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SLEEP BREATHING PHYSIOLOGY AND DISORDERS • ORIGINAL ARTICLE

NoSAS score predicts cardiovascular disease in patients with obstructive sleep apnea

Handan Inonu Koseoglu[1] · Ahmet Cemal Pazarli[1] · Asiye Kanbay[2] · Halil İbrahim Yakar[1] · Gökhan Aykun[1] · Osman Demir[3]

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Abstract

Purpose The Lausanne NoSAS (Neck circumference, Obesity, Snoring, Age, Sex) score is a new tool for the identification of high-risk patients for obstructive sleep apnea (OSA). Up to now, no study has attempted to determine the role of NoSAS score in cardiovascular morbidity of patients with OSA. We aimed to investigate the relationships between NoSAS scores and CVD and also between severity of OSA, polysomnographic parameters, and NoSAS scores in patients with OSA.

Methods Patients with diagnosis of OSA by full-night polysomnography were recruited in the study. Based on apnea–hypopnea index (AHI) scores, the patients were categorized as OSA-negative (AHI < 5), mild OSA (5 ≤ AHI < 15), moderate OSA (15 ≤ AHI < 30), and severe OSA (AHI ≥ 30). The definition of cardiovascular diseases (CVD) included the presence of any of the diseases such as hypertension, coronary artery disease, heart failure, or arrhythmia.

Results A total of 1514 patients including cases with 199 OSA-negative, 391 mild, 342 moderate, and 582 severe OSA were enrolled in the study. NoSAS scores were significantly different between mild, moderate, and severe OSA groups. NoSAS scores were negatively correlated with minimum oxygen saturation values and positively with AHI and ODI (oxygen desaturation index) values ($P < 0.001$). NoSAS scores were significantly higher in patients with CVD, diabetes mellitus, and cerebrovascular disease compared with those without ($P < 0.005$). NoSAS cut-off values for hypertension (14), congestive heart failure (8.5), coronary artery disease (9), cerebrovascular event (11), and diabetes mellitus (10) were also determined.

Conclusion NoSAS scores are associated with CVD and the severity of OSA. NoSAS scores may be useful to predict CVD in patients with OSA.

Keywords Cardiovascular disease · NoSAS score · Obstructive sleep apnea · Polysomnography · Screening

✉ Handan Inonu Koseoglu
handaninonu@gmail.com; hnd1inonu@yahoo.com

Ahmet Cemal Pazarli
drcemalpazarli@gmail.com

Asiye Kanbay
kanbaydr@yahoo.com

Halil İbrahim Yakar
halil_yakar@hotmail.com

Gökhan Aykun
gokhanaykun@gmail.com

Osman Demir
mosmandemir@hotmail.com

[1] Department of Pulmonary Diseases, Faculty of Medicine, Tokat Gaziosmanpasa University, 60100 Tokat, Turkey

[2] Medicana Atasehir Hospital, Istanbul, Turkey

[3] Department of Biostatistics and Medical Informatics, Faculty of Medicine, Tokat Gaziosmanpasa University, Tokat, Turkey

Introduction

Recurrent episodes of partial or total collapse of the upper airway while sleeping cause obstructive sleep apnea (OSA), accompanied by oxygen desaturation, increased activity of the sympathetic nervous system with frequent arousals. The approximate prevalence of OSA has been reported as 23.4% in women and 49.7% in men [1]. A global meta-analysis has estimated that around 425 million adults aged 30–69 years suffer from mostly undiagnosed and untreated moderate to severe OSA [2, 3]. OSA has been revealed to be an independent risk factor for the development and progression of various cardiovascular and metabolic diseases [1]. Abnormal sympathetic activity, inadequate oxygen supply, elevated circulating inflammatory mediators, or imbalances in the coagulation/fibrinolysis system may be speculated as the pathophysiological mechanisms underlying the strong relationship

between cardiovascular diseases (CVD) and OSA. There is also an association between OSA and increased plasma fibrinogen levels, platelet activation and hypercoagulability, and decreased fibrinolytic capacity [4, 5]. Therefore, early diagnosis and treatment of OSA is important to prevent cardiovascular comorbidities. Full-night polysomnography (PSG) is currently used as the gold standard procedure in the diagnosis of OSA. However, the procedure is time-consuming, expensive, and complex, and requires experienced technical personnel. In addition, the fact that the patients have to wait for long periods for the completion of sleep studies significantly precludes widespread use of these diagnostic tests in suspect cases of OSAS [6].

Screening methods have become important due to increasing prevalence of obesity worldwide with a resultant growing number of suspect cases of OSA referred to sleep clinics. In the prioritization of eligible patients, simple, easy to use, and low-cost screening questionnaires may be employed. For this purpose, many clinical prediction models have been developed based on clinical, demographic, and anthropometric variables [7, 8]. Among these screening tools, a newly developed NoSAS scoring system has recently been developed. Studies have indicated that the NoSAS score is a validated assessment tool much like the STOP-Bang, Berlin, or Epworth Sleepiness Scale (ESS) questionnaires in the prediction of OSA in different populations [10–14].

However, no previous studies have explored the clinical utility of NoSAS scores in predicting CVD in patients with OSA. To fill this gap, in the present study was aimed to investigate the predictive ability of NoSAS scores for CVD in patients with OSA.

Methods

Subjects

This study was conducted retrospectively with patients who had undergone polysomnography at our sleep unit between January 2014 and March 2020 based on the clinical suspicion of OSA. A standardized questionnaire was administered to the patients to collect information on their demographic characteristics (e.g., age and sex), anthropometric measurements (height, weight, body mass index [BMI], and neck circumference), medical history, sleeping habits, and medical treatments they were receiving before the sleep study. The definition of CVD included the presence of any of the diseases such as hypertension, coronary artery disease, heart failure, or arrhythmia. The diagnosis of CVD was made by an expert cardiologist based on medical history and medications the patients were receiving and according to the results of the examinations.

The inclusion criteria of the study were: (1) age > 18 years; (2) lack of any previous diagnosis made and treatment received because of sleep-disordered breathing; (3) having all data regarding anthropometric measurements including neck circumference and body mass index (BMI); and (4) a total sleep time of over 4 h on PSG. Patients who did not meet these criteria and those with central sleep apnea syndrome, narcolepsy, upper airway-resistance syndrome, hypoxemic lung disease, and active psychiatric disorder were excluded from the study.

The NoSAS scores range between 0 and 17 points. The NoSAS scoring system is as follows: neck circumference > 40 cm, 4pts; BMI 25–30 kg/m^2, 3 pts; BMI ≥ 30 kg/m^2, 5 pts; snoring, 2 pts; age > 55 years, 4 pts; and being male, 2 pts. A NoSAS score of 8 or higher is indicative of high risk for sleep-disordered breathing [9].

In-laboratory polysomnography

Overnight PSG (6–8 h) was performed using the Alice Sleepware (Philips Respironics) polysomnography device. Electrooculography (ROG, LOG), electroencephalography (C3A2, C4A1, O1A2, O2A1), electromyography (submental and anterior tibial muscle), electrocardiography, and body position records were taken. Respiratory recordings were obtained using a thermal sensor and pressure transducer, snore microphone, piezoelectric belts wrapped around chest and abdomen, and pulse oximetry probe. Recordings were evaluated by a certified sleep specialist, scored according to the guidelines of the American Academy of Sleep Medicine (AASM) [15]. The total number of apnea-hypopneas per sleep hour was defined as the apnea–hypopnea index (AHI). The oxygen desaturation index (ODI), the lowest oxygen saturation at night, and the sleep time period in which the oxygen saturation during the night was below 90% were evaluated.

Statistical analysis

Quantitative variables are expressed as mean ± standard deviation. Independent samples *t* test and one-way analysis of variance (one-way ANOVA) were used for intergroup comparisons of continuous data. For post hoc pairwise comparisons between groups, the Tukey HSD test was used. Qualitative variables are presented as numbers and percentages. Chi-square test was used for intergroup comparisons of the categorical data. Receiver operating characteristic (ROC) analysis was applied to determine the statistical power of NoSAS scoring system in predicting significant congestive heart failure (CHF), coronary artery disease (CAD), cerebrovascular event (CVE), CVD, hypertension (HT), diabetes mellitus (DM), and classifying types of arrhythmia. A P value of < 0.05 was considered the level of statistical

significant. Analyses were performed using SPSS 22 (IBM SPSS Statistics 22, SPSS inc., an IBM Co., Somers, NY).

Results

A total of 1514 participants including 956 (63.1%) male and 558 (36.9%) female patients were included in the study (Fig. 1). Mean age and body mass index (BMI) of the patients were 49.2 ± 12.1 years and 32.2 ± 6.4 kg/m2, respectively.

The clinical characteristics and PSG findings of the study population are presented in Table 1. Based on their AHI scores, the patients were categorized into four groups: OSAS-negative (AHI < 5), mild OSAS (5 ≤ AHI < 15), moderate OSAS (15 ≤ AHI < 30), and severe OSAS (AHI ≥ 30). A total of 1315 (87%) patients were classified as having OSAS, whereas 199 (13%) patients included in the OSAS-negative group. Patients had mild ($n = 391$: 26%), moderate ($n = 342$: 22%), and severe ($n = 582$: 38%) OSAS. The rates of CVD, HT, and DM were significantly different among groups ($P < 0.05$; Table 1). NoSAS scores were different between mild, moderate, and severe OSAS groups. The NoSAS scores increased in parallel with the severity of OSAS (Table 1).

Polysomnographic study results are shown in Table 1. As expected; AHI, ODI, minimum oxygen saturation, and desaturation levels (%) differed significantly among OSAS groups ($P < 0.05$).

Correlations between polysomnographic parameters and NoSAS scores were also determined. NoSAS scores were correlated negatively with minimum oxygen saturation values and positively with AHI, ODI, and desaturation percentage values ($P < 0.001$; Table 2).

The cases were divided into 2 groups as those with or without cardiometabolic diseases. NoSAS scores were higher in patients with CVD, diabetes mellitus, hypertension, and cerebrovascular disease compared to those without (Table 3).

The ROC analysis was performed to determine the cut-off value of NoSAS (≥ 8.5) in predicting clinically significant cases with CVD. This cut-off value of NoSAS had 82% sensitivity and 33% specificity with an AUC value of 0.61. The cut-off values for arrhythmia (8), CHF (8.5), CAD (9), CVE (11), DM (10), and HT (14) were also determined ($P < 0.001$; Table 4, Fig. 2).

Discussion

This study adds novel and comprehensive information about the predictive ability of NoSAS for the detection of CVD in patients with OSA. First, NoSAS scores increase in parallel with the severity of OSA and degree of hypoxemia. Second, NoSAS scores are significantly higher in

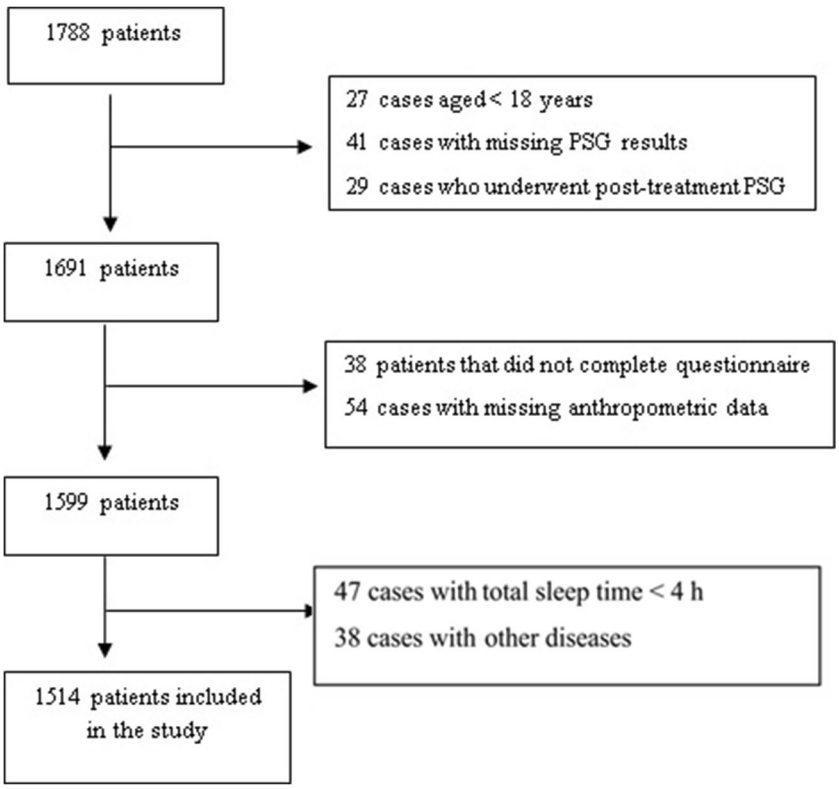

Fig. 1 Number of patients included in polysomnography test according to various exclusion criteria

Table 1 Clinical characteristics of participants

Variables	Total	OSAS-negative group	Mild OSAS	Moderate OSAS	Severe OSAS	*P*
Gender						
Male (*n*, %)	956 (63.1)	87 (43.7) (a)	242 (61.9) (b)	221 (64.6) (b)	406(69.8) (b)	< 0.001
Female (*n*, %)	558 (36.9)	112 (56.3) (a)	149 (38.1) (b)	121 (35.4) (b)	176(30.2) (b)	
Age (years) (mean ± SD)	49.2 ± 12.1	42.4 ± 12.5 (a)	46.8 ± 11.8 (b)	50.5 ± 11.5 (c)	52.4 ± 11.2 (d)	< 0.001
BMI (kg/m^2) (mean ± SD)	32.2 ± 6.4	28.5 ± 5.4 (a)	31.0 ± 5.9 (b)	32.0 ± 5.9 (b)	34.3± 6.6 (c)	< 0.001
Neck circumference (cm)	42.8 ± 6.4	41.0 ± 5.3 (a)	41.5 ± 6.1 (ab)	42.6 ± 5.9 (b)	44.3 ± 6.9 (c)	< 0.001
NoSAS (mean ± SD)	10.8 ± 3.5	8.1 ± 3.5 (a)	9.9 ± 3.3 (b)	10.9 ± 3.2 (c)	12.2 ± 3.2 (d)	< 0.001
CHF (*n*, %)	94 (6.2)	1 (0.5) (a)	11 (2.8) (ab)	16 (4.7) (b)	66 (11.3) (c)	< 0.001
CAD (*n*, %)	184 (12.2)	8 (4) (a)	22 (5.6) (a)	42 (12.3) (b)	112 (19.2) (c)	< 0.001
Arrhythmia (*n*, %)	98 (6.5)	4 (2) (a)	13 (3.3) (a)	15 (4.4) (a)	66 (11.3) (b)	< 0.001
CVE (*n*, %)	37 (2.4)	1 (0.5) (a)	1 (0.3) (a)	3 (0.9) (a)	32 (5.5) (b)	< 0.001
HT (*n*, %)	478 (31.6)	22 (11.1) (a)	81 (20.8) (b)	122 (35.7) (c)	253 (43.5) (c)	< 0.001
CVD (*n*, %)	601 (39.7)	25 (12.6) (a)	103 (26.3) (b)	136 (39.8) (c)	337 (57.9) (d)	< 0.001
DM (*n*, %)	304 (20.1)	8 (4) (a)	63 (16.1) (b)	79 (23.1) (bc)	154 (26.5) (c)	< 0.001
Polysomnographic findings						
AHI (mean ± SD)	29.1 ± 26.3	2.2 ± 1. (a)	9.2 ± 2.8 (b)	20.6 ± 3.8 (c)	56.8 ± 21.7 (d)	< 0.001
SE (%) (mean ± SD)	82.1 ± 10.8	82.1 ± 10.6	82.6 ± 10.6	82.0 ± 10.9	81.7 ± 10.9	0.597
Minimum O_2 sat (%) (mean ± SD)	80.7 ± 11.8	90.1 ± 4.7 (a)	86.5 ± 5.5 (b)	82.0 ± 8.4 (c)	72.9 ± 13.4 (d)	< 0.001
Desaturation (%) (mean ± SD)	10.1 ± 18.5	0.4 ± 1.8 (a)	1.8 ± 7.6 (a)	5.5 ± 12.5 (c)	21.8 ± 23.0 (d)	< 0.001
ODI (mean ± SD)	24.8 ± 28.5	1.7 ± 2.1 (a)	6.3 ± 7.8 (b)	15.6 ± 10.5 (c)	50.5 ± 29.8 (d)	< 0.001

Abbreviations: *CHF*, congestive heart failure; *CAD*, coronary artery disease; *CVE*, cerebrovascular event; *CVD*, cardiovascular diseases; *HT*, hypertension; *DM*, diabetes mellitus; *BMI*, body mass index; *AHI*, apnea–hypopnea index; *SE*, sleep efficiency; desaturation (%), sleep time of $SpO_2 < 90\%$; *ODI*, oxygen desaturation index; *O_2 sat*, oxygen saturation

Data are shown as frequency, percentage, or mean and standard deviation. Small letters (a, b, c, d) in parentheses indicate lack of any statistical significance

patients with OSA with CVD compared to those without CVD.

Due to the high prevalence of undiagnosed OSA and its comorbidities, a reliable screening tool would be useful for prompt prediction of OSA [16]. For this purpose, many questionnaires have been used to date [7, 8]. The NoSAS scoring system was first developed in a population-based study as a simple but effective test for OSA screening [9]. The NoSAS questionnaire contains a limited number of items compared to the Berlin, ESS, and STOP-Bang questionnaires. Since age, gender, BMI, and neck circumference variables of the NoSAS questionnaire

Table 2 Correlation analysis between NoSAS score and PSG parameters

	NoSAS	
	*r**	*P* values
Apnea–hypopnea index	0.330	< 0.001
Minimum O_2 saturation (%)	− 0.333	< 0.001
Desaturation (%)	0.287	< 0.001
Oxygen desaturation index	0.322	< 0.001

*Pearson correlation coefficient was used

Table 3 Mean NoSAS scores in patients with and without cardiometabolic diseases

Variables		NoSAS		*P* values
		n (%)	Mean ± SD	
CHF	Absent	1420 (93.8)	10.7 ± 3.5	0.002
	Present	94 (6.2)	11.8 ± 3.5	
CAD	Absent	1330 (87.8)	10.6 ± 3.5	< 0.001
	Present	184 (12.2)	12.0 ± 3.4	
Arrhythmia	Absent	1416 (93.5)	10.7 ± 3.6	0.116
	Present	98 (6.5)	11.3 ± 3.2	
CVE	Absent	1477 (97.6)	10.7 ± 3.5	0.018
	Present	37 (2.4)	12.1 ± 3.1	
HT	Absent	1035 (68.4)	10.3 ± 3.5	< 0.001
	Present	478 (31.6)	11.7 ± 3.4	
CVD	Absent	913 (60.3)	10.2 ± 3.5	< 0.001
	Present	601 (39.7)	11.6 ± 3.4	
DM	Absent	1210 (79.9)	10.4 ± 3.5	< 0.001
	Present	304 (20.1)	12.0 ± 3.3	

Independent samples *t* test was used

Abbreviations: *CHF*, congestive heart failure; *CAD*, coronary artery disease; *CVE*, cerebrovascular event; *CVD*, cardiovascular diseases; *HT*, hypertension; *DM*, diabetes mellitus

Table 4 Performance of the NoSAS score in predicting of cardiovascular diseases in patients with OSAS

Variable	Cut-off	AUC (95% CI)	Se	Sp	PPV	NPV	*P*
CHF	≥ 8.5	0.58 (0.52–0.64)	0.85	0.28	0.07	0.97	0.007
CAD	≥ 9	0.60 (0.56–0.65)	0.84	0.29	0.08	0.96	< 0.001
Arrhythmia	≥ 8	0.54 (0.48–0.60)	0.82	0.28	0.07	0.96	0.197
CVE	≥ 11	0.61 (0.52–0.69)	0.81	0.38	0.03	0.99	0.026
HT	≥ 14	0.61 (0.58–0.64)	0.89	0.27	0.53	0.73	< 0.001
CVD	≥ 8.5	0.61 (0.58–0.64)	0.82	0.33	0.45	0.73	< 0.001
DM	≥ 10	0.63 (0.59–0.66)	0.76	0.40	0.24	0.87	< 0.001

AUC, area under curve; *PPV*, positive predictive value; *NPV*, negative predictive value; *Se*, sensitivity, *Sp*, specificity

Abbreviations: *CHF*, congestive heart failure; *CAD*, coronary artery disease; *CVE*, cerebrovascular event; *CVD*, cardiovascular diseases; *HT*, hypertension; *DM*, diabetes mellitus

can be easily and accurately determined, this diagnostic tool can be used effectively and conveniently by clinicians and participants [10–12, 14, 17]. The sensitivity of the NoSAS score increases as the AHI increases [13, 16, 18, 19]. Similarly, we have also found that the NoSAS score was significantly associated with the severity of OSA as indicated by AHI in our study. Another important and novel finding of this study was that NoSAS score was negatively correlated with minimum oxygen saturation values and positively with ODI and desaturation percentage values which probably explains increases in hypoxia in line with aggravation of OSA. As supported by published data, patients with OSA carry an increased risk for CVD [20, 21]. Apart from predominant etiologic factors for increased rates of cardiac pathologies and risks of venous thromboembolism including vascular endothelial dysfunction, increased platelet aggregation, hypercoagulability, hypoxia-induced inflammation, and increased sympathetic tone, other additional factors may contribute to the development of CVD [22, 23].

This is the first study investigating the predictive performance of NoSAS in detecting CVD in patients with OSA. In a large cohort of healthy individuals, Cicero et al. showed that higher NoSAS score was associated with increased mean arterial pressure. While this study does not suggest a causal relationship between OSA and CVD, it is clinically relevant to use this score to identify individuals at higher cardiovascular risk who are likely to have OSA [24]. In our study, NoSAS score was significantly higher in cases with CVD, diabetes mellitus, hypertension, and cerebrovascular disease compared to those without.

Delesie et al. investigated the importance of screening questionnaires in demonstrating the presence of OSA in cases with atrial fibrillation and stated that use of these questionnaires in the decision-making process may refrain the clinician from referring the patients with atrial fibrillation and a NoSAS score below 8 to the sleep clinic [25]. In our study, the NoSAS threshold value for arrhythmia was found as 8 in patients with OSA.

In another study, Giampá et al. compared Berlin and NoSAS score performance for screening OSA in patients with treatment-refractory hypertension. Especially for the most severe forms of OSA, the NoSAS score has a higher accuracy in detecting OSA than the Berlin questionnaire. However, neither questionnaire was found useful to screen

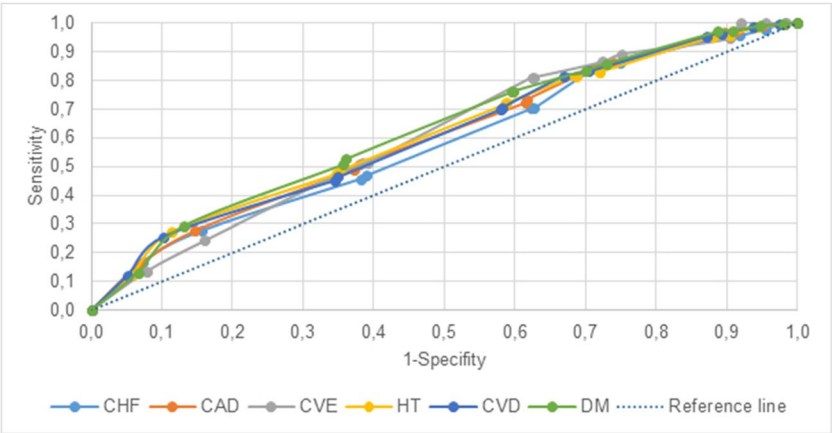

Fig. 2 NoSAS cut-off values to predict the presence of cardiovascular diseases and related ROC curves. Abbreviations: CHF, congestive heart failure; CAD, coronary artery disease; HT, hypertension; CVD, cardiovascular disease; DM, diabetes mellitus

for OSA in treatment-resistant hypertension. The authors stated that the poor performance of the NoSAS score might be related to the different phenotypes of OSA in patients with CVD [26]. In our study, the mean NoSAS score was 11.7 and 10.3 in patients with and without hypertension, respectively, with a statistically significant intergroup difference. However, we did not compare patients with and without treatment-refractory hypertension in our cohort based on NoSAS scoring system. NoSAS cut-off value was determined as 14, in hypertensive patients. Other important and novel finding of this study was that the mean NoSAS score was significantly higher in patients with congestive heart failure, coronary artery disease, cerebrovascular event, and diabetes mellitus than those without. NoSAS cut-off values for these diseases were determined as 8.5, 9, 11, and 10, respectively. According to these results, NoSAS score may be a useful marker that may be used to predict not only the presence of OSA but also cardiometabolic diseases in patients with OSA.

There are imitations of our study that deserve to be mentioned. This was a retrospective study. NoSAS score was obtained by reviewing medical records. The use of retrospective analysis to evaluate a screening tool is less ideal than a prospective study. However, this is an observational study with a large sample size. Phenotyping studies showed that using arousal index may predict cardiovascular outcome in OSA better than using only AHI score [27]. Due to the retrospective design of the study, we did not have arousal index and other relevant parameters in all patients' data to categorize the cohort according to this new scoring system. We could not test the association between NoSAS and arousal index. This should be investigated in future studies.

In conclusion, the NoSAS score is associated with the severity of OSA and CVD in patients with OSA. The findings suggest that the NoSAS score may be useful to predict CVD in patients with OSA. Further prospective studies are warranted to evaluate the prognostic value of NoSAS for the development of new cardiovascular events in patients with OSA.

Author contribution All authors contributed to the conception and design of the study. Preparation, collection, and analysis of data were performed by Handan Inonu Koseoglu, Asiye Kanbay, Ahmet Cemal Pazarli, and Osman Demir. The first draft of the manuscript was written by Handan Inonu Koseoglu and all authors commented on previous versions of the manuscript. All authors have read and approved the final version of the manuscript.

Data availability The datasets generated and/or analyzed during the current study are available from the corresponding author on reasonable request.

Declarations

Ethics approval This retrospective study involving human participants has been conducted in accordance with the ethical standards of the institutional and national research committee and with the 1964 Helsinki Declaration and its later amendments or comparable ethical standards. The Human Investigation Committee of Tokat Gaziosmanpasa University approved this study (Project Number: 22-KAEK-195).

Conflict of interest The authors declare no competing interests.

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