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Sleep-disordered breathing and nocturnal hypoxemia in patients followed for pulmonary hypertension

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ABSTRACT

Aims: To describe sleep-disordered breathing and nocturnal hypoxemia in patients followed for pulmonary hypertension (PH), and explore associations with cardiopulmonary characteristics.

Methods: This retrospective cross-sectional study examined 32 adults who underwent polysomnography (PSG) during PH care between August 2008 and September 2014. Detailed PSG data were available for 29 patients. The Apnea-Hypopnea Index (AHI) quantified respiratory events per hour of sleep. Nocturnal hypoxemia was defined as mean sleep oxygen saturation ≤88%. Mann-Whitney U and Fisher exact tests were used.

Results: Twenty-five patients (78.1%) were women; mean age was 58.3±14.5 years. Among patients with PSG data, 20 (69.0%) had AHI ≥5/h, 10 (34.5%) AHI ≥15/h, and seven (24.1%) AHI ≥30/h. Nocturnal hypoxemia occurred in 12 patients (41.4%) and was associated with a larger right ventricular diameter (median 4.05 vs 3.20 cm; p=0.002). Hypoxemia was more frequent in pulmonary arterial hypertension than other PH groups (66.7% vs 23.5%; p=0.029). Patients with AHI ≥15/h had higher body-mass index and lower minimum sleep oxygen saturation (p=0.005 and p=0.029).

Conclusion: In patients undergoing PSG during PH follow-up, sleep-disordered breathing affected approximately two-thirds and nocturnal hypoxemia two-fifths. Hypoxemia was associated with larger right ventricular diameter. These findings support considering nocturnal oxygenation alongside AHI in clinically selected patients, particularly those with right ventricular enlargement. Prospective studies should clarify whether hypoxemia predicts subsequent right ventricular remodeling.

Keywords: Hypertension, pulmonary, sleep apnea syndromes, hypoxia, polysomnography

INTRODUCTION

Pulmonary hypertension (PH) occurs in association with diverse cardiac, pulmonary, and vascular conditions and affects right heart structure and function. Assessment requires identification of the underlying cause and accompanying conditions that may influence symptoms and functional capacity.[1] Sleep-disordered breathing and impaired nocturnal oxygenation are among the conditions encountered in patients with PH.[2,3]

The Apnea-Hypopnea Index (AHI), which quantifies respiratory event frequency during sleep, may not capture all aspects of nocturnal oxygenation. Patients with PH may have reduced nocturnal oxygen saturation in association with apneas and hypopneas or without a substantial respiratory event burden. Studies involving pulmonary arterial hypertension (PAH) and other PH groups have reported frequent sleep-related breathing abnormalities, although estimates vary with patient selection and definitions.[2-5] Hypoxia can increase pulmonary vascular tone and right ventricular afterload, providing a physiological basis for examining nocturnal oxygenation in relation to right ventricular size.[4]

Recent studies have linked the duration and severity of nocturnal hypoxemia to pulmonary hemodynamics and right ventricular characteristics, mainly in selected populations with obstructive sleep apnea, PAH, or chronic thromboembolic pulmonary disease.[6-8] The relationship between low mean sleep oxygen saturation and recorded right ventricular size in clinically selected patients spanning all PH groups is less well characterized. This study re-evaluated historical records of patients who underwent polysomnography (PSG) during PH follow-up to describe sleep-disordered breathing and nocturnal hypoxemia and explore their cardiopulmonary associations. Daytime oxygenation and six-minute walk distance (6MWD) were examined to place nocturnal findings in the context of baseline gas exchange and functional capacity.

METHODS

Ethics

This study was approved by the Dokuz Eylül University Ethics Committee for Non-interventional Researches (Date: 29.11.2013, Decision No: 2013/43-03). All procedures were

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conducted by the ethical standards of the Declaration of Helsinki.

Study Design and Patients

This single-center retrospective cross-sectional study re-evaluated records from a joint pulmonology and cardiology PH clinic at a university hospital. The record period extended from the establishment of the PH clinic in August 2008 through September 2014. PSG had been performed during PH follow-up in patients with clinical suspicion of a sleep-related breathing disorder. Patients were eligible if they were adults, had a documented clinical diagnosis of PH under follow-up, underwent PSG during the specified period, and had available clinical records. Records of 32 patients were reviewed; PSG-based analyses included the 29 patients with available detailed PSG parameters. Previously established PH diagnoses and etiological classifications in the follow-up records were retained. Historical diagnoses were not independently revalidated or reclassified using current hemodynamic criteria.

The clinical classification comprised 13 patients with group 1 PAH, nine with group 2 PH, six with group 3 PH, three with group 4 PH, and one with group 5 PH. Right heart catheterization was available in 24 patients, including all 13 patients with PAH. Catheterization had not been performed in the other eight patients because of technical reasons or refusal of consent. Given the small sample, etiological comparisons contrasted group 1 PAH with the remaining PH groups combined.

Clinical and Cardiopulmonary Assessment

Age, gender, height, body weight, and body-mass index (BMI) were recorded. Systolic pulmonary artery pressure (sPAP), right ventricular diameter, and tricuspid annular plane systolic excursion (TAPSE) were extracted from echocardiographic records. Right ventricular diameter was analyzed in centimeters as documented on the original echocardiographic data collection form. The available records did not specify the echocardiographic window, anatomical measurement level, or cardiac phase. Mean pulmonary artery pressure (mPAP) and right atrial pressure were obtained from catheterization records. 6MWD was used as a measure of exercise capacity. The arterial partial pressure of oxygen (PaO_2) measured on room air before PSG was recorded.

Polysomnographic Assessment

PSG recordings included electroencephalography, electrooculography, submental electromyography, electrocardiography, oronasal airflow, thoracoabdominal respiratory movements, and pulse oximetry. Recorded apnea and hypopnea counts, durations of N1, N2, N3, and rapid eye movement sleep, and mean and minimum sleep oxygen saturation (SpO_2) were evaluated. Total sleep time was obtained by summing the durations of the sleep stages. AHI was calculated by dividing the total number of apneas and hypopneas by total sleep time in hours. Respiratory events were taken from the existing scoring records; raw PSG signals were not rescored.

AHI was categorized as <5, 5 to <15, 15 to <30, and ≥30 events/h. AHI ≥5/h was used as the PSG-based threshold for sleep-disordered breathing, and AHI <15/h and ≥15/h groups were used for clinical comparisons. Obstructive and central event subtypes were not analyzed separately. Nocturnal hypoxemia was defined as mean sleep SpO_2 ≤88% to retain the classification used in the original thesis study. This study-specific definition describes low average nocturnal oxygenation; it does not quantify the duration spent below 88% saturation or the frequency of desaturation events.

Statistical Analysis

Data were analyzed using IBM SPSS Statistics version 22.0 (IBM Corp., Armonk, NY, USA). Continuous variables are presented as mean±standard deviation or median (25th–75th percentile), and categorical variables as counts and percentages. Given the small sample and variable distributions, two-sided Mann-Whitney U tests were used for independent-group comparisons and Fisher exact tests for categorical comparisons. Wilson 95% confidence intervals (CIs) were calculated for the principal proportions.

Missing values were not imputed; each analysis included patients with the relevant available measurements. PSG analyses included 29 patients, 6MWD assessment 28, PaO_2 assessment 31, and descriptive catheterization assessment 24. The numbers of valid observations for each comparison are reported in the tables. Statistical significance was defined as $p<0.05$. No adjustment for multiple comparisons or multivariable modeling was performed; all group comparisons were interpreted as exploratory.

RESULTS

Patient Characteristics and PSG Findings

Of the 32 patients, 25 (78.1%) were women and seven (21.9%) were men. Mean age was 58.3±14.5 years and mean BMI was 30.6±7.2 kg/m². Median sPAP was 75.0 mmHg, right ventricular diameter 3.35 cm, and TAPSE 19.0 mm. Among the 24 patients with catheterization data, median mPAP was 42.0 mmHg. Clinical and PSG characteristics are summarized in **Table 1**.

Among the 29 patients with detailed PSG data, median total sleep time was 319.9 minutes, AHI 7.5 events/h, mean sleep SpO_2 91.2%, and minimum sleep SpO_2 76.0%. AHI ≥5/h was present in 20 patients (69.0%; 95% CI, 50.8%-82.7%), AHI ≥15/h in 10 (34.5%; 95% CI, 19.9%-52.7%), and AHI ≥30/h in seven (24.1%; 95% CI, 12.2%-42.1%). Nocturnal hypoxemia was present in 12 patients (41.4%; 95% CI, 25.5%-59.3%).

Comparisons According to Nocturnal Hypoxemia

The 12 patients with nocturnal hypoxemia had a larger right ventricular diameter than the 17 patients without hypoxemia: 4.05 (3.75-4.50) vs 3.20 (2.80-3.40) cm (p=0.002). Among patients with PSG data, nocturnal hypoxemia was present in eight of 12 patients with PAH (66.7%) and four of 17 patients in the other PH groups (23.5%; p=0.029). Age, BMI, sPAP, TAPSE, mPAP, right atrial pressure, 6MWD, PaO_2, and AHI did not differ significantly between the hypoxemia groups (**Table 2**).

Comparisons According to AHI

The 10 patients with AHI ≥15/h had a higher BMI than the 19 patients with AHI <15/h (median 33.7 vs 25.7 kg/m²; p=0.005). Minimum sleep SpO_2 was lower in the AHI

Table 1. Clinical and polysomnographic characteristics of the patients

Variable	Valid n	Value
Age, years	32	58.3±14.5
Female gender, n (%)	32	25 (78.1)
BMI, kg/m²	32	30.6±7.2
Group 1 PAH, n (%)	32	13 (40.6)
Group 2 PH, n (%)	32	9 (28.1)
Group 3 PH, n (%)	32	6 (18.8)
Group 4 PH, n (%)	32	3 (9.4)
Group 5 PH, n (%)	32	1 (3.1)
sPAP, mmHg	32	75.0 (60.0-91.2)
RV diameter, cm	32	3.35 (2.90-3.92)
TAPSE, mm	32	19.0 (15.3-22.0)
mPAP, mmHg	24	42.0 (37.2-47.5)
6MWD, m	28	380.0 (322.5-420.0)
PaO_2, mmHg	31	70.1 (62.7-79.0)
Total sleep time, min	29	319.9 (284.5-412.0)
AHI, events/h	29	7.5 (3.0-24.8)
Mean sleep SpO_2, %	29	91.2 (84.0-93.2)
Minimum sleep SpO_2, %	29	76.0 (67.0-83.0)
AHI <5/h, n (%)	29	9 (31.0)
AHI 5 to <15/h, n (%)	29	10 (34.5)
AHI 15 to <30/h, n (%)	29	3 (10.3)
AHI ≥30/h, n (%)	29	7 (24.1)
Nocturnal hypoxemia, n (%)	29	12 (41.4)

Age and BMI are presented as mean±standard deviation; other continuous variables are presented as median (25^{th}-75^{th} percentile). Percentages use the number of valid observations as the denominator. Nocturnal hypoxemia was defined as mean sleep SpO_2 ≤88%. PH: Pulmonary hypertension, PAH: Pulmonary arterial hypertension, BMI: Body-mass index, sPAP: Systolic pulmonary artery pressure, RV: Right ventricular, TAPSE: Tricuspid annular plane systolic excursion, mPAP: Mean pulmonary artery pressure, 6MWD: Six-minute walk distance, PaO_2: Arterial partial pressure of oxygen, AHI: Apnea-Hypopnea Index, SpO_2: Oxygen saturation by pulse oximetry

Table 2. Comparisons according to nocturnal hypoxemia

Variable	n[a]	Hypoxemia (n=12)	No hypoxemia (n=17)	p
Age, years	12/17	59.0 (43.0-64.8)	65.0 (55.0-70.0)	0.278
Female gender, n (%)	12/17	8 (66.7)	14 (82.4)	0.403
BMI, kg/m²	12/17	33.0 (26.9-35.8)	29.7 (24.4-33.3)	0.232
Group 1 PAH, n (%)	12/17	8 (66.7)	4 (23.5)	0.029
sPAP, mmHg	12/17	82.5 (70.0-100.0)	70.0 (60.0-85.0)	0.131
RV diameter, cm	12/17	4.05 (3.75-4.50)	3.20 (2.80-3.40)	0.002
TAPSE, mm	12/17	16.5 (12.8-21.2)	19.0 (17.0-22.0)	0.213
mPAP, mmHg	10/12	43.0 (39.0-57.8)	41.5 (34.2-44.0)	0.208
RA pressure, mmHg	10/12	10.0 (9.0-11.8)	8.0 (6.8-10.5)	0.207
6MWD, m	12/13	335.0 (322.5-385.0)	400.0 (240.0-420.0)	0.228
PaO_2, mmHg	12/16	64.9 (59.5-69.3)	74.0 (66.5-85.1)	0.082
AHI, events/h	12/17	10.4 (6.3-54.6)	6.9 (2.9-16.4)	0.177

Values are median (25th-75th percentile) or n (%). [a]Valid observations in the hypoxemia/no hypoxemia groups, respectively. Nocturnal hypoxemia was defined as mean sleep SpO_2 ≤88%. Continuous variables were compared using the Mann-Whitney U test and categorical variables using the Fisher exact test. P-values are unadjusted for multiple comparisons. Percentages in the group 1 PAH row describe the composition of the hypoxemia groups. Conversely. hypoxemia occurred in 8/12 patients with PAH (66.7%) and 4/17 patients in the other PH groups (23.5%). BMI: Body-mass index, PAH: Pulmonary arterial hypertension, PH: Pulmonary hypertension, sPAP: Systolic pulmonary artery pressure, RV: Right ventricular, TAPSE: Tricuspid annular plane systolic excursion, mPAP: Mean pulmonary artery pressure, RA: Right atrial, 6MWD: Six-minute walk distance, PaO_2: Arterial partial pressure of oxygen, AHI: Apnea-Hypopnea Index, SpO_2: Oxygen saturation by pulse oximetry

≥15/h group (median 69.0% vs 81.0%; p=0.029). There were no statistically significant differences in age, sPAP, right ventricular diameter, TAPSE, mPAP, 6MWD, PaO_2, or mean sleep SpO_2 (**Table 3**).

Table 3. Comparisons according to Apnea-Hypopnea Index

Variable	n[a]	AHI ≥15/h (n=10)	AHI <15/h (n=19)	p
Age, years	10/19	66.0 (61.0-67.8)	55.0 (42.0-67.5)	0.142
BMI, kg/m²	10/19	33.7 (32.6-40.5)	25.7 (23.4-32.0)	0.005
sPAP, mmHg	10/19	70.0 (65.5-85.0)	80.0 (60.0-97.5)	0.696
RV diameter, cm	10/19	3.55 (3.35-3.77)	3.20 (2.85-4.10)	0.505
TAPSE, mm	10/19	19.0 (17.2-19.8)	18.0 (14.0-22.0)	0.713
mPAP, mmHg	7/15	43.0 (40.5-50.0)	42.0 (36.5-48.0)	0.697
6MWD, m	7/18	330.0 (270.0-335.0)	390.0 (340.0-420.0)	0.106
PaO_2, mmHg	10/18	66.7 (56.8-70.5)	71.3 (62.4-77.5)	0.350
Mean sleep SpO_2, %	10/19	88.4 (85.2-91.6)	91.5 (83.2-93.3)	0.614
Minimum sleep SpO_2, %	10/19	69.0 (66.2-77.8)	81.0 (73.5-85.5)	0.029

Values are median (25^{th}-75^{th} percentile). [a]Valid observations in the AHI ≥15/h and AHI <15/h groups, respectively. Comparisons used the Mann-Whitney U test. P-values are unadjusted for multiple comparisons. AHI: Apnea-Hypopnea Index, BMI: Body-mass index, sPAP: Systolic pulmonary artery pressure, RV: Right ventricular; TAPSE: Tricuspid annular plane systolic excursion, mPAP: Mean pulmonary artery pressure, 6MWD: Six-minute walk distance, PaO_2: Arterial partial pressure of oxygen, SpO_2: Oxygen saturation by pulse oximetry

DISCUSSION

The principal observation was the association of study-defined nocturnal hypoxemia with a larger recorded right ventricular diameter in patients undergoing PSG during PH follow-up. This small, heterogeneous cohort allows the structural finding to be viewed alongside respiratory event burden, daytime oxygenation, and exercise capacity. These findings support prospective assessment of nocturnal oxygenation and right ventricular structure across PH etiologies.

The frequency of nocturnal hypoxemia in our PSG sample (41.4%) was lower than in several earlier series. Ulrich et al.[2] reported nocturnal desaturation in 68% of 38 patients with PAH or chronic thromboembolic PH, while Minic et al.[4] reported it in 60% of 52 patients with group 1 PAH; both used oxygen saturation <90% for more than 10% of the night or total sleep time. Jilwan et al.[3] reported nocturnal hypoxemia in 82.6% of 46 patients with idiopathic PAH or chronic thromboembolic PH. These estimates differ in patient selection, PH composition, and hypoxemia definitions from our mean sleep SpO_2 ≤88% criterion. A low mean saturation and time below a saturation threshold describe different aspects of oxygenation, limiting direct comparison. Yüce et al.[9] reported obstructive sleep apnea syndrome in 88.2% and nocturnal desaturation in 60.8% of 51 patients with group 1 or group 4 PH. Our AHI-based sleep-disordered breathing frequency (69.0%) also includes events without separation into obstructive and central subtypes.

Huang et al.[6] studied 205 patients with obstructive sleep apnea undergoing right heart catheterization, 134 of whom had PH. The percentage of time spent with SpO_2 <90% (T90) was associated with mPAP, pulmonary vascular resistance, and right ventricular stroke work index after adjustment for confounders. Lowery et al.[7] studied 186 patients with group 1 PAH and found that sleep-related hypoxia was more consistently associated than AHI with right ventricular

impairment and adverse outcomes. The association we observed with right ventricular diameter is directionally consistent with these observations, but concerns a structural measurement rather than demonstrated ventricular dysfunction. In contrast to these larger studies, neither TAPSE nor pulmonary artery pressures differed significantly between our hypoxemia groups. Differences in sample size, PH etiologies, oxygenation measures, and statistical adjustment may help explain the findings.

Han et al.[8] evaluated 77 patients with chronic thromboembolic pulmonary disease, including 44 with PH and 33 without PH. Both T90 and mean sleep SpO_2 were associated with mPAP and pulmonary vascular resistance after adjustment for AHI and daytime PaO_2, among other covariates. Their assessment of both mean oxygenation and hypoxemia duration provides context for our mean-saturation finding, although our smaller study identified a diameter difference rather than a pressure difference. Çınar et al.[5] studied 50 patients with chronic thromboembolic PH eligible for pulmonary endarterectomy. Severe nocturnal hypoxemia, defined by SpO_2 <90% for more than 20% of total sleep time, occurred in 62%; age, mPAP, and AHI were independent determinants. Their finding for AHI differs from our non-significant between-group comparison, emphasizing the relevance of PH subtype, hypoxemia definition, sample size, and analytic approach.

AHI did not differ significantly between patients with and without nocturnal hypoxemia (p=0.177; **Table 2**), and mean sleep SpO_2 did not differ significantly between the AHI <15/h and ≥15/h groups (p=0.614; **Table 3**). Together, these observations suggest that respiratory event frequency alone may incompletely characterize nocturnal oxygenation in this population. Mean sleep saturation reflects overall oxygenation across the night, whereas AHI counts discrete respiratory events. This distinction is consistent with the complementary information provided by hypoxemia measures in previous studies.[6-8] The clinical question is therefore how nocturnal oxygenation adds to AHI when characterizing patients with PH.

Patients with nocturnal hypoxemia also had numerically lower daytime PaO_2 than those without hypoxemia (median 64.9 versus 74.0 mmHg), although the difference was not statistically significant (p=0.082; **Table 2**). Low mean sleep SpO_2 may therefore partly reflect lower baseline oxygenation, with additional sleep-related changes in ventilation and gas exchange. Jilwan et al.[3] identified ventilation-perfusion mismatch, alone or together with obstructive events, as an important contributor to nocturnal hypoxemia. In 163 patients with chronic thromboembolic PH, Taniguchi et al.[10] linked nocturnal desaturation to physiological dead space and ventilation-perfusion mismatch; nocturnal desaturation improved after balloon pulmonary angioplasty but persisted despite substantial hemodynamic improvement. These findings provide plausible context for our observations. Distinguishing baseline impairment from an additional nocturnal component requires paired daytime and overnight measurements with appropriate adjustment.

The higher BMI and lower minimum sleep SpO_2 in patients with AHI ≥15/h indicate that respiratory event burden remains relevant to individual desaturation episodes. The contrast between minimum and mean sleep SpO_2 may help explain why AHI groups differed in the lowest recorded saturation but not in average nocturnal oxygenation. Pulmonary artery pressures and right ventricular diameter did not differ significantly between the AHI groups, consistent with the absence of differences in most hemodynamic variables by obstructive sleep apnea status in the series by Yüce et al.[9]

Although 6MWD was numerically lower in patients with nocturnal hypoxemia, the difference was not statistically significant; only 25 patients had both measurements available. Murta et al.[11] studied 52 patients with precapillary PH and found an association between exercise-induced desaturation and sustained nocturnal hypoxemia in multivariable analysis. Walking distance and exercise desaturation reflect different aspects of functional assessment. Future studies incorporating both could better characterize the relationship between nocturnal gas exchange and exercise performance.

The ESC/ERS guidelines recommend overnight oximetry or PSG when sleep-disordered breathing or hypoventilation is suspected.[1] Within this clinical setting, our findings support attention to nocturnal oxygenation alongside AHI, particularly when right ventricular enlargement or impaired daytime oxygenation raises concern about additional gas-exchange abnormalities. Prospective studies should combine duration-based measures such as T90 with the oxygen desaturation index, which measures desaturation frequency, and standardized right ventricular imaging. Analyses should account for PH etiology, AHI, daytime oxygenation, and treatment. Longitudinal and interventional studies could then determine whether nocturnal hypoxemia precedes adverse right ventricular remodeling and whether its correction modifies that process.

Limitations

This was a single-center record-based analysis of a small sample with heterogeneous PH etiologies. The findings describe patients who underwent PSG during PH follow-up and cannot be directly generalized to the broader PH population. Data from 2008-2014 may not fully represent contemporary patient profiles or treatment approaches. Diagnoses were based on follow-up records and were not independently revalidated. The absence of catheterization in eight patients, unequal distribution of PH groups, and missing measurements limit interpretation.

AHI was calculated from recorded event counts and sleep-stage durations; raw PSG signals were not rescored. Supplemental oxygen use during PSG, treatments, and the intervals between investigations could not be evaluated systematically. The absence of documentation of the echocardiographic window, anatomical level, and cardiac phase for right ventricular diameter limits verification of measurement standardization. The mean saturation threshold retained from the original study is study-specific and is not a universal diagnostic or treatment threshold; it does not quantify hypoxemia duration or desaturation frequency. Neither adjustment for confounders nor correction for multiple comparisons was performed. The greater representation of PAH in the hypoxemia group and possible differences in baseline oxygenation may have contributed to

the right ventricular diameter association. Nonsignificant AHI comparisons do not establish independence from apnea burden, particularly in a small sample. These exploratory cross-sectional findings do not establish causality or justify universal screening. Mortality, hospitalization, and treatment response were not evaluated, precluding conclusions about prognosis or therapeutic benefit.

CONCLUSION

In this clinically selected cohort encompassing all PH groups, PSG identified frequent nocturnal hypoxemia, particularly among patients with PAH, and an association with larger recorded right ventricular diameter. Considering nocturnal oxygenation alongside AHI may provide a fuller description of sleep-related abnormalities in patients undergoing assessment during PH follow-up. Prospective studies using duration-based oximetry measures, adjustment for daytime oxygenation and apnea burden, and standardized right ventricular imaging are needed to clarify the clinical significance and temporal direction of this association.

ETHICAL DECLARATIONS

Ethics Committee Approval

This study was approved by the Dokuz Eylül University Ethics Committee for Non-interventional Researches (Date: 29.11.2013, Decision No: 2013/43-03).

Informed Consent

The requirement for written informed consent was waived by the ethics committee because of the retrospective design and the use of anonymized data. No additional intervention was performed, and there was no direct patient contact.

Peer Review Process

This manuscript was subject to external peer review.

Conflict of Interest

The authors declare no conflicts of interest related to this study.

Financial Disclosure

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Author Contributions

Concept: CS; Design: CS; Control: CS, Oİ; Validation: CS; Resources: ÖK; Materials: Oİ; Data Collection and/or Processing: ÖK; Analysis and/or Interpretation: Oİ; Visualization: ÖK; Literature Review: ARB; Article Writing: ÖK, ARB; Critical Review: All Authors.

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