ARAŞTIRMA MAKALESİ / *RESEARCH ARTICLE*

Relationship Between Electrical Risk Score and Obstructive Sleep Apnea

Elektriksel Risk Skoru ile Obstrüktif Uyku Apnesi Arasındaki İlişki

Dogan Ilis[1], Ayca Arslan[1], Inanc Artac[1], Muammer Karakayali[1], Omer Kertmen[2], Ihsan Topaloglu[3], Cagri Atasoy[3], Yuksel Erata[1], Yavuz Karabag[1], Ibrahim Rencuzogullari[1]

[1]Department of Cardiology, Kafkas University Faculty of Medicine, Kars; [2]Department of Cardiology, Amasya University Faculty of Medicine, Amasya; [3]Department of Pulmonology, Kafkas University Faculty of Medicine, Kars, Türkiye

ABSTRACT

Aim: *The most common type of sleep disordered breathing in industrial societies is obstructive sleep apnea (OSA). The Electrical Risk Score (ERS) comprises 6 distinct electrocardiographic parameters, each representing aspects of the heart's electrical activity and structural damage. Our study aimed to investigate the association between the ERS and the severity of OSA in patients diagnosed with OSA.*

Material and Methods: *This retrospective study included 255 patients diagnosed with OSA after polysomnography. The patients were then divided into three groups based on apnea-hypopnea index (AHI). The groups were analyzed based on clinical, laboratory, and electrocardiogram (ECG) characteristics.*

Results: *When we compared the groups, OSA was significantly associated with hypertension (HT) (p <0.001) and a higher body mass index (BMI) (p=0.003). Group 3 showed a statistically higher ERS value (p <0.001), higher heart rate (p <0.001), increased electrocardiographic left ventricular hypertrophy (LVH) rates (p <0.001), a delayed QRS transition zone (p <0.001), prolonged QTc (p <0.001), a larger QRS-T angle (p=0.005), and a longer Tpeak-Tend interval (p <0.001).*

Conclusion: *Our study showed that ERS is linked to OSA severity and acts as an independent predictor of severe OSA.*

Key words: *obstructive sleep apnea OSA; electrical risk score ERS; sleep disorders*

ÖZET

Amaç: *Obstrüktif uyku apnesi (OUA), endüstriyel toplumlarda en sık görülen uykuda solunum bozukluğu türüdür. Elektriksel Risk Skoru (ERS), her biri kalbin elektriksel aktivitesini ve yapısal hasarını temsil eden altı farklı elektrokardiyografik parametreden oluşur. Çalışmamızın amacı, OUA tanısı konan hastalarda ERS ile OSA şiddeti arasındaki ilişkiyi incelemek.*

Gereç ve Yöntem: *Bu retrospektif çalışmaya polisomnografi sonrası OUA tanısı konan 255 hasta dâhil edilmiştir. Daha sonra hastalar apne-hipopne endeksine (AHE) göre üç gruba ayrılmıştır. Gruplar klinik, laboratuvar ve EKG özelliklerine göre analiz edilmiştir.*

Bulgular: *Gruplar karşılaştırıldığında, OUA'nın hipertansiyon varlığı (p <0,001) ve daha yüksek vücut kitle endeksi (p=0,003) ile istatiksel olarak ilişkili olduğu izlendi. Grup 3'te istatistiksel olarak daha yüksek ERS değeri (p <0,001), daha yüksek kalp hızı (p <0,001), artmış sol ventrikül hipertrofisi oranları (p <0,001), gecikmiş R dalga transizyon zonu (p <0,001), uzamış QTc (p <0,001), artmış QRS-T açısı (p=0,005) ve daha uzun Tpeak-Tend aralığı (p <0,001) görüldü.*

Sonuç: *Çalışmamız, ERS'nin OUA şiddeti ile ilişkili olduğunu ve şiddetli OSA'nın bağımsız bir öngörücüsü olduğu göstermiştir.*

Anahtar kelimeler: *obstrüktif uyku apnesi OUA; elektriksel risk skoru ERS; uyku bozuklukları*

İletişim/Contact: *Doğan İliş, Kafkas University Faculty of Medicine, Department of Cardiology, Kars, Türkiye* • ***Tel:*** *0541 523 18 54* • ***E-mail:*** *ilisdogan@hotmail.com* • ***Geliş/Received:*** *02.10.2025* • ***Kabul/Accepted:*** *22.10.2025*

ORCID: *Doğan İliş: 0000-0002-1871-5157 • Ayça Arslan: 0000-0002-9152-1199 • İnanç Artaç: 0000-0003-2694-8978 • Muammer Karakayalı: 0000-0001-7385-120X • İhsan Topaloğlu: 0000-0001-9645-6579 • Çağrı Atasoy: 0000-0001-6662-9600 • Ömer Kertmen: 0000-0002-9951-2617 • Yüksel Erata: 0009-0008-2400-0781 • Yavuz Karabağ: 0000-0002-8156-315X • İbrahim Rencuzoğulları: 0000-0002-0070-9197*

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Introduction

Obstructive sleep apnea (OSA) is characterized by periodic pauses in breathing lasting more than 10 seconds that occur during sleep owing to upper airway occlusion, leading to increased respiratory effort[1]. Obstructive sleep apnea is a widespread issue globally. Research involving large populations shows that OSA impacts 2% to 5% of adult women and 3% to 7% of adult men. However, some groups, such as elderly persons and those who are overweight or obese, have a higher prevalence [2]. Multiple organ systems are affected systemically by OSA. Long-term health issues like depression, heart disease, diabetes, stroke, metabolic problems, and high blood pressure are linked to untreated OSA. Furthermore, untreated OSA has been linked to cognitive dysfunction, decreased working productivity, and a higher chance of accidents that cause injuries and fatalities[3].

For OSA patients, several negative effects can occur, including hypoxemia, hypercapnia, acidosis, increased adrenergic system and afterload, and rapid fluctuations in heart rate, leading to heart conduction problems and arrhythmias. Previous studies have examined OSA and electrocardiogram (ECG) parameters, including their changes[4–7]. However, ECG parameters were typically analyzed individually in these studies. To predict risks and adverse effects in a variety of patient populations, numerous studies have employed the electrical risk score (ERS), which comprises the following: heart rate, a diagnosis of left ventricular hypertrophy (LVH), heart rate >75 bpm, the R transition zone, the corrected QT interval (QTc), the T peak to T end interval (Tp-e), and the frontal QRS-T angle[8–12].

Given the systemic effects of OSA, it is likely associated with ERS, which provides practical information on the structure and function of the heart. Our study aimed to investigate the association between the ERS and OSA, as well as the severity of OSA, in patients diagnosed with OSA.

Material and Method

Patient Selection

This retrospective study included 255 patients who underwent sleep laboratory testing between January 2023 and December 2024 and were subsequently diagnosed with OSA after the test. The study's inclusion criteria were being over eighteen years old and having an apnea-hypopnea index greater than five. Patients were excluded if they had morbid obesity, pulmonary embolism, pulmonary hypertension, abnormal thyroid function, cardiomyopathies, pacemaker implantation, conduction disturbances (atrioventricular or intraventricular), a history of atrial fibrillation (paroxysmal or permanent), valvular heart disease, pericarditis, abnormal serum electrolytes, use of antiarrhythmic drugs, or coronary artery disease.

This study was approved by the Kafkas University Faculty of Medicine's Ethics Committee (Approval Number: 80576354–050–99/740, dated 30.10.2024) and carried out in compliance with the Declaration of Helsinki criteria. As it was a retrospective study, no written or verbal consent was obtained from the patients.

Polysomnography

Arousal index, oxygen desaturation index, apnea-hypopnea index (AHI), resting room air pulse oximetry (SpO_2), total sleep time (TST), sleep efficiency (the percentage of total bedtime spent sleeping), and sleep duration with oxygen saturation below 90% were among the parameters analyzed. To diagnose OSA and determine its severity, the AHI, which indicates the number of apnea and hypopnea episodes per hour during sleep, was used. Obstructive sleep apnea severity was categorized as mild (5 to <15 events/h), moderate (15 to <30 events/h), and severe (≥30 events/h). Patients with an AHI of 5–15 were classified as having mild OSA (Group 1), those with an AHI of 15–30 as moderate (Group 2), and an AHI above 30 as severe (Group 3). The study included only individuals whose AHI was at least five incidents per hour.

Electrocardiographic analyses

An archival resting 12-lead ECG, recorded at 25 mm/sec and calibrated to 10 mm/mV, was available for analysis in each of the study's patients and controls. The ECG's heart rate, QTc interval, Tpeak-to-Tend (TpTe) interval, frontal QRS-T angle, delayed QRS transition zone, and LVH were all assessed. The QTc interval was considered prolonged if it exceeded 450 ms in men and 460 ms in women. These parameters are used to calculate the electrical risk score[9]. Each abnormal parameter earned 1 point; normal parameters scored 0. Points were assigned as follows: heart rate >75 bpm; LVH per Sokolow–Lyon criteria; QRS transition zone at or beyond V4; QTc >450 ms in men and >460 ms in women; Tp-e >89 ms; and frontal QRS-T angle >90°[13]. Left ventricular hypertrophy was defined as a voltage sum (V1 S

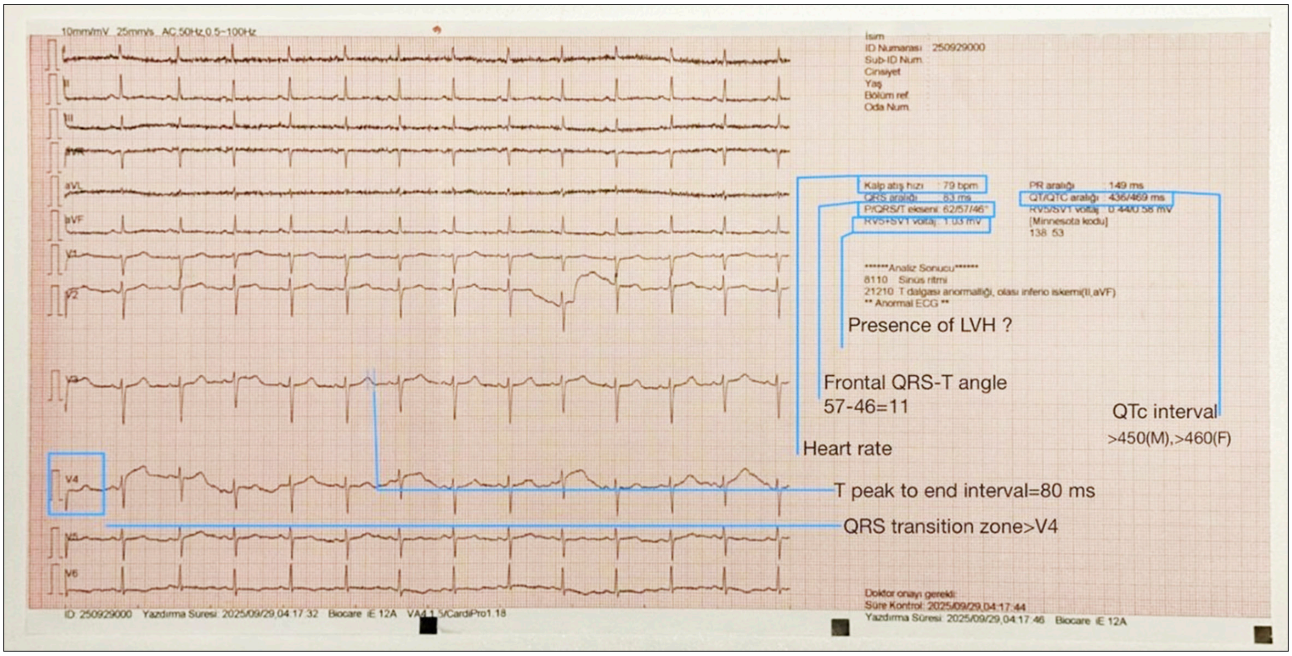

Figure 1. *Parameters and methods used to calculate the electrical risk score.*

wave + V5/6 R wave) ≥35 mm (3.5 mV), according to the Sokolow–Lyon criteria[14]. Bazett's formula (QTc=QT / √RR) was used to adjust the QT interval, which was measured from the beginning of QRS to the conclusion of T. The Tp-e interval was from the T wave peak to its end[14]. The precordial lead in which the R-wave height equaled or exceeded the S-wave height was determined to be the QRS transition zone[15]. When the difference between the QRS and T axis was more than 180°, the frontal QRS-T angle was computed as 360° minus the difference[16]. Some parameters, such as heart rate, QTc interval, frontal QRS-T angle, and Sokolow-Lyon criteria, were available directly from the automated ECG report. Others, such as the Tp-e interval and the QRS transition zone, were manually measured. An example of the ERS assessment from the 12-lead ECG and its automatic report is shown in Fig. 1.

Statistical Analysis

We performed statistical analysis using IBM Statistical Package for Social Sciences (SPSS) program version 24.0 software. The Kolmogorov-Smirnov test was employed to assess the normality of the data distribution. The t-test or Mann-Whitney U test was used to compare continuous variables between groups, while Fisher's exact test or the chi-square test was applied for categorical variables. Continuous data are presented as mean ± standard deviation (SD) or median with interquartile range (minimum-maximum). Categorical variables are expressed as absolute (n) and relative frequencies (%), as well as percentages (%n). Variables identified as significant in univariate analysis were further analyzed with multivariate methods. An odds ratio (OR) with a 95% confidence interval was calculated for each independent variable. Multicollinearity between the Electrical risk score and its components was evaluated using eigenvalues and the condition index. The optimal cut-off points for predicting OSA patients were determined using receiver operating characteristic (ROC) curves and their corresponding area under the curve (AUC) values. Statistical significance was defined as p-values below 0.05.

Results

The study included 255 patients, with 91 males (36%) and 164 females (64%). The average age was 49±12 years. There were 69 (27%), 66 (26%), and 120 (47%) patients in OSA Groups 1, 2, and 3, respectively.

The distribution of general characteristics among the patient groups is shown in Table 1. When demographic data were compared, OSA was significantly associated with the presence of HT ($p < 0.001$) and a higher BMI ($p=0.003$). However, OSA was not significantly associated with age, gender, the presence of diabetes mellitus, heart failure, COPD, or current smoking status (Table 1). When we compared the ERS and

Table 1. *Demographic, clinical, and laboratory data for all patients*

	OSA Severity							
Variables	Group 1 (Mild) n: 69	Group 2 (Moderate) n: 66	Group 3 (Severe) n: 120	Total n: 255	P value	1 vs 2	1 vs 3	2 vs 3
Age, years	46±12	50±14	51±12	49±12	0.118	0.172	0.059	0.970
Male, n (%)	26 (38)	25 (38)	40 (33)	91 (36)	0.761	1.000	0.821	0.811
Hypertension, n (%)	13 (19)	17 (26)	55 (46)	85 (33)	**<0.001**	0.656	**<0.001**	**0.013**
Diabetes mellitus, n (%)	16 (23)	16 (24)	34 (28)	66 (26)	0.695	0.989	0.719	0.817
Heart failure, n (%)	5 (7)	10 (15)	17 (14)	32 (13)	0.294	0.351	0.352	0.980
COPD, n (%)	8 (12)	6 (9)	9 (8)	23 (9)	0.640	0.869	0.614	0.931
BMI, kg/m^2 (SD)	31. ±5.8	31.8±6.1	34.1±5.8	32.8±5.9	**0.003**	0.982	**0.017**	**0.033**
Current smoker, n (%)	37 (54)	29 (44)	58 (48)	124 (49)	0.530	0.502	0.765	0.835
WBC count, 10^3/μL (SD)	7.9±2	7.8±2	8.2±2	8±2	0.319	0.906	0.619	0.344
Hemoglobin, g/dL (SD)	14.7±2	14.5±2	14.8±2	14.7±2	0.189	0.720	0.880	0.373
Platelet, 10^3/L (SD)	239±60	235±63	245±70	241±66	0.436	0.945	0.793	0.579
Glucose, mg/dL (SD)	113±26	115± 35	119±33	116±32	0.237	0.970	0.414	0.586
Creatinine, mg/dL (SD)	0.90±0.15	0.94± 0.30	0.96±0.22	0.94±0.23	0.091	0.663	0.228	0.792
ALT, (SD)	24±11	22±12	29±20	26±16	0.760	0.710	0.171	0.022
AST, (SD)	22±5	21±9	22±8	22±7	0.101	0.751	0.876	0.399
Total-Cholesterol, mg/dL (SD)	193±40	195±47	199±41	196±42	0.349	0.962	0.639	0.823
Albumin, g/L (SD)	4.2±0.4	4.1±0.4	4.2±0.4	4.2±0.4	0.578	0.885	0.881	0.577
C-reactive protein, mg/dL (IQR)	4 (2–7)	5 (2–9)	4 (2–9)	4 (2–9)	0.553	0.571	0.990	0.413
LVEF, % (SD)	56±8	56±9	54±8	55±8	0.104	0.873	0.163	0.429
TAPSE, mm (SD)	25±7	25±5	23±6	24±6	0.212	0.917	0.172	0.382
TRV, m/s (SD)	1.7±0.7	1.7±0.6	1.7±0.7	1.7±0.7	0.937	0.921	0.992	0.946
Pulmonary artery diameter, cm (SD)	2.1±0.3	2.2±0.3	2.3±0.2	2.2±0.3	0.645	0.659	0.987	0.686
RV basal diameter, cm (SD)	4.1±0.6	4.0±0.6	4.0±0.7	4.0±0.7	0.477	0.678	0.519	0.990
ERS (IQR)	0 (0–1)	2 (1–3)	3 (2–3)	2 (1–3)	**<0.001**	**<0.001**	**<0.001**	**<0.001**
Heart rate >75bpm, n (%)	16 (23)	30 (46)	86 (72)	132 (52)	**<0.001**	**0.014**	**<0.001**	**0.001**
Electrocardiographic LVH, n (%)	5 (7)	18 (27)	45 (38)	68 (27)	**<0.001**	0.019	**<0.001**	0.263
Delayed QRS transition zone, n (%)	13 (19)	18 (27)	55 (46)	86 (34)	**<0.001**	0.538	**<0.001**	0.024
Prolonged QTc^a, n (%)	3 (4)	8 (12)	34 (28)	45 (18)	**<0.001**	0.440	**<0.001**	**0.012**
QRS-T angle >90, n (%)	9 (13)	20 (30)	42 (35)	71 (28)	**0.005**	0.062	**0.003**	0.767
Tpeak-Tend >89 ms, n (%)	10 (15)	10 (15)	46 (38)	66 (26)	**<0.001**	0.996	**0.001**	**0.001**
Tpeak-Tend, ms (SD)	72±15	72±16	79±17	75±17	**0.004**	0.983	**0.020**	**0.038**
QTc, ms (SD)	428±17	433±17	439±21	434±20	**0.006**	0.268	**0.001**	0.157
QRS-T angle, ms (IQR)	41 (28–70)	45 (28–91)	65 (34–93)	45 (29–91)	**0.004**	0.369	**0.002**	0.140
Heart rate, bpm	74±10	77±11	79±9	77±10	**<0.001**	0.072	**0.004**	0.758

COPD: chronic obstructive pulmonary disease; BMI: body mass index; WBC: white blood cell; ALT: alanine transaminase; AST: aspartate aminotransferase; LDL-C: low-density lipoprotein cholesterol; LVEF: left ventricular ejection fraction; TAPSE: tricuspid annular plane systolic excursion; TRV: tricuspid regurgitation velocity; LVH: left ventricular hypertrophy; ERS: electrical risk score; a: QTc >450 ms in men and >460 ms in women.

ECG parameters across the groups, group 3 exhibited a significantly higher ERS value ($p<0.001$), higher heart rate ($p <0.001$), increased rates of electrocardiographic LVH ($p<0.001$), a delayed QRS transition zone ($p<0.001$), prolonged QTc interval ($p <0.001$), a larger QRS-T angle ($p=0.005$), and a longer Tpeak-Tend interval ($p<0.001$) (Table 1).

Univariate logistic regression analyses revealed that HT ($p <0.001$), BMI ($p=0.002$), and ERS ($p <0.001$) are significant prognostic factors for patients with OSA. Subsequently, variables that were significant in univariate analyses were included in the multivariate model. Hypertension (OR 2.628, 95% CI 1.400–4.932, $p=0.003$) and ERS (OR 2.487, 95% CI 1.941–3.188, $p <0.001$) emerged as independent predictors of OSA based on the multivariate logistic regression analysis (Table 2).

In ROC curve analysis, a cutoff value of ≥2 for the ERS was found to have 78.3% sensitivity and 64.4% specificity in predicting OSA patients. The area under the curve (AUC) for the ERS was calculated to be 0.794 (95% CI: 0.739–0.842) (Fig. 2).

Table 2. *Analysis of variables predicting severe OSA —univariate and multivariate*

	Univariate			Multivariate		
Variables	Odds ratio	95% CI	P value	Odds ratio	95% CI	P value
Hypertension	2.962	1.723–5091	**<0.001**	2.628	1.400–4.932	**0.003**
BMI	1.072	1.026–1.119	**0.002**			
ERS	2.545	1.991–3.255	**<0.001**	2.487	1.941–3.188	**<0.001**

BMI: body mass index; ERS: electrical risk score.

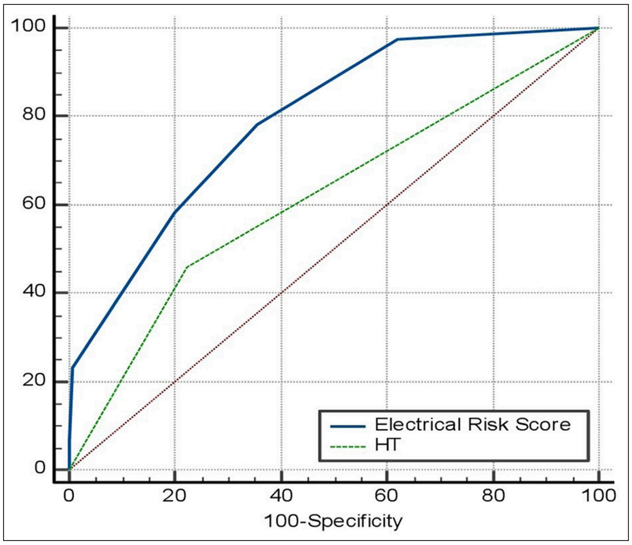

Figure 2. *The receiver operating characteristic (ROC) curve analysis for hypertension and electrical risk score in relation to OSA severity.*

Discussion

Our study showed that ERS is associated with OSA severity and is an independent predictor of severe OSA. As far as we know, this is the initial study to investigate this relationship in patients with OSA.

In developed countries, OSA is the most widespread form of sleep-disordered breathing[17]. Obstructive sleep apnea involves repeated episodes in which the upper airway collapses (apneas) or nearly collapses (hypopneas) during sleep, leading to intermittent low oxygen levels (hypoxemia) and increased sympathetic arousal. Obstructive sleep apnea/hypopnea syndrome occurs when daytime fatigue, sleepiness, and other neurological symptoms are caused by apneas and hypopneas during sleep[17–19]. Previous studies have demonstrated that hypertension is common among individuals with OSA[20]. Repeated episodes of apnea cause intermittent hypoxia, which triggers a range of autonomic, hemodynamic, and biochemical changes that play a role in developing hypertension and cardiovascular issues. It has been demonstrated that, in a human model, intermittent hypoxia raises mean arterial pressure, cerebral vascular resistance, and the pressor response[21]. In our study, hypertension was more common in Group 3. Additionally, a higher ERS value is associated with greater OSA severity. Weight gain, like hypertension, is strongly associated with OSA[22,23]. A longitudinal study found that in normal-weight individuals, gaining 10% of body weight increased the risk of OSA sixfold[24]. Conversely, losing 10% of weight led to a 26% reduction in the apnea-hypopnea index. Like hypertension, the link between obesity and OSA is bidirectional and mutually reinforcing. Tasali et al. showed in their study that obesity is a risk factor for OSA and, in addition, OSA increases the likelihood of obesity due to metabolic disorders[25]. In our study population, the mean BMI was 32. Furthermore, among the OSA groups, we observed the highest BMI in the severe OSA group.

To date, only a limited number of studies have explored the clinical importance of ERS across various populations. Aro et al.[9] discovered that ERS was linked to sudden cardiac death in patients carrying a high number of cardiovascular risk factors. The ERS is reported to include 6 distinct electrocardiographic parameters, each representing aspects of the heart's electrical activity and structural damage. Electrocardiogram parameters in this risk score have been identified as significant. They may act as predictors of poor clinical outcomes, as they reflect repolarization abnormalities (QRS angle, QTc, and Tp-e), imbalanced neuro-autonomic regulation (QTc, heart rate, and Tp-e), and cardiac hypertrophy (QTc, QRS angle, QRS transition, and Tp-e)[11,26]. Numerous cardiovascular problems, including atrial fibrillation and other supraventricular and ventricular arrhythmias, stroke, heart failure, hypertension, coronary artery disease, metabolic syndrome, pulmonary hypertension, diabetes, and an elevated risk of cardiovascular death, have been connected to OSA[26]. Acute apneic episodes lead to hypoxia and hypercapnia, increase sympathetic activity, change intrathoracic pressure, and cause autonomic dysregulation. Repeated episodes over time and sudden negative shifts in intrathoracic pressure can result in structural and functional remodeling, as well as electrophysiological changes[26,27].

Several ECG parameters have been examined separately in OSA patients; however, to our knowledge, no studies have investigated the clinical significance of ERS in individuals with OSA. The group of individuals with severe OSA in our study had a considerably higher mean ERS score. Aro et al.[9] discovered that ERS was linked to sudden cardiac death in patients with numerous cardiovascular risk factors. In patients receiving transcatheter aortic valve replacement, ERS was found to be the most significant predictor of cardiovascular or all-cause death by Piccirillo et al.[11]. Elmas AN et al.[8] observed that NSTEMI patients with an ERS of 3 or higher at admission experienced significantly increased in-hospital adverse events and mortality rates. Our research determined that the optimal ERS cutoff for predicting severe OSA was 2.0, based on ROC curve analysis. Performing an ECG, a simple, inexpensive, and easily accessible test, before referring patients with suspected OSA to the sleep laboratory, will be beneficial for clinical practice. It is essential to optimize the efficiency of polysomnography, a time-consuming procedure performed with limited resources, for accurate diagnosis of OSA. This straightforward score can help identify patients' risk levels and allocate limited resources more effectively. Furthermore, due to limitations in healthcare infrastructure and the cost of polysomnography testing, identifying these patients is often challenging and delayed. ECG-derived ERS, which is inexpensive and easily available, could potentially be used in clinical practice to risk-stratify patients and confirm the diagnosis of high-priority patients.

Conclusion

It may be important for predicting patients with suspected OSA and could help in identifying those who might need early testing and intervention. Additionally, this simple ECG-derived score can be used in clinical practice to detect the cardiac aspects of systemic adverse effects caused by OSA. Further research with larger groups of participants is needed to better understand the role of ERS in patients with OSA.

Limitations

Our research has several limitations. The primary one is its retrospective nature and the small sample size of patients. Additionally, ethnic differences could not be identified because the cohort was mainly from one location. Another limitation is the absence of a comparison between our patient population and the control group. Finally, we were unable to perform serial ECG monitoring of patients throughout their hospitalization. Calculating ERS on the final pre-discharge ECG, monitoring the ERS score, and analyzing the dynamic changes in the ERS score, along with assessing its relationship with long-term adverse events, could have enhanced the significance of our study.

Declaration of Interest

The authors have no conflicts of interest related to the publication of this article.

Data Availability Statement

Data available upon request due to privacy and ethical restrictions (The data supporting the findings of this study are available upon request from the corresponding author. The data are not publicly available because of privacy or ethical concerns).

References

1. Somers VK, White DP, Amin R, Abraham WT, Costa F, Culebras A, et al. Sleep apnea and cardiovascular disease: an American Heart Association/American College of Cardiology Foundation Scientific Statement from the American Heart Association Council for High Blood Pressure Research Professional Education Committee, Council on Clinical Cardiology, Stroke Council, and Council on Cardiovascular Nursing. J Am Coll Cardiol. 2008;52(8):686–717.
2. Punjabi NM. The epidemiology of adult obstructive sleep apnea. Proc Am Thorac Soc. 2008;5(2):136–43.
3. Morsy NE, Farrag NS, Zaki NFW, Badawy AY, Abdelhafez SA, El-Gilany AH, et al. Obstructive sleep apnea: personal, societal, public health, and legal implications. Rev Environ Health. 2019;34(2):153–169.
4. Sharma H, Sharma KK. An algorithm for sleep apnea detection from single-lead ECG using Hermite basis functions. Comput Biol Med. 2016;77:116–24.
5. Wang L, Lin Y, Wang J. Computer Methods and Programs in Biomedicine A RR interval based automated apnea detection approach using residual network. Comput Methods Programs Biomed. 2019;176:93–104.
6. Hassan AR, Haque MA. Computer-aided obstructive sleep apnea screening from single-lead electrocardiogram using statistical and spectral features and bootstrap aggregating. Biocybernetics and Biomedical Engineering. 2016;36(1):256–66.

7. Sharma H, Sharma KK. Sleep apnea detection from ECG using variational mode decomposition. Biomed Phys Eng Express. 2020;6(1):015026.
8. Elmas AN, Fedai H, Toprak K, Taşcanov MB, Altıparmak İH, Biçer A, et al. The Association of Electrical Risk Score with Prognosis in Patients with Non-ST Elevation Myocardial Infarction Undergoing Coronary Angiography. Anatol J Cardiol. 2024;29(1):11–8.
9. Aro AL, Reinier K, Rusinaru C, Uy-Evanado A, Darouian N, Phan D, et al. Electrical risk score beyond the left ventricular ejection fraction: prediction of sudden cardiac death in the Oregon Sudden Unexpected Death Study and the Atherosclerosis Risk in Communities Study. Eur Heart J. 2017;38(40):3017–3025.
10. Öncel CR, Çoner A, Köseoğlu C. Determining ECG Parameters for Electrical Risk Score in Patients with Non-ST Elevation Myocardial Infarction. Anatol J Cardiol. 2025;29(5):265–6.
11. Piccirillo G, Moscucci F, Mastropietri F, Di Iorio C, Mariani MV, Fabietti M, et al. Possible predictive role of electrical risk score on transcatheter aortic valve replacement outcomes in older patients: preliminary data. Clin Interv Aging. 2018;13:1657–1667.
12. Atilan Fedai U, Fedai H, Tanriverdi Z. Cardiac Clues in Major Depressive Disorder: Evaluating Electrical Risk Score as a Predictive Electrocardiography Biomarker. Medicina (Kaunas). 2025;61(6):1026.
13. Rautaharju PM, Surawicz B, Gettes LS, Bailey JJ, Childers R, Deal BJ, et al. American Heart Association Electrocardiography and Arrhythmias Committee, Council on Clinical Cardiology; American College of Cardiology Foundation; Heart Rhythm Society. AHA/ACCF/HRS recommendations for the standardization and interpretation of the electrocardiogram: Part IV. the ST segment, T and U waves, and the QT interval: a scientific statement from the American Heart Association Electrocardiography and Arrhythmias Committee, Council on Clinical Cardiology; the American College of Cardiology Foundation; and the Heart Rhythm Society. Endorsed by the International Society for Computerized Electrocardiology. J Am Coll Cardiol. 2009;53(11):982–91.
14. You Z, He T, Ding Y, Yang L, Jiang X, Huang L. Predictive value of electrocardiographic left ventricular hypertrophy in the general population: A meta-analysis. J Electrocardiol. 2020 Sep-Oct;62:14–19.
15. Mirvis DM, Goldberger AL. Electrocardiography. In: Libby P, Bonow RO, Mann DL, Zipes DP, eds. Braunwald's Heart Disease. A Textbook of Cardiovascular Medicine. 8th ed. Philadelphia: Saunders Elsevier;2008:149 193.
16. Colluoglu T, Tanriverdi Z, Unal B, Ozcan EE, Dursun H, Kaya D. The role of baseline and post-procedural frontal plane QRS-T angles for cardiac risk assessment in patients with acute STEMI. Ann Noninvasive Electrocardiol. 2018;23(5):e12558. (10. 1111/anec. 12558)
17. Jennum, Pc, and Renata L. Riha. "Epidemiology of sleep apnoea/hypopnoea syndrome and sleep-disordered breathing. " European Respiratory Journal 33. 4(2009):907–914.
18. American Academy of Sleep Medicine. "International classification of sleep disorders—third edition (ICSD-3). " AASM Resour Libr 281(2014):2313.
19. Riha, Renata L. "Diagnostic approaches to respiratory sleep disorders. " Journal of thoracic disease 7. 8(2015):1373.
20. Hou H, Zhao Y, Yu W, Dong H, Xue X, Ding J, Xing W, Wang W. Association of obstructive sleep apnea with hypertension: A systematic review and meta-analysis. J Glob Health. 2018;8(1):010405.
21. Foster GE, Brugniaux JV, Pialoux V, Duggan CT, Hanly PJ, Ahmed SB, Poulin MJ. Cardiovascular and cerebrovascular responses to acute hypoxia following exposure to intermittent hypoxia in healthy humans. J Physiol. 2009;587(Pt 13):3287–99.
22. Dudenbostel T, Calhoun DA. Resistant hypertension, obstructive sleep apnoea and aldosterone. J Hum Hypertens. 2012;26(5):281–7.
23. Loube DI, Loube AA, Erman MK. Continuous positive airway pressure treatment results in weight less in obese and overweight patients with obstructive sleep apnea. J Am Diet Assoc. 1997;97(8):896–7.
24. Peppard PE, Young T, Palta M, Dempsey J, Skatrud J. Longitudinal study of moderate weight change and sleep-disordered breathing. JAMA. 2000b;284(23):3015–21.
25. Tasali E, Pamidi S, Covassin N, Somers VK. Obstructive Sleep Apnea and Cardiometabolic Disease: Obesity, Hypertension, and Diabetes. Circ Res. 2025;137(5):764–787.
26. Fathima S, Ahmed M. Data driven phenotyping and longitudinal feature modeling of sleep apnea subtypes using interpretable machine learning. Sleep Epidemiology. 2025;5:100113.
27. Patel N, Donahue C, Shenoy A, Patel A, El-Sherif N. Obstructive sleep apnea and arrhythmia: a systemic review. Int J Cardiol. 2017;228:967–970.