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OBSTRUCTIVE SLEEP APNEA: A CLINICAL AND RADIOLOGICAL PERSPECTIVE

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ABSTRACT

Background: Obstructive Sleep Apnea (OSA) is a common underdiagnosed sleep-related breathing disorder characterized by frequent upper airway obstruction during sleep with intermittent hypoxia and sleep fragmentation. **Objectives:** The aim of this study is to explore OSA from both clinical and radiologic points of view, with emphasis placed on the correlation between symptomatology, anthropometric measurements, and imaging parameters to enhance diagnostic precision and treatment planning. **Materials and Methods:** A 12-month cross-sectional observational study was conducted among 100 suspected OSA patients. There was comprehensive clinical evaluation, polysomnography, and radiological imaging namely cephalometry and upper airway CT scan. Radiological findings were correlated with Apnea-Hypopnea Index (AHI) scores and clinical presentations such as snoring, daytime sleepiness, and BMI. **Result:** Most patients presented with typical symptoms such as noisy snoring, non-restorative sleep, and day hypersomnia. Radiological imaging of characteristic homogeneous findings with regard to reduced retropalatal and retroglossal spaces of airways, elevated soft palate thickness, and high mandibular plane angles in moderate and severe OSA patients was seen. There existed a significant association between these anatomical features and AHI severity supporting the application of imaging in the stratification of disease. **Conclusion:** OSA is linked with a characteristic clinical presentation and structural changes of describable anatomy that can be optimally assessed using targeted imaging modalities. Correlation of radiologic variables with clinical and polysomnographic data can enhance diagnostic efficiency and facilitate individualized therapeutic management.

INTRODUCTION

Obstructive Sleep Apnea (OSA) is a chronic sleep-disordered breathing disorder characterized by periodic partial or complete closure of the upper airway during sleep, resulting in non-uniform ventilation, intermittent hypoxemia, hypercapnia, and arousals from sleep.[1] This condition results in striking changes in sleep architecture and quality of sleep, generating a variable extent of clinical manifestations ranging from obstructive snoring and hypersomnolence to major systemic consequences like hypertension, arrhythmias, insulin resistance, cognitive dysfunction, and cardiovascular risk.[2]
OSA occurs in almost one billion individuals worldwide and is emerging rapidly due to rising obesity levels, physical inactivity, and rising age.[2]
The pathophysiology of OSA is also multifactorial and has been described as the interplay of upper airway anatomical narrowing, blunted dilatory tone of the pharyngeal dilator muscles during sleep, increased collapsibility of the soft tissues, and neural regulation abnormalities of upper airway patency.[3]
Patients usually present with a variety of symptoms, which are habitual snoring, non-restorative sleep, witnessed apneas, morning headaches, dry mouth, irritability, poor concentration, and hypersomnolence during the day. However, due to the co-existence of these symptoms with some other common conditions such as insomnia, depression, and chronic fatigue syndrome, OSA tends to be under-diagnosed, particularly in the outpatient settings and primary care.[4]
Polysomnography (PSG) remains the diagnostic gold standard for OSA and provides objective information in the form of the Apnea-Hypopnea Index (AHI), oxygen desaturation level, sleep stage, and effort of breathing. Although PSG provides functional

information about sleep disorders of breathing, it does not lead to direct visualization of anatomical sites responsible for upper airway obstruction. Thus, imaging modalities such as lateral cephalometry, computed tomography (CT), and magnetic resonance imaging (MRI) have become clinically relevant over the past few years by providing visualization of site-specific airway constriction and soft tissue abnormalities.[5]

Radiologic examination of the upper airway in OSA is valuable in disease pathogenesis and also forms a beneficial addition to patient-individualized treatment planning. Reduced retropalatal and retroglossal airway space, soft palate thickening, hyoid bone position inferior to normal, macroglossia, and mandibular position variation or craniofacial anatomy anomalies are common findings.[6] These anatomical parameters are highly correlated with disease severity as measured by AHI and are typically used to stratify patients for non-invasive vs. surgical management. Cephalometric analysis, in particular, has been reported to be helpful in assessing craniofacial risk factors for OSA, especially in non-obese subjects or in atypical presentations.[7]

Despite these developments, diagnosis for OSA remains primarily symptom-based with no concern for structural factors that probably would influence treatment outcome. An approach combining clinical-radiological could be the missing diagnosis link, enhance early detection and optimize decision on management. Detection of radiological parameters of airway obstruction, especially in high-risk patients, is more specific and personalized approach whether in choosing continuous positive airway pressure (CPAP), positional therapy, mandibular advancing devices, or maxillofacial surgery.

Thus, in the current study, it is the aim to study OSA from an integrative clinical and radiologic perspective by assessing symptom profiles, anthropometric data, polysomnographic findings, and upper airway imaging findings in subjects with sleep-disordered breathing. By using a multidimensional evaluation methodology, this investigation aims to better understand the anatomical and functional etiology of OSA, underscore the association between radiologic data and clinical severity, and consolidate the position of imaging as a secondary diagnostic method in current sleep medicine.

MATERIALS AND METHODS

Study Design and Duration

A prospective cross-sectional observational study was conducted over a period of 12 months, from January 2023 to December 2023. The study aimed to evaluate both clinical and radiological features in patients with suspected Obstructive Sleep Apnea (OSA).

Study Setting

The research was undertaken in the Pulmonary Medicine and Radiology departments of a South Indian tertiary care teaching hospital, where there was one sole-purpose sleep laboratory, full-night polysomnography (PSG) equipment, and modern radiological imaging equipment for diagnosis.

Sample Size and Selection Criteria

In all, 100 patients between the age group of 18 and 65 years having symptoms suggestive of OSA were enrolled in the study in accordance to a predefined selection protocol. Inpatient and outpatient referrals based on screening questionnaires such as the STOP-BANG and Epworth Sleepiness Scale (ESS) were used as indicators of moderate to high risk for OSA.

Inclusion Criteria

- Adults with clinical features suggestive of OSA (e.g., habitual snoring, witnessed apneas, excessive daytime sleepiness).
- STOP-BANG score ≥ 3 and/or ESS score ≥ 10.
- Consent to undergo overnight polysomnography and radiological imaging.

Exclusion Criteria

- Known history of central sleep apnea or mixed apnea.
- Structural abnormalities of the face/neck due to trauma or surgery.
- Neuromuscular diseases affecting upper airway musculature.
- Refusal to participate or contraindications to imaging studies (e.g., CT).

Clinical Evaluation

All the patients enrolled were to thoroughly and clinically evaluated for detailed history, physical examination, BMI, neck circumference, and questionnaire-based screening of daytime sleepiness and snoring severity. Comorbidities like hypertension, type 2 diabetes, and dyslipidemia were also recorded.

Polysomnography (PSG)

All subjects underwent full-night attended in-lab polysomnography using standard American Academy of Sleep Medicine (AASM) protocols. Parameters recorded included:

- Apnea-Hypopnea Index (AHI)
- Oxygen desaturation index (ODI)
- Total sleep time
- Sleep efficiency
- Sleep stage distribution

OSA was defined and classified based on AHI:

- Mild: AHI 5–14 events/hour
- Moderate: AHI 15–29 events/hour
- Severe: AHI ≥30 events/hour

Radiological Evaluation

Radiological assessment of the upper airway was performed using:

1. **Lateral Cephalometry** – to assess craniofacial parameters such as soft palate length, mandibular plane angle, and posterior airway space.

2. **CT Imaging (Head and Neck)** – to evaluate three-dimensional anatomy of the airway including retropalatal, retroglossal, and hypopharyngeal spaces, tongue volume, and soft tissue hypertrophy.

Images were analyzed by two independent radiologists blinded to the PSG results. Measurements were standardized and recorded for comparison with clinical and polysomnographic data.

Data Management and Statistical Analysis

Collected data were compiled using Microsoft Excel and analyzed with SPSS software (Version 22.0). Continuous variables were expressed as mean ± SD and categorical variables as percentages. Pearson's correlation coefficient and chi-square test were used to assess associations between clinical symptoms, AHI severity, and radiological parameters. A p-value < 0.05 was considered statistically significant.

RESULTS

The study included 100 adult patients with clinically suspected Obstructive Sleep Apnea (OSA), evaluated between January 2023 and December 2023. The mean age of the study population was 47.3 ± 9.2 years, with a male predominance (71%). Obesity and increased neck circumference were common anthropometric findings. Polysomnographic analysis revealed that 24% of patients had mild OSA, 39% had moderate OSA, and 37% had severe OSA, based on Apnea-Hypopnea Index (AHI) criteria.

Radiological analysis using cephalometry and CT imaging revealed consistent anatomical abnormalities in moderate-to-severe OSA groups, including reduced posterior airway space, elongated soft palate, and increased tongue volume. A statistically significant correlation was observed between AHI and radiological parameters such as soft palate thickness, mandibular plane angle, and retropalatal space. Clinical symptoms such as excessive daytime sleepiness, loud snoring, and non-refreshing sleep were reported in over 80% of subjects and showed a positive association with AHI severity. Misalignment between cephalometric markers and clinical symptoms was more frequent in cases of mild OSA. The integration of clinical, polysomnographic, and imaging data allowed for improved classification and interpretation of OSA severity patterns.

Table 1: Age and Gender Distribution of the Study Population

Age Group (Years)	Male (n)	Female (n)	Total (n)
18–30	5	3	8
31–40	11	6	17
41–50	20	8	28
51–60	24	11	35
> 60	11	1	12
Total	**71**	**29**	**100**

Table 1 highlights the demographic profile of the study participants, showing a higher incidence of OSA among males, particularly in the age group of 41–60 years.

Table 2: Clinical Symptoms Reported Among Participants

Symptom	Frequency (n)	Percentage (%)
Loud snoring	88	88%
Daytime fatigue/somnolence	81	81%
Witnessed apnea	59	59%
Morning headaches	42	42%
Dry mouth upon waking	33	33%

Table 2 shows the frequency of clinical symptoms suggestive of OSA, with loud snoring and daytime fatigue being the most common complaints.

Table 3: Body Mass Index (BMI) and OSA Severity Correlation

BMI Category (kg/m²)	Mild OSA	Moderate OSA	Severe OSA	Total
< 25 (Normal)	8	2	1	11
25–29.9 (Overweight)	10	14	9	33
≥ 30 (Obese)	6	23	27	56

Table 3 compares BMI ranges with OSA severity, revealing a strong association between obesity and moderate to severe OSA.

Table 4: Neck Circumference and AHI Severity

Neck Circumference (cm)	Mild OSA	Moderate OSA	Severe OSA	Total
< 38	7	5	2	14
38–40	11	10	6	27
> 40	6	24	29	59

Table 4 shows that patients with neck circumference >40 cm are more likely to have moderate-to-severe OSA.

Table 5: Polysomnographic Classification of OSA Severity

OSA Severity	AHI Range (events/hr)	Number of Patients	Percentage (%)
Mild	5–14	24	24%
Moderate	15–29	39	39%
Severe	≥ 30	37	37%

Table 5 categorizes patients based on their AHI values, highlighting that the majority fall into moderate or severe OSA categories.

Table 6: Cephalometric Findings Across OSA Severity Levels

Parameter (Mean ± SD)	Mild OSA	Moderate OSA	Severe OSA
Soft Palate Length (mm)	34.2 ± 2.1	38.5 ± 2.4	41.6 ± 2.7
Posterior Airway Space (mm)	10.8 ± 1.2	8.4 ± 1.1	6.1 ± 1.0
Mandibular Plane Angle (°)	25.3 ± 1.5	29.1 ± 2.0	32.7 ± 2.3

Table 6 compares cephalometric parameters and shows significant variations in airway dimensions correlating with OSA severity.

Table 7: CT-Based Retropalatal and Retroglossal Space Measurements

Region	Mild OSA (mm)	Moderate OSA (mm)	Severe OSA (mm)
Retropalatal space	8.2 ± 0.9	6.1 ± 0.7	4.3 ± 0.6
Retroglossal space	9.5 ± 1.0	7.4 ± 0.8	5.2 ± 0.7

Table 7 depicts radiological measurements of airway segments, showing a consistent decrease in airway caliber in severe OSA cases.

Table 8: Correlation Between AHI and Radiological Parameters

Parameter	Correlation Coefficient (r)	Significance (p-value)
Retropalatal space	-0.72	< 0.001
Retroglossal space	-0.68	< 0.001
Soft palate thickness	+0.65	< 0.001

Table 8 summarizes statistical correlations, confirming significant inverse relationships between AHI and various upper airway dimensions.

Table 9: Epworth Sleepiness Scale (ESS) Scores vs OSA Severity

ESS Score Range	Mild OSA	Moderate OSA	Severe OSA	Total
< 10	9	5	1	15
10–15	13	20	16	49
> 15	2	14	20	36

Table 9 shows that ESS scores increase proportionally with OSA severity, supporting its clinical predictive value.

Table 10: Distribution of AHI Values by Gender

Gender	Mild OSA	Moderate OSA	Severe OSA	Total
Male	15	27	29	71
Female	9	12	8	29

Table 10 reveals a higher prevalence of moderate-to-severe OSA among male participants, consistent with known epidemiological trends.

Table 11: Associated Comorbidities in OSA Patients

Comorbidity	Mild OSA	Moderate OSA	Severe OSA	Total
Hypertension	4	15	23	42
Type 2 Diabetes	3	10	19	32
Dyslipidemia	2	7	11	20
GERD	1	3	5	9

Table 11 outlines the common comorbid conditions observed, with hypertension and diabetes being most frequent among severe OSA cases.

Table 12: Correlation Between Clinical Symptoms and Radiological Findings

Clinical Symptom	Retropalatal Space (mm)	Soft Palate Length (mm)
Loud snoring	5.0 ± 1.1	41.8 ± 2.5
No snoring or mild snoring	8.9 ± 1.2	35.1 ± 2.0
Daytime sleepiness present	5.6 ± 1.0	40.9 ± 2.2
Daytime sleepiness absent	8.1 ± 1.1	36.2 ± 2.3

Table 12 shows that radiological narrowing is more prominent in patients reporting severe snoring and excessive daytime sleepiness.

Table 1: Age and Gender Distribution of the Study Population: This table illustrates the demographic breakdown of the study cohort, with a clear male predominance (71%) and the highest prevalence of OSA occurring in the 41–60 years age group. These findings align with existing

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epidemiological data suggesting age and male gender as major risk factors.

Table 2: Clinical Symptoms Reported Among Participants: The table lists the key symptoms reported by patients. Loud snoring (88%) and excessive daytime sleepiness (81%) were the most prevalent, reinforcing their importance in initial clinical screening and risk stratification for OSA.

Table 3: Body Mass Index (BMI) and OSA Severity Correlation: This table highlights a strong relationship between increasing BMI and OSA severity. A notable 56% of patients with BMI ≥30 kg/m² had moderate or severe OSA, indicating obesity as a prominent modifiable risk factor.

Table 4: Neck Circumference and AHI Severity: Larger neck circumference was associated with more severe OSA. Among patients with neck circumference >40 cm, the majority (53/59) had moderate or severe disease, confirming its predictive value in physical examination.

Table 5: Polysomnographic Classification of OSA Severity: This table classifies the study population based on Apnea-Hypopnea Index (AHI). Most patients fell into the moderate (39%) and severe (37%) categories, suggesting that referrals were largely made for symptomatic or high-risk individuals.

Table 6: Cephalometric Findings Across OSA Severity Levels: Cephalometric measurements demonstrated a trend toward anatomical compromise with increasing OSA severity. Reduced posterior airway space and increased soft palate length and mandibular angle were characteristic of severe OSA.

Table 7: CT-Based Retropalatal and Retroglossal Space Measurements: The CT analysis further confirmed narrowing of the upper airway, especially in the retropalatal and retroglossal regions. These structural changes were most prominent in patients with severe OSA and are key markers of anatomical obstruction.

Table 8: Correlation Between AHI and Radiological Parameters: A statistically significant inverse correlation was observed between AHI and retropalatal/retroglossal spaces. A positive correlation with soft palate thickness was also evident, reinforcing the role of imaging in assessing OSA severity.

Table 9: Epworth Sleepiness Scale (ESS) Scores vs OSA Severity: ESS scores were proportionally aligned with AHI severity. Patients with severe OSA had higher ESS scores, supporting the use of this simple questionnaire in evaluating daytime dysfunction due to sleep apnea.

Table 10: Distribution of AHI Values by Gender: This table shows that men exhibited higher AHI values compared to women. Nearly 80% of male patients had moderate or severe OSA, further supporting gender-specific screening and diagnostic approaches.

Table 11: Associated Comorbidities in OSA Patients: Hypertension, type 2 diabetes, and dyslipidemia were more common among moderate-to-severe OSA patients, indicating a clear link between OSA and systemic metabolic and cardiovascular disorders.

Table 12: Correlation Between Clinical Symptoms and Radiological Findings: Radiological findings like decreased retropalatal space and augmented soft palate length were more egregious in patients with usual OSA complaints, favoring the anatomical cause of clinical presentations.

DISCUSSION

Obstructive Sleep Apnea (OSA) is still one of the most underdiagnosed sleep-related disorders globally, even though it has a firm link with systemic and metabolic illnesses. The aim of this research was to fill the gap between radiological and clinical approaches to OSA by examining the relationship between patient symptomatology, anthropometric measures, polysomnographic severity, and radiological findings. The findings from this study confirm established patterns of OSA pathophysiology and emphasize the importance of imaging modalities in complementing the clinical and polysomnographic assessment.[8]

Demographically, the results showed a definite predominance in males (71%) and the highest incidence in the 41–60-year age group, a reflection of the known literature. Increasing age and male gender are established non-modifiable risk factors for OSA due to variations in fat distribution, upper airway morphology, and hormonal effect. This is consistent with previous reports that postmenopausal women catch up with men in the prevalence of OSA but men continue to be inappropriately affected earlier in life.[9]

Clinically, the symptoms most commonly reported were loud snoring, daytime sleepiness, and witnessed apneas. These classic symptoms were extremely common (reported by >80% of participants), emphasizing their diagnostic potential. The application of validated clinical screening instruments such as the STOP-BANG and Epworth Sleepiness Scale (ESS) served to enhance the subjective burden of disease and facilitate correct referral for polysomnography. Consistent increase in ESS scores with higher Apnea-Hypopnea Index (AHI) again confirmed the use of ESS as a straightforward and low-resource tool in both primary and specialist care.[10]

Body Mass Index (BMI) and neck circumference anthropometric measurements were significant predictors of the severity of OSA. Over 80% of patients with moderate-to-severe OSA also had a BMI ≥30 kg/m², re-emphasizing obesity as a key modifiable risk factor. Neck circumference of more than 40 cm was also linked with more severe disease, a result that is consistent with the hypothesis that extra neck soft tissue causes narrowing and collapsibility of the upper airway.[11] Such results are

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important in informing public health and lifestyle interventions aimed at reducing OSA risk.

Polysomnographic assessment allocated a large percentage of the study population to moderate-to-severe OSA. This could be an artifact of referral bias to more symptomatic patients; nevertheless, it also supports the necessity of greater awareness and proactive screening in patients with even mild sleep-related symptoms. As the study has documented established associations between OSA and hypertension, diabetes, cardiovascular disease, and dyslipidemia, early diagnosis and stratification are indispensable for mitigating long-term consequences.[12]

Radiological assessment provided insightful structural information. Cephalometric analysis demonstrated an increasing decrease in posterior airway space and rising soft palate length and mandibular plane angles in relation to worsening OSA severity. These observations indicate that some craniofacial morphologies predispose patients to airway collapse during sleep. CT scans offered additional confirmation, with narrowing of retropalatal and retroglossal spaces being most significant in severe OSA cases.[13] These anatomical alterations not only elucidate the pathogenesis of airway obstruction but also direct the appropriateness of directed interventions such as mandibular advancement devices or surgical airway alteration procedures.

Statistically significant correlation was found between AHI values and several radiological parameters, most notably decreased airway space and soft palate thickness. This further supports imaging's not only diagnostic but also severity evaluation and treatment planning role. In addition, radiological narrowing was most pronounced in patients with severe snoring and excessive daytime sleepiness, which indicates that imaging can support anatomical verification of symptom source.[14]

A notable finding was the correlation of moderate-to-severe OSA with comorbidities including hypertension and type 2 diabetes mellitus. These correlations underpin the systemic inflammatory and oxidative stress hypotheses related to untreated OSA. Intermittent hypoxia and sleep disruption are likely to result in endothelial dysfunction, insulin resistance, and increased sympathetic activity accounting for the augmented cardiovascular and metabolic burden seen in this group.[15]

Although strengths of the study reside in its integrated clinical, polysomnographic, and radiologic design, some limitations must be appreciated. Sample size, although sufficient for initial analysis, potentially restricts the applicability of the findings. Although CT imaging has superior spatial resolution, it involves subjecting patients to ionizing radiation and might not be practical for regular screening. MRI, although radiation-free, could not be utilized due to economic and availability factors. Additionally, longitudinal follow-up to evaluate the effect of interventions on clinical and radiological parameters was not within the scope of this investigation.

Finally, this investigation reiterates the need for a multi-dimensional assessment strategy in OSA diagnosis and treatment. Clinical evaluation will always have a place, but radiological information gives invaluable anatomical background that can help tailor therapy. By mapping structural irregularities to functional sleep parameters and symptomatology, a more specific and personalized strategy to OSA management can be formulated. The future should look at utilizing dynamic sleep imaging and combining machine learning algorithms for predictive modeling in the diagnostics and treatment of OSA.

CONCLUSION

This study reaffirms that Obstructive Sleep Apnea is a multifactorial disease with strong clinical, anatomical, and polysomnographic correlates. The combination of radiological and clinical assessment improves diagnostic sensitivity and provides indispensable information on the anatomical mechanisms of upper airway obstruction. Posterior airway space, length of soft palate, and retropalatal diameter are significant radiological parameters that correlate with severity of OSA, indicating their role in patient stratification and treatment strategy. A multi-disciplinary, patient-tailored strategy that includes clinical symptoms, findings from sleep studies, and imaging can largely enhance the early detection, risk stratification, and individualized therapeutic interventions in OSA. Larger cohorts and dynamic imaging during sleep in future studies may further define our knowledge and enhance outcomes in this prevalent but underdiagnosed disorder.

REFERENCES

1. Jordan AS, McSharry DG, Malhotra A. Adult obstructive sleep apnoea. Lancet. 2014 Feb 22;383(9918):736-47. doi: 10.1016/S0140-6736(13)60734-5. Epub 2013 Aug 2. PMID: 23910433; PMCID: PMC3909558.
2. Bouzerda A. Risque cardiovasculaire et syndrome d'apnées obstructives du sommeil [Cardiovascular risk and obstructive sleep apnea]. Pan Afr Med J. 2018 Jan 18; 29:47. French. doi: 10.11604/pamj.2018.29.47.11267. PMID: 29875929; PMCID: PMC5987146.
3. Lévy P, Kohler M, McNicholas WT, Barbé F, McEvoy RD, Somers VK, Lavie L, Pépin JL. Obstructive sleep apnoea syndrome. Nat Rev Dis Primers. 2015 Jun 25; 1:15015. doi: 10.1038/nrdp.2015.15. PMID: 27188535.
4. Bonsignore MR, Saaresranta T, Riha RL. Sex differences in obstructive sleep apnoea. Eur Respir Rev. 2019 Nov 6;28(154):190030. doi: 10.1183/16000617.0030-2019. PMID: 31694839; PMCID: PMC9488655.
5. Arnaud C, Bochaton T, Pépin JL, Belaidi E. Obstructive sleep apnoea and cardiovascular consequences: Pathophysiological mechanisms. Arch Cardiovasc Dis. 2020 May;113(5):350-358. doi: 10.1016/j.acvd.2020.01.003. Epub 2020 Mar 26. PMID: 32224049.
6. Savini S, Ciorba A, Bianchini C, Stomeo F, Corazzi V, Vicini C, Pelucchi S. Assessment of obstructive sleep apnoea (OSA) in children: an update. Acta Otorhinolaryngol Ital. 2019 Oct;39(5):289-297. doi: 10.14639/0392-100X-N0262. PMID: 31708576; PMCID: PMC6843580.

ISSN (O): 2687-5365; ISSN (P): 2753-6556

7. Challamel MJ, Beydon N, Coutier L, Launois S, Seailles T, Vecchierini MF, Franco P. Critères diagnostiques des troubles respiratoires obstructifs du sommeil de l'adolescent [Diagnostic criteria for obstructive sleep apnea syndrome in adolescent]. Rev Mal Respir. 2021 Oct;38(8):829-839. French. doi: 10.1016/j.rmr.2021.06.006. Epub 2021 Sep 23. PMID: 34565640.
8. Thurnheer R. Obstructive sleep apnea and cardiovascular disease - time to act! Swiss Med Wkly. 2007 Apr 21;137(15-16):217-22. doi: 10.4414/smw.2007.11671. PMID: 17525876.
9. Kent BD, Ryan S, McNicholas WT. Obstructive sleep apnea and inflammation: relationship to cardiovascular co-morbidity. Respir Physiol Neurobiol. 2011 Sep 30;178(3):475-81. doi: 10.1016/j.resp.2011.03.015. Epub 2011 Mar 23. PMID: 21439407.
10. Thoonsen H, van der Zeijden J, Hoogeboom PN, Copper MP, Schure PJCM, Teunissen LL. Obstructieve slaapapneu [Obstructive sleep apnoea]. Ned Tijdschr Geneeskd. 2019 May 3;163: D3399. Dutch. PMID: 31120225.
11. Malhotra A, White DP. Obstructive sleep apnoea. Lancet. 2002 Jul 20;360(9328):237-45. doi: 10.1016/S0140-6736(02)09464-3. PMID: 12133673.
12. Fisher E, Youngs R, Hussain M, Fishman J. A focus on paediatric obstructive sleep apnoea. J Laryngol Otol. 2018 Apr;132(4):283. doi: 10.1017/S002221511800049X. PMID: 29722643.
13. Gillerot G, Jadoul M. Obstructive sleep apnoea. Lancet. 2002 Dec 21-28;360(9350):2079. doi: 10.1016/S0140-6736(02)11965-9. PMID: 12504435.
14. Deflandre E, Brichant JF. Obstructive sleep apnoea and postoperative complications: A significant link? Anaesth Crit Care Pain Med. 2019 Oct;38(5):431-433. doi: 10.1016/j.accpm.2019.06.010. Epub 2019 Jun 27. PMID: 31255834.
15. Wei DYW, Chew M, Sabanayagam C. Obstructive Sleep Apnoea, Other Sleep Parameters and Diabetic Retinopathy. Curr Diab Rep. 2021 Dec 13;21(12):58. doi: 10.1007/s11892-021-01425-0. PMID: 34902078.