ARAŞTIRMA MAKALESİ / *RESEARCH ARTICLE*

Evaluation of Cardiac Findings Before Laboratory-Based Polysomnography

Laboratuvar Temelli Polisomnografi Öncesi Kardiyak Bulguların Değerlendirilmesi

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ABSTRACT

Aim: *This study aimed to determine the demographic characteristics of the patients who applied to the Cardiology Out-patient Clinic before laboratory-based polysomnography was performed.*

Material and Method: *123 adult patients undergoing laboratory-based polysomnography were included in this retrospective study. The demographic features of the patients were recorded. Routine laboratory tests, electrocardiography (ECG), transthoracic echocardiography (TTE), 24-hour ambulatory blood pressure monitoring, cardiovascular stress test, and laboratory-based polysomnography were performed in the same hospital. The apnea-hypopnea index (AHI) was used to diagnose obstructive sleep apnea syndrome (OSAS).*

Results: *In this study, we found the following patient findings. The mean age of the patients was 49±1.61 years. The majority of the patients were in the middle age group. Forty-two (34.10%) patients were female and 81 (65.90%) were male. Normal AHI value was detected in 5 (4.2%) patients. Mild OSAS was detected in 30 (24.2%) of the patients, moderate OSAS in 24 (18.9%), and severe OSAS in 64 (51.6%). Continuous positive airway pressure (CPAP) device report was given to 67 (54.7%) of the patients. The most accompanying comorbidity of the patients was hypertension (HT). There was a weak positive correlation between AHI and HT; r=0.280, N=95, and the relationship was statistically significant (p=0.006). Nine (7.30%) of the patients were newly diagnosed with hypertension. Invasive coronary angiography was recommended for two (1.6%) patients with positive test results, and coronary computed tomographic angiography was recommended for four (3.2%) patients. Myocardial perfusion scintigraphy was recommended to nine (7.3%) patients who could not perform cardiovascular stress tests.*

Conclusion: *Cardiac evaluation should be performed before laboratory-based polysomnography is performed. Electrocardiography and TTE recording should be performed on the patients, and 24-hour ABPM should be inserted. Additional tests should be performed to investigate ischemia in patients with myocardial ischemia findings.*

Keywords: *obstructive sleep apnea syndrome; polysomnography; cardiology*

ÖZET

Amaç: *Bu çalışmanın amacı, laboratuvar temelli polisomnografi yapılmadan önce Kardiyoloji Polikliniğine başvuran hastaların demografik özelliklerini belirlemekti.*

Materyal ve Metot: *Bu retrospektif çalışmaya laboratuvar temelli polisomnografi yapılan 123 yetişkin hasta dâhil edildi. Hastaların demografik özellikleri kaydedildi. Rutin laboratuvar testleri, elektrokardiyografi (EKG), transtorasik ekokardiyografi (TTE), 24 saatlik ambulatuvar kan basıncı monitörizasyonu (AKBM), kardiyovasküler stres testi ve laboratuvar bazlı polisomnografi aynı hastanede yapıldı. Apne-hipopne endeksi (AHİ), obstrüktif uyku apne sendromunu (OUAS) teşhis etmek için kullanıldı.*

Bulgular: *Bu çalışmada hastalarda aşağıdaki bulguları bulduk. Hastaların yaş ortalaması 49±1,61 yıl idi. Hastaların 42'si (%34,1) kadın, 81'i (%65,9) erkekti. Hastaların beşinde (%4,2) normal AHİ saptandı. Hastaların 30'unda (%24,2) hafif OUAS, 24'ünde (%18,9) orta OUAS ve 64'ünde (%51,6) ağır OUAS saptandı. Hastaların 67'sine (%54,7) sürekli pozitif hava yolu basıncı (CPAP) cihazı raporu verildi. Hastalara en fazla eşlik eden komorbidite hipertansiyon idi. Apne-hipopne endeksi ile HT arasında zayıf pozitif korelasyon vardı; r=0,280, N=95, bu ilişki istatistiksel olarak anlamlıydı (p=0,006). Dokuz (%7,3) hastaya ambulatuvar kan basıncı takibi (AKBM) yapıldı ve yeni hipertansiyon tanısı konuldu. Test sonucu pozitif olan iki (%1,6) hastaya invaziv koroner anjiyografi, dört (%3,2) hastaya koroner bilgisayarlı tomografik anjiyografi önerildi. Kardiyovasküler stres testi yapamayan dokuz (%7,3) hastaya miyokardiyal perfüzyon sintigrafisi önerildi.*

Sonuç: *Laboratuvar tabanlı polisomnografi yapılmadan önce kardiyak değerlendirme yapılmalıdır. Hastalara EKG, TTE ve 24 saatlik AKBM yapılmalıdır. Miyokardiyal iskemi bulguları olan hastalarda iskemiyi araştırmak için ek testler yapılmalıdır.*

Anahtar kelimeler: *obstrüktif uyku apne sendromu; polisomnografi; kardiyoloji*

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Introduction

Obstructive sleep apnea syndrome (OSAS) is a common disorder, and it is a cardiovascular disease risk factor. Snoring, apnea, frequent awakening at night, and constant drowsiness during the day occur in patients with OSAS. Recurrent apneas and hypopneas during sleep are accompanied by hypoxia, increased sympathetic activity, and frequent awakenings. Some studies showed that OSAS is associated with an increased risk of cardiovascular morbidity and mortality. Obstructive sleep apnea syndrome has clearly been shown to be an independent risk factor for the development of hypertension and has also been implicated in the pathogenesis of pulmonary hypertension, congestive heart failure, cardiac arrhythmias, stroke, and atherosclerosis. Continuous positive airway pressure (CPAP) therapy was effective in treating OSAS[1]. Polysomnography is performed to diagnose OSAS. We have done laboratory-based polysomnography in our hospital. Before laboratory-based polysomnography is performed, patients are evaluated in Neurology, Otolaryngology, and Cardiology outpatient clinics. This study aimed to determine the demographic characteristics of patients who applied to the Cardiology Out-patient Clinic before laboratory-based polysomnography was performed.

Materials and Methods

Study design and setting

This study was an observational-retrospective study. One hundred and twenty-three adult patients undergoing laboratory-based polysomnography and cardiac evaluation in the Cardiology Out-patient Clinic before laboratory-based polysomnography were included in this study. Patients who applied to our clinic between October 15, 2021, and October 15, 2022, were included in the study. Past medical histories, examination notes, and laboratory results were obtained from our hospital's electronic records. Demographic features of the patient's age, gender, chronic diseases [diabetes mellitus (DM), hypertension (HT), hyperlipidemia (HL), documented coronary artery disease (CAD), chronic obstructive pulmonary disease (COPD), stroke history, impaired fasting glucose, thyroid gland disease, anemia, polycythemia, splenomegaly, cancer ratio were recorded. We recorded the laboratory data from electronic medical records using a standardized data collection form. The patients admitted to the sleep unit had the following complaints: Snoring (100%), awakening with the feeling of suffocation (76.4%), witnessed apnea (66.6%), and excessive daytime sleepiness (66.6%). Laboratory findings, i. e., glucose (normal range: 74–106 mg/dL), hemoglobin (normal range: 12.6–17.4 g/dL), white blood cells (WBC) (normal range: 4.23–9.07 $10^3/\mu L$), platelet (normal range: 150–360 $10^3/\mu L$), C reactive protein (CRP) (normal range: 0–5 mg/L), creatinine (normal range: 0.67–1.17 mg/dL), alanine aminotransferase (ALT) (normal range: 0–50 U/L), thyroid stimulating hormone (normal range: 0.35–4.94 mIU/L), thyroxine (normal range: 0.70–1.48 ng/dL), low-density lipoprotein (normal range: 0–130 mg/dL), triglyceride (normal range: 0–150 mg/dL were recorded on admission, and they were evaluated as normal or abnormal according to the normal range of the laboratory assays. Anaemia was defined as a hemoglobin (Hb) level of <12.0 g/dL in women and <13.0 g/dL in men. If the plasma fasting glucose was 100–126 mg/dL, it was defined as impaired plasma fasting glucose.

After taking the anamnesis of the patients, electrocardiography (ECG) was taken, and transthoracic echocardiography (TTE) was performed for all patients. Cardiology outpatient clinic nurses performed ECG. Normal sinus rhythm and atrial fibrillation were recorded on the ECG of the patients. Transthoracic echocardiography was conducted by the cardiologists at the Cardiology Out-patient Clinic. Left ventricular ejection fraction (LVEF) was measured. Left ventricular ejection fraction below 50% was defined as systolic heart failure. A 24-hour ambulatory blood pressure monitoring (ABPM) was inserted in patients who did not have hypertension before but whose blood pressure increased occasionally. The cardiovascular stress test was performed on patients with chest pain, shortness of breath, and effort dyspnea. Invasive coronary angiography or coronary computed tomography angiography was recommended for patients who were found to have signs of ischemia as a result of the test. Myocardial perfusion scintigraphy was recommended for patients who could not perform cardiovascular stress testing.

Polysomnography was performed in our hospital's sleep laboratory. The apnea-hypopnea index (AHI) was used to diagnose OSAS. The AHI value is obtained by dividing the sum of the apnea and hypopnea numbers by the individual's sleep time. Hourly AHI values were used. Apnea-hypopnea index values were categorized as Normal: AHI <5, mild sleep apnea: 5 ≤ AHI <15,

moderate sleep apnea: 15 ≤ AHI <30, and severe sleep apnea: AHI ≥30.

Inclusion criteria

- Be over 18 years old
- Admission to the hospital with complaints of snoring, awakening with the feeling of suffocation, witnessed apnea and excessive daytime sleepiness
- Admitted to the Cardiology Out-patient Clinic for cardiac control

Exclusion criteria

- Be under the age of 18
- Patients whose laboratory results could not be reached

Data Analysis

IBM Statistical Package for Social Sciences (SPSS) software for Windows release 25.0.0 (IBM, Chicago, IL) was used for statistical analysis. The Kolmogorov-Smirnov test was used to determine the normality of the variables. Continuous variables were reported as mean ± standard deviation (SD) or median (IQR). Categorical variables were reported as the number (percentage) of participants. A p-value of less than 0.05 was considered statistically significant. P values were two-tailed. Mann-Whitney U test was used to compare medians of two independent groups. A Pearson correlation analysis was used to measure the strength of the linear relationship between two variables.

Ethical Consideration

The study was ethically conducted by the Declaration of Helsinki. Approval was obtained from the local Clinical Research Ethics Committee for the study (approval number: 2022–51).

Results

One hundred and twenty-three consecutive patients were included in the study. The patients were over 18, undergoing polysomnography in our hospital sleep laboratory, and had cardiac evaluations in the Cardiology Out-patient Clinic before polysomnography. The mean age of the patients was 49±1.61 years. The majority of the patients were in the middle age group. Forty-two (34.1%) patients were female and 81 (65.9%) were male. The accompanying comorbidities of the patients were as follows: HT 45 (36.6%), impaired fasting plasma glucose 31 (25.2%), DM 27 (22.0), HL 23 (18.7%), CAD 12 (9.8%), COPD 9 (7.3%), smoker 14 (11.4%), stroke history 3 (2.4%), metallic valve prosthesis 1 (0.8%), hypothyroidism 3 (2.4%), hyperthyroidism 3 (2.4%), anemia 5 (4.1%), polycythemia 1 (0.8%), splenomegaly 1 (0.8%), pancreatic cancer 1 (0.8%), endometrial cancer 1 (0.8%), breast cancer 1 (0.8%). The basic clinical characteristics of the patients, including age, gender, and comorbidities, are listed in Table 1.

Cardiac findings of the patients were as follows: Atrial fibrillation 1 (0.8 %), right bundle branch block 2 (1.6%), metallic valve prosthesis 1 (0.8 %), patent foramen ovale (pfo) 1 (0.8%), mild mitral regurgitation 10 (8.1%), mild aortic regurgitation 6 (4.9%), mild tricuspid regurgitation 5 (4.1%), bicuspid aorta 2 (1.6 %), aortic aneurysm 5 (4.1%), mild left ventricular hypertrophy 10 (8.1%), left ventricular diastolic dysfunction 10 (8.1%), left ventricular systolic dysfunction 1 (0.8%) and LVEF was median 60% (32–65). Cardiac findings of patients on admission are shown in Table 2.

Table 1. *The basic clinical characteristics of the patients*

Total patients number	123
Gender (female/male) (n) (%)	42/81 (34.1/65.9)
Age (years) mean ± SD	49±1.61
Hypertension (n) (%)	45 (36.6)
Diabetes mellitus (n) (%)	27 (22.0)
Hyperlipidemia (n) (%)	23 (18.7)
Coronary artery disease (n) (%)	12 (9.8)
Impaired fasting glucose (n) (%)	31 (25.2)
Chronic Obstructive Pulmonary Disease (n) (%)	9 (7.3)
Metallic valve prosthesis (n) (%)	1 (0.8)
Stroke history (n) (%)	3 (2.4)
Smoker (n) (%)	14 (11.4)
Hypothyroidism (n) (%)	3 (2.4)
Hyperthyroidism (n) (%)	3 (2.4)
Anemia (n) (%)	5 (4.1)
Polycythemia (n) (%)	1 (0.8)
Splenomegaly (n) (%)	1 (0.8)
Pancreatic cancer (n) (%)	1 (0.8)
Endometrial cancer (n) (%)	1 (0.8)
Breast cancer (n) (%)	1 (0.8)

n: number; SD: standard deviation

Table 2. *Cardiac findings of the patients*

Atrial fibrillation (n) (%)	1 (0.8)
Right bundle branch block (n) (%)	2 (1.6)
Patent foramen ovale (n) (%)	1 (0.8)
Mild mitral regurgitation (n) (%)	10 (8.1)
Mild aortic regurgitation (n) (%)	6 (4.9)
Mild tricuspid regurgitation (n) (%)	5 (4.1)
Aortic aneurysm (n) (%)	5 (4.1)
Mild left ventricular hypertrophy (n) (%)	10 (8.1)
Left ventricular diastolic dysfunction (n) (%)	10 (8.1)
Bicuspid aorta	2 (1.6)
Left ventricular systolic dysfunction (n) (%)	1 (0.8)
Left atrial dilatation (n) (%)	5 (4.1)
LVEF % median (IQR)	60 (32–65)

n: number; IQR: Inter Quantile Range; LVEF: Left ventricular ejection fraction

Table 3. *Laboratory findings of patients*

Glucose (mg/dl) median (IQR)	104.00 (79.00–301.00)	74–106 mg/dl
Creatinine (mg/dl) median (IQR)	0.79 (0.44–1.70)	0.5–0.9 mg/dL
Glomerular filtration rate (mL/min/1.73 m2) median (IQR)	102 (42–128)	-
Alanine aminotransferase (U/L) mean ± SD	29.46±12.58	0–33 U/L
CRP (mg/L) median (IQR)	2.60 (0.14–39.30)	0–5 mg/L
Thyroid stimulating hormone (mIU/L) median (IQR)	1.75±1.19	0.35–4.94 mIU/L
White blood count (10^3/mL) mean ± SD	7.91±2.04	3.98–10.04 10^3/mL
Hemoglobin (g/dL) mean ± SD	14.18±1.58	11.7–16.0 g/ dL
Platelet count (10^3/µL) mean ± SD	255.46±63.26	150–360 10^3/µL
Low density lipoprotein mg/dL mean ± SD	117.60±36.85	0–130 mg/dL
Triglyceride mg/dL mean ± SD	159.06±69.50	0–150 mg/dL

n: number; IQR: Inter Quantile Range; SD: standard deviation; mg/dl: milligram/deciliter; mmol/L: millimole/liter; mIU/mL: micro international unit/milliliter; mg/mL: microgram/ milliliter; mL: microliter; mg/L: milligram/liter; pg/mL: picogram/ milliliter; CRP: C reactive protein; ng/L: nanogram/liter; U/L: unit/liter

Median glucose level was 104.00 (79.00–301.00) mg/dl; median creatinine level was 0.79 (0.44–1.70) mg/dL, mean alanine aminotransferase level was 29.46±2.58 U/L, median CRP level was 2.60 (0.14–39.30) mg/L, mean thyroid stimulating hormone level was 1.75±1.19 IU/ mL, mean hemoglobin level was 14.18±1.58 g/dL, mean WBC level was 7.91±2.04 10^3/mL, mean platelet count was 255.46±63.26, mean low-density lipoprotein level was 117.60±36.85 mg/dL, mean triglyceride level was 159.06±69.50 mg/dL. Laboratory findings of patients are shown in Table 3.

Normal AHI value was detected in 5 (4.2%) patients. Mild OSAS was detected in 30 (24.2%) of the patients, moderate OSAS in 24 (18.9%), and severe OSAS in 64 (51.6%). The median AHI value was 30.98 (10.79–56.13). When the patients with and without HT were compared, the AHI values of the HT group were statistically higher (p=0.001). There was a weak positive correlation between AHI and HT, r=0.280, N=95; the relationship was statistically significant (p=0.006). No statistically significant relationship was found between the presence of other comorbidities and AHI values (obesity p=0.735, smoking p=0.979, HL p=0.086, CAD p=0.503, DM p=0.257, COPD p=0.145, stroke history p=0.758, impaired fasting plasma glucose p=0.922 and male gender p=0.904).

Continuous positive airway pressure (CPAP) device report was given to 67 (54.7%) of the patients.

Discussion

Obstructive sleep apnea syndrome is particularly common in middle-aged and older adults[2]. Similarly, the patients in our study were in the middle age group. Recent studies show that the prevalence of OSAS is 2–4% in average-aged men and 1–2% in women[3]. Compared to the rates in the literature, we found the prevalence of OSAS in women to be higher in our study. Obstructive sleep apnea syndrome prevalence was found to be high in our region, as women's socioeconomic levels are higher, and the rate of admission to our hospital is higher for women. Obstructive sleep apnea syndrome is also associated with cardiovascular comorbidities such as arrhythmias, hypertension, stroke, coronary atherosclerosis, and overall increased cardiovascular mortality as well as metabolic dysfunction. Voulgaris et al. conducted a study to investigate the prevalence of comorbidities in a patient cohort of OSAS and COPD-OSAS overlap syndrome patients[4]. They found the prevalence of HT 58.9%, dyslipidemia 28.2%, DM 17.8%, cardiovascular disease 13.5%, atrial fibrillation 4.3%. Their findings were similar to those of this study. When the literature was searched, studies revealing the relationship between OSAS and HT were found. Nieto et al. conducted the Sleep Heart Health Study[5]. This study is a community-based study. It includes 6132 subjects from ongoing population-based studies (age >40 years) and investigates sleep-disordered breathing. They observed that incident HT increased compared to baseline. Bixler et al. conducted another prospective two-stage study involving >16000 individuals in the first phase and >1700 individuals in the second phase; sleep-disordered breathing was independently associated with HT[6]. Increasing evidence suggests that OSAS may be cause related to

various metabolic abnormalities, including insulin resistance, glucose intolerance, DM and metabolic syndrome regardless of adiposity[7–12]. Otake et al found that in an Asian population, >25% of OSAS patients were diagnosed with DM[13]. In Hispanic and African Americans, the prevalence of DM was 30% in OSAS patients[14]. Meslier et al. reported that the frequency of DM in OSAS patients was 30%, and Levinson et al. reported that the frequency of DM in OSAS patients was 11% in the study of the European population[15–16]. When compared with these studies, it was observed that the rate of DM was lower in our country. Impaired glucose intolerance in these studies was higher than ours. In the Sleep Heart Health Study, individuals with severe sleep apnea were shown to be four times more likely to have AF[17]. However, our study found that the prevalence of AF was similar to the normal population. The probability of finding Af in the resting ECG is low, and it would be appropriate to insert a 24-hour rhythm holter in these patients. Javaheri et al. and Sin et al., found the prevalence of heart failure 11% and 37%, respectively, in patients with OSAS[18–19]. Compared to these studies, the heart failure rate was lower in our patients. In our daily practice, dyspnea is usually associated with heart failure, and OSAS is ignored. When the demographic characteristics of the patients in our study were compared with the studies in the literature, it was observed that they were similar.

Laboratory-based polysomnography or home-based polysomnography can be used to diagnose OSAS[20]. Laboratory-based polysomnography was preferred in our hospital. Continuous positive airway pressure (CPAP) device report is given to patients diagnosed with moderate and severe OSAS. The diagnosis of OSAS in patients should encourage treatment with CPAP[21]. Wuest W et al. evaluate the long-term effect of CPAP therapy on cardiac functional parameters with cardiac Magnetic Resonance Imaging[22]. They found that left ventricular stroke volume and right ventricular ejection fraction were significantly improved with CPAP treatment ($p=0.04$). They found that all other cardiac parameters did not change significantly, while mean systolic and diastolic blood pressure improved significantly ($p<0.01$).

We included the adult patients who undergoing laboratory-based polysomnography in our hospital in this study. The most common chronic disease is HT in the patients. There was a weak positive correlation between AHI and HT, the relationship was statistically significant. 24-hour ABPM was performed in nine patients and a new diagnosis of hypertension was made. Antihypertensive treatment was started in these patients. Cardiovascular stress test was performed on 22 patients. Invasive coronary angiography was recommended for two patients and coronary computed tomographic angiography was recommended for four patients with positive test results. Myocardial perfusion scintigraphy was recommended to nine patients who could not perform cardiovascular stress test.

One patient was diagnosed with pfo. Transesophageal echocardiography was recommended for this patient. More than half of the patients participating in the study had a CPAP device report.

This study has several limitations. Firstly, the study was a single-center and retrospective analysis. Secondly, the number of patients participating in the study was small. Third, our hospital has no coronary angiography unit and nuclear medicine imaging center. For this reason, patients go to other hospitals to have the tests we recommend to investigate myocardial ischemia. Patients have yet to come to show test results. Therefore, the test results could not be written.

Cardiac evaluation should be performed before laboratory-based polysomnography is performed. Fasting blood glucose measurement, ECG, and TTE recording should be performed on the patients, and 24-hour ABPM should be inserted. Additional tests should be performed to investigate ischemia in patients with myocardial ischemia findings. It would be appropriate to check the cardiac functions of patients receiving CPAP treatment at specific intervals.

Conflict of Interest

The author declares that there is no conflict of interest regarding the publication of this manuscript.

References

1. Dunai A, Mucsi I, Juhasz J, et al. Obstructive sleep apnea and cardiovascular disease. Orv Hetil. 2006;147(48):2303–2311.
2. Levy P, Kohler M, McNicholas W, et al. Obstructive sleep apnoea syndrome. Nat Rev Dis Primers. 2015;1:15015.
3. Maspero C, Giannini L, Galbiati G, et al. Obstructive sleep apnea syndrome: a literature review. Minerva Stomatol. 2015;64(2):97–109.
4. Voulgaris A, Archontogeorgis K, Pataka A, et al. Burden of Comorbidities in Patients with OSAS and COPD-OSAS Overlap Syndrome. Medicina (Kaunas) . 2021;57(11):1201.
5. Nieto FJ, Young TB, Lind BK, et al. Association of sleep-disordered breathing, sleep apnea, and hypertension in a large community-based study. Sleep Heart Health Study. JAMA. 2000;283(14):1829–1836.
6. Bixler EO, Vgontzas AN, Lin HM, et al. Association of hypertension and sleep-disordered breathing. Arch Intern Med. 2000;160(15):2289–2295.
7. Botros N, Concato J, Mohsenin V, et al. Obstructive sleep apnea as a risk factor for type 2 diabetes. Am J Med. 2009;122(12):1122–1127.
8. Shaw JE, Punjabi NM, Wilding JP, et al. International Diabetes Federation Taskforce on Epidemiology and Prevention. Sleep-disordered breathing and type 2 diabetes: a report from the International Diabetes Federation Taskforce on Epidemiology and Prevention. Diabetes Res Clin Pract. 2008;81(1):2–12.
9. Keckeis M, Lattova Z, Maurovich-Horvat E, et al. I have impaired glucose tolerance in sleep disorders. PLoS ONE. 2010;5(3):e9444.
10. Punjabi NM, Shahar E, Redline S, et al. Sleep Heart Health Study Investigators. Sleep-disordered breathing, glucose intolerance, and insulin resistance: the Sleep Heart Health Study. Am J Epidemiol. 2004;160(6):521–530.
11. Punjabi NM, Beamer BA. Alterations in Glucose Disposal in Sleep-disordered Breathing. Am J Respir Crit Care Med. 2009;179(3):235–240.
12. Aronsohn RS, Whitmore H, Van Cauter E, et al. Impact of untreated obstructive sleep apnea on glucose control in type 2 diabetes. Am J Respir Crit Care Med. 2010;181(5):507–513.
13. Otake K, Sasanabe R, Hasegawa R, et al. Glucose intolerance in Japanese patients with obstructive sleep apnea. Intern Med. 2009;48(21):1863–1868.
14. Mahmood K, Akhter N, Eldeirawi K, et al. Prevalence of type 2 diabetes in patients with obstructive sleep apnea in a multi-ethnic sample. J Clin Sleep Med. 2009;5(3):215–221.
15. Meslier N, Gagnadoux F, Giraud P, et al. Impaired glucoseinsulin metabolism in males with obstructive sleep apnoea syndrome. Eur Respir J. 2003;22(1):156–160.
16. Levinson PD, McGarvey ST, Carlisle CC, et al. Adiposity and cardiovascular risk factors in men with obstructive sleep apnea. Chest. 1993;103(5):1336–1342.
17. Mehra R, Benjamin EJ, Shahar E, et al. Sleep Heart Health Study. Association of nocturnal arrhythmias with sleepdisordered breathing: The Sleep Heart Health Study. Am J Respir Crit Care Med. 2006;173(8):910–916.
18. Javaheri S, Parker TJ, Liming JD, et al. Sleep apnea in 81 ambulatory male patients with stable heart failure. Types and their prevalences, consequences, and presentations. Circulation. 1998;97(21):2154–2159.
19. Sin DD, Fitzgerald F, Parker JD, et al. Risk factors for central and obstructive sleep apnea in 450 men and women with congestive heart failure. Am J Respir Crit Care Med. 1999;160(4):1101–1106.
20. Harrison E, Roth RH, Lobo JM, et al. Sleep time and efficiency in patients undergoing laboratory-based polysomnography. J Clin Sleep Med. 2021;17(8):1591–1598.
21. Adir Y, Humbert M, Chaouat A. Sleep-related breathing disorders and pulmonary hypertension. Eur Respir J. 2021;57(1):2002258.
22. Wuest W, May MS, Wiesmueller M, et al. Effect of long term CPAP therapy on cardiac parameters assessed with cardiac MRI. Int J Cardiovasc Imaging. 2021;37(2):613–621.