG) was performed for all patients with SOMNOscreen Plus (SOMNOmedics Company, Germany) for confirmation of OSA and recording the physiologic signals such as electroencephalogram (EEG): F3/A2, F4/A1, C3/A2, C4/A1, O1/A2, O2/A1, electrooculogram (EOG) lead II, chin electromyogram (EMG), airflow, oxygen saturation, respiratory effort, and electrocardiogram (ECG). Data (age, sex, abnormal heart rate, arrhythmia) were recorded in a checklist. Cardiac and echocardiographic assessments of participants were all normal.

Analysis and interpretation of PSG results including respiratory and cardiac results was performed according to American Academy of Sleep Medicine (AASM) manual by trained sleep medicine specialist. Patients with Apnea-Hypopnea Index (AHI) less than 5 per hour were considered normal and included as study controls (n = 30) and those with AHI more than 15 per hour were selected as cases (n = 30).

Results

The mean age of participants was 49.4 years in control group and 55.4 years in moderate to severe OSA (AHI > 15/h) group, respectively. In control group, 43.3% were men and 59.7% were women. In case group, 35.7% were women and 64.3% were men.

Among 30 patients with moderate to severe OSA, 28.6% had arrhythmia including AF and bigeminy PVC as well as sinus arrest. The majority of arrhythmias were due to apnea and elongated hypoxemia.

In control group, 6.7% of arrhythmia cases were PVC. Despite higher rate of arrhythmia in cases compared to controls, there was no significant difference (P = 0.070), but the trend was clinically significant (Figure 1).

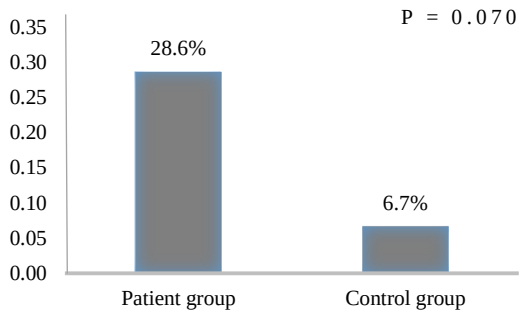

Figure 1. Arrhythmia during sleep between case and control groups

Hypertension (HTN) was present in 50% and 7% of cases and controls, respectively; while, the difference was statistically significant. Mean body mass index (BMI) in severe OSA, moderate OSA, and controls was 40.40, 32.40, and 28.45 kg/m^2, respectively, showing a significant difference (P = 0.003) (Figure 2).

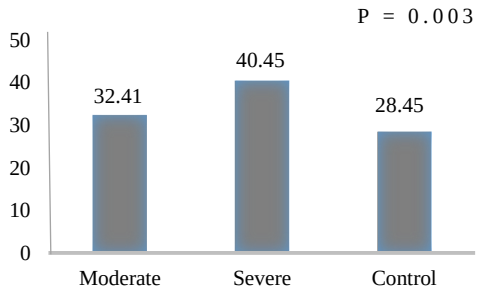

Figure 2. Mean body mass index (BMI) between control and patients with moderate and severe obstructive sleep apnea (OSA)

Snoring index calculated by PSG device was 21 per hour and 427 per hour in controls and cases, respectively, with a significant difference (P = 0.004). Mean of minimum and maximum heart rate was assessed for bradycardia and tachycardia. The mean of minimum heart rate was 45 per minute and 68 per minute in cases and controls, respectively, with a significant difference (P = 0.0001). For tachycardia assessment, the

mean of maximal heart rate in cases was 107 per minute, whereas it was 125 per minute in controls with a significant difference (P = 0.003).

Discussion

OSA is the most common type of sleep-related breathing disorders characterized by complete or partial episodes of obstruction in upper airways leading to respiratory arrest or decreased frequency of breathing. The main risk factors for sleep-disordered breathing (SDB) include obesity, male gender, older ages, and craniopharyngeal abnormalities (8). OSA is seen in 4% and 2% of men and women aging between 30 and 65 years, respectively (9). OSA diagnosis is established when the apnea and hypopnea are more than 5 episodes per hour and diurnal somnolence is present or AHI is more than 15 per hour with or without diurnal symptoms. Association of OSA and cardiac arrhythmia is established before (10). Recording of ECG during PSG is useful to assess cardiac arrhythmias, especially the heart blocks and sinus pauses (3).

Exact prevalence rate of cardiac arrhythmias is unknown; however, it is more common in patients with OSA. Furthermore, related mechanism is not fully understood and hypoxemia, arousal, and autonomic dysregulation are known contributing factors for arrhythmia in OSA. VT, sinus arrest, second-degree AV block, and AF are the most common types of arrhythmia in patients with OSA (11).

The prevalence of AF is 0.4% and 6% in general population and patients with OSA, respectively (12). Severe hypoxemia in rapid eye movement (REM) sleep is related to cardiac arrhythmia (13). Desaturation due to OSA would result in increased CO_2 and dysfunction of baroreceptors and chemoreflexes, leading to autonomic disorders. For instance, increased sympathetic activity would result in electrical atrial remodeling and supraventricular (SV) arrhythmia. The respiratory arousals due to stimulation of action potential would develop arrhythmia (14).

Moreover, respiratory attempts against closed glottis during apnea would result in shift in transmural pressure and tension changes in the heart leading to bradyarrhythmia. Bradyarrhythmia is reported in OSA that is usually during apnea and hypopnea, but not the hyperventilation. Additionally, the severity of apnea and bradyarrhythmia are related (15). Bradyarrhythmia is related to hypoxemia extent. Closed glottis in OSA results in vagus stimulation leading to bradyarrhythmia. Brady-arrhythmias such as AV block and asystole are seen even without overt cardiac disorder in cases with OSA (16). This disorder is usually seen in REM sleep (17). Brady-arrhythmia is accompanied with high BMI, severe OSA, and desaturation level, as seen in the current study. Unfortunately, the cardiac arrhythmia in patients with OSA is accompanied with increased mortality rate. OSA treatment with continuous positive airway pressure (CPAP) may result in decreased cardiac arrhythmia, especially brady-arrhythmia (17).

The results of the current study demonstrated that cardiac arrhythmia in patients despite lack of a significant difference was higher and in PSG, different arrhythmias such as AF, bigeminy PVC, sinus arrest, and premature atrial contraction (PAC), especially after hypoxemia, were seen.

Moreover, mean minimal heart rate in cases and controls was significantly differed. It demonstrated that OSA was related to cardiac arrhythmia and also hypoxemia and increased vagus activity would result in higher brady-arrhythmia in patients with OSA.

Conclusion

In patients with cardiac arrhythmia especially without overt cardiac disease, the presence of OSA should be assessed and successful treatment would result in decreased arrhythmia and mortality in these patients.

Conflict of Interests

Authors have no conflict of interests.

Acknowledgments

Authors would like to thank Ali Naghipour for his nice collaboration during this study.

References

1. Kasper DL, Fauci AS, Hauser SL, et al. Harrison's principles of internal medicine. 19th. New York, NY: McGraw-Hill Education; 2016. p. 1723-6.
2. Verrier RL, Josephson ME. Impact of sleep on arrhythmogenesis. Circ Arrhythm Electrophysiol 2009; 2: 450-9.
3. Guilleminault C, Pool P, Motta J, et al. Sinus arrest during REM sleep in young adults. N Engl J Med 1984; 311: 1006-10.
4. Kong TQ, Goldberger JJ, Parker M, et al. Circadian

variation in human ventricular refractoriness. Circulation 1995; 92: 1507-16.
5. Svatikova A, Wolk R, Shamsuzzaman AS, et al. Serum amyloid A in obstructive sleep apnea. Circulation 2003; 108: 1451-4.
6. Sabharwal R, Zhang Z, Lu Y, et al. Receptor activity-modifying protein 1 increases baroreflex sensitivity and attenuates Angiotensin-induced hypertension. Hypertension 2010; 55: 627-35.
7. Kanagala R, Murali NS, Friedman PA, et al. Obstructive sleep apnea and the recurrence of atrial fibrillation. Circulation 2003; 107: 2589-94.
8. Parati G, Lombardi C, Narkiewicz K. Sleep apnea: Epidemiology, pathophysiology, and relation to cardiovascular risk. Am J Physiol Regul Integr Comp Physiol 2007; 293: R1671-R1683.
9. Young T, Palta M, Dempsey J, et al. The occurrence of sleep-disordered breathing among middle-aged adults. N Engl J Med 1993; 328: 1230-5.
10. Wolk R, Kara T, Somers VK. Sleep-disordered breathing and cardiovascular disease. Circulation 2003; 108: 9-12.
11. Hoffstein V, Mateika S. Cardiac arrhythmias, snoring, and sleep apnea. Chest 1994; 106: 466-71.
12. Shepard JW, Garrison MW, Grither DA, et al. Relationship of ventricular ectopy to oxyhemoglobin desaturation in patients with obstructive sleep apnea. Chest 1985; 88: 335-40.
13. Wit AL, Rosen AR. After depolarization triggered activity in: Fozzard HA, Haber E, Jenning RB, et al, editors. The heart and cardiovascular system. New York, NY: Raven Press; 1986. p. 1449-90.
14. Mooe T, Gullsby S, Rabben T, et al. Sleep-disordered breathing: a novel predictor of atrial fibrillation after coronary artery bypass surgery. Coron Artery Dis 1996; 7: 475-8.
15. Guilleminault C, Connolly SJ, Winkle RA. Cardiac arrhythmia and conduction disturbances during sleep in 400 patients with sleep apnea syndrome. Am J Cardiol 1983; 52: 490-4.
16. Koehler U, Fus E, Grimm W, et al. Heart block in patients with obstructive sleep apnoea: pathogenetic factors and effects of treatment. Eur Respir J 1998; 11: 434-9.
17. Grimm W, Koehler U, Fus E, et al. Outcome of patients with sleep apnea-associated severe bradyarrhythmias after continuous positive airway pressure therapy. Am J Cardiol 2000; 86: 688-92, A9.