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Health consequences of Obstructive Sleep Apnoea Syndrome

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Introduction:
Obstructive Sleep Apnoea Syndrome (OSAS) is a common sleep disorder characterized by recurrent episodes of partial or complete upper airway obstruction during sleep. OSAS affects millions of people worldwide, with varying degrees of severity. It contributes to a range of adverse health outcomes, including cardiovascular, neurocognitive, metabolic, and psychosocial effects. Understanding the wide-ranging impact of OSAS is crucial in emphasizing the importance of early recognition, accurate diagnosis, and tailored interventions to improve the quality of life for affected individuals.

Aim of the study:
The aim of this study is to explore the diverse health consequences of OSAS and highlight the importance of early recognition, accurate diagnosis, and tailored interventions for managing this sleep disorder effectively. By understanding the impact of OSAS on various aspects of health, we can emphasize the need for comprehensive evaluation and a multidisciplinary approach to improve the overall health of individuals affected by OSAS.

Material and methods:
Literature available in the PubMed database was reviewed using the following keywords: “Obstructive sleep apnoea”; “Obstructive sleep apnoea pathophysiology”; “Obstructive sleep

apnoea complications"; "Obstructive sleep apnoea epidemiology"; "Cardiovascular consequences of Obstructive Sleep Apnoea"; "Obstructive sleep apnoea treatment".

Conclusions:

OSAS is a prevalent sleep disorder with significant health consequences. It affects various systems within the body, particularly the cardiovascular system, leading to an increased risk of cardiovascular complications such as coronary artery disease, heart failure, arrhythmias, and stroke. Early diagnosis and appropriate treatment of OSAS are crucial in reducing the health burden and improving overall quality of life for affected individuals.

Keywords: "Obstructive sleep apnoea"; "Obstructive sleep apnoea pathophysiology"; "Obstructive sleep apnoea complications"; "Obstructive sleep apnoea epidemiology"; "Cardiovascular consequences of Obstructive Sleep Apnoea"; "Obstructive sleep apnoea treatment".

Obstructive sleep apnoea syndrome

Obstructive sleep apnea syndrome (OSAS) is the most frequent sleep-related breathing disorder, characterized by repeated episodes of upper airway obstruction during sleep, resulting in disruption of normal ventilation. [1] Moreover, this syndrome is associated with major adverse health outcomes, including increased cardiovascular risk and neurocognitive deficits. [2] Repetitive pharyngeal collapse during sleep causes respiratory disturbances, which manifest as partial reductions (hypopnea) and complete pauses (apnea) in ventilation, leading to oxygen desaturation and sleep fragmentation. [3] The obstruction is usually caused by the relaxation of the muscles in the throat, leading to airway collapse or narrowing. [4] OSAS presents with symptoms such as loud snoring, choking episodes during sleep, and excessive daytime sleepiness. Morning headaches, fatigue, and irritability are common, along with difficulty concentrating. Medical evaluation including a sleep study is crucial for diagnosis and management. [3]

Etiology and pathogenesis

The pathophysiology of OSAS is multifactorial, and the mechanisms remain incompletely understood. The upper airway, particularly in the region behind the tongue and soft palate, has relatively little bony or rigid support. Thus, dilator muscles and soft tissues are crucial for maintaining pharyngeal patency. With the onset of sleep, the activity of the pharyngeal dilating muscles is considerably reduced, which is one of the reasons for repetitive pharyngeal collapse. [5], [6] However, there are other equally important non-anatomic factors, including inadequate responsiveness of the upper-airway dilator muscles during sleep, waking up prematurely due to airway narrowing, and having an oversensitive ventilatory control system. [4] The causes of OSAS vary among patients. Functional alterations, such as obesity, are correlated with OSAS. [7] Moreover, most patients with obstructive apnea have anatomically small pharyngeal airways. [8] Additionally, factors such as alcohol consumption, sedative medications, and smoking can exacerbate the condition by further relaxing the muscles and increasing the likelihood of airway collapse during sleep. These factors collectively result in repeated episodes of partial or complete obstruction of the upper airway during sleep, leading to the characteristic symptoms of OSAS. [1]

Diagnosis and classification

The American Association of Sleep Medicine (AASM) has released third edition of the International Classification of Sleep Disorders (ICSD) which defines the clinical and sleep testing criteria for OSAS. [9] The diagnosis requires five or more predominantly obstructive respiratory events (obstructive and mixed apneas, hypopneas, or respiratory effort-related arousals) per hour of sleep during polysomnography (PSG) and at least one of the following symptoms (associated sleepiness, fatigue, insomnia, snoring, subjective nocturnal respiratory disturbance, or observed apnea) or an associated medical or psychiatric disorder (hypertension, coronary artery disease, atrial fibrillation, congestive heart failure, stroke, diabetes, cognitive dysfunction, or mood disorder). The presence of 15 or more respiratory events per hour of sleep is sufficient for a diagnosis, despite the lack of clinical symptoms. [10]

The apnoea-hypopnea index (AHI) is a measurement used in sleep medicine to quantify the severity of OSAS. It represents the total number of apneas (complete pauses in breathing)

and hypopneas (partial reductions in breathing) that occur per hour of sleep during PSG. A higher AHI indicates a more severe form of sleep apnea, while a lower AHI suggests a milder condition. It is a key parameter used by healthcare professionals to diagnose and classify the severity of sleep-disordered breathing. [1]

Epidemiology

OSAS is most common among older males, but it can also affect females and children. [11] Studies reveal OSAS is prevalent and can vary based on many factors including location, lifestyle, and individual health conditions. The estimated prevalence in United States is approximately 15 to 30 percent in males and 10 to 15 percent in females, when OSAS is defined broadly as an AHI greater than five events per hour of sleep. When more stringent definitions are used (e.g., AHI ≥5 events per hour plus symptoms or AHI ≥15 events per hour), the estimated prevalence is approximately 15 percent in males and 5 percent in females. [12] The prevalence of OSAS in Poland is not very well documented. However, a study by Pływaczewski et al. estimated the prevalence of OSAS in Poland at 7.5% based on a group of 676 patients from Warsaw. [13] In Poland, as in many other countries, OSAS is recognized as a significant health concern; however, more studies are necessary to define the actual epidemiology of OSAS.

Polysomnography

The diagnosis of OSAS can be made during a sleep study – polysomnography (PSG). PSG is a fundamental diagnostic tool used in the evaluation of sleep disorders. It involves the simultaneous recording of sleep staging, eye movements, electromyographic tone, respiratory parameters, and electrocardiogram. PSG is particularly helpful in the assessment of sleep-disordered breathing and its management, propensity for excessive sleepiness, complex behaviors during sleep, including motor disturbances of sleep, sleep-related epilepsy, and parasomnias. [14] OSAS is the leading cause of referral to sleep laboratories worldwide, accounting for at least 75–80% of diagnoses. The global prevalence of OSAS was recently estimated at up to 1 billion people worldwide. Even using a stricter definition (based on AHI > 15/h) there are still up to 500 million estimated cases worldwide. The current definitions of sleep apnea are not uniform, but most of them attempt to characterize the frequency of sleep-disordered breathing events (e.g., AHI “Apnea–Hypopnea Index” or RDI “Respiratory

Disturbance Index") along with the severity (e.g., oxygen desaturation) of each event (e.g., complete (apnea) and partial (hypopnea) cessation of breathing during sleep). By convention, an apnea is defined as greater than 90% reduction of airflow for at least 10 s. A hypopnea is defined as a reduction in airflow that is followed by an arousal from sleep or a decrease in oxyhemoglobin saturation. While AHI is the most commonly used parameter to assess sleep apnea severity, several additional measures of sleep such as the degree of nocturnal oxyhemoglobin desaturation and the extent of carbon dioxide elevation have been used to characterize disease severity in clinical and research settings. [15]

PSG is a critical tool in the diagnosis and management of sleep disorders, including OSAS. It provides comprehensive data on a patient's sleep patterns, allowing for accurate diagnosis and effective treatment planning. As our understanding of sleep disorders continues to evolve, so too will the techniques and technologies used in PSG, promising even more precise and effective diagnostic capabilities in the future. [14], [15]

Obstructive sleep apnoea complications

OSAS is a complex sleep disorder with wide-ranging health consequences. From cardiovascular and metabolic derangements to neurocognitive dysfunction and psychosocial distress, OSAS can significantly impact an individual's overall well-being. Here, we will explore the primary health consequences of OSAS based on the latest data.

Cardiovascular Consequences

OSAS is an independent predictor for cardiovascular disorders. The American Heart Association and American College of Cardiology issued a joint statement in 2008 communicating that sleep apnea is the underlying cause of cardiovascular disease in some patients, whereas it exacerbates this pathological condition in others. Cardiovascular diseases associated with OSA include hypertension, heart failure, stroke, cardiac arrhythmias, myocardial ischemia and infarction, and pulmonary arterial hypertension. OSA is associated with metabolic dysregulation, such as insulin resistance and lipid disorders, which in turn is a risk factor for cardiovascular diseases. [16]

A study published in PLOS ONE investigated this association. The study included individuals who were 50 years of age or older, underwent overnight polysomnographic for the

evaluation of OSAS, and performed MRI and transcranial Doppler (TCD) within 12 months of interval without a neurological event between the evaluations. The study found that the mean middle cerebral artery resistance index ratio (MRIR), a parameter for cerebrovascular compliance, was associated with a higher respiratory distress index. However, the middle cerebral artery pulsatility index (MCA PI), another parameter for cerebrovascular compliance, was not associated with any of the PSG markers for OSAS severity. The AHI was associated with the log-transformed total white matter hyperintensity (WMH) volume, subcortical WMH volume, total enlarged perivascular space (ePVS) score, and centrum semiovale ePVS score. The oxygen-desaturation index was associated with periventricular WMH volume, independently from age, MCA PI, and MRIR. In conclusion, the study found that OSA is associated with reduced cerebrovascular compliance and also with SVD independently from cerebrovascular compliance. The underlying pathomechanistic link might be region-specific. This suggests that OSAS could promote the development of SVD through mechanisms related to reduced cerebrovascular compliance.

Hypertension

There is a high prevalence of hypertension among individuals with OSAS with 30% to 50% having both conditions. In resistant hypertension, up to 80% may have OSAS. [17] The repeated apneas cause fluctuations in blood oxygen levels, triggering a cascade of events that lead to increased sympathetic nervous activity and elevated blood pressure. [18] However, the effects of Continuous Positive Airway Pressure (CPAP) therapy on blood pressure (BP) in hypertensive patients with OSAS have been inconsistent, showing reductions of 2-3 mm Hg. [17] The chronic elevation in blood pressure can then further exacerbate the cardiovascular complications associated with OSAS, such as coronary artery disease and heart failure.

Coronary Artery Disease (CAD)

One of the most significant cardiovascular consequences of OSAS is the increased risk of developing and progressing coronary artery disease (CAD). CAD occurs when the blood vessels that supply the heart with oxygen and nutrients become narrowed or blocked by atherosclerosis. [17] OSAS contributes to the development and progression of atherosclerosis through various mechanisms. The intermittent hypoxia and hypercapnia experienced during

apneas lead to endothelial dysfunction, oxidative stress, and inflammation, all of which promote the formation and progression of atherosclerotic plaques. Additionally, the increased sympathetic nervous system activity and elevated blood pressure associated with OSAS contribute to the development of CAD. [1][17]

Heart Failure

OSAS is also strongly associated with heart failure (HF), both left and right-sided. HF occurs when the heart is unable to pump sufficient blood to meet the body's metabolic demands. This condition can result from structural or functional abnormalities of the heart, leading to inadequate cardiac output and/or elevated intracardiac pressures. [19] The pathophysiological ramifications of OSAS on HF are orchestrated through several mechanisms, encompassing neurohormonal activation, escalated oxidative stress and inflammation, acute elevation in preload and afterload stemming from pronounced intrathoracic pressure oscillations, and the exacerbation of systemic hypertension. [17]

Sleep-disordered breathing (SDB), including OSAS, is common in HF patients, affecting 50–80% of patients who have HF with reduced ejection fraction (HFrEF) and 55% in HF with preserved ejection fraction (HFpEF). [20] Numerous studies have revealed a high prevalence of OSAS in heart failure patients, particularly those with preserved ejection fraction. CPAP treatment has consistently improved the apnea-hypopnea index, left ventricular ejection fraction, oxygen saturation, and overall quality of life in patients with OSAS. Emerging evidence suggests that sodium-glucose cotransporter-2 inhibitors (SGLT2i) and sacubitril/valsartan might influence OSAS outcomes through weight loss, improved metabolic profiles, and potential direct effects on upper airway muscles. Sleep apnea, whether in the presence or absence of HF, is associated with a higher risk of negative cardiovascular outcomes, including aggravation of heart failure-related symptoms, increased hospitalizations, and higher mortality rates. [21]

Stroke

Stroke patients have a higher risk of OSAS, which can worsen their cognitive and functional disabilities, prolong their hospitalization, and increase their mortality rates. The incidence of OSAS in stroke patients increased from 61% in 2011 to 75% in 2019, a rate

significantly higher than the 35% found in the general population. Both the American Heart Association and the American Stroke Association recommend that the diagnosis and treatment of OSAS should be part of secondary prevention programs for stroke. [22] A study published in the European Journal of Medical Research investigated the association of OSAS with cardiovascular events in patients with acute coronary syndrome (ACS) with or without prior stroke. The study found that the presence of OSAS was associated with an increased risk of cardiovascular events in patients with prior stroke. The multivariate analysis showed that patients with OSAS had 2.0 times the risk of major adverse cardiovascular and cerebrovascular events (MACCEs) in the prior stroke group. [23] A study published in The Lancet Neurology discussed the current knowledge and future directions of sleep apnea and ischemic stroke. The study highlighted the importance of understanding the pathophysiological mechanisms linking OSAS and ischemic stroke, and the potential benefits of OSAS treatment for stroke prevention and recovery. [24]

Arrythmias

OSAS has been proven to be a risk factor for hypertension and vascular dysfunction and has been proposed to be causally related to cardiac arrhythmias and sudden cardiac death. Findings from cross-sectional studies suggest a high prevalence of cardiac arrhythmias in patients with OSAS and a high prevalence of OSAS in those with cardiac arrhythmias. Several mechanisms seem to underpin the association between OSAS and cardiac arrhythmias, including intermittent hypoxia associated with autonomic nervous system activation and increased oxidative stress, which may lead to cardiac cellular damage and alterations in myocardial excitability. Recurrent arousals result in sympathetic activation and coronary vasoconstriction. Increased negative intrathoracic pressure may mechanically stretch the myocardial walls and promote acute changes in myocardial excitability as well as structural remodeling of the myocardium. OSAS has mainly been associated with premature atrial complex short runs, sinus bradycardia, sinus pauses, premature ventricular complexes, and paroxysmal atrial fibrillation. Preliminary evidence from uncontrolled interventional studies suggests that the treatment of OSAS may prevent cardiac arrhythmias. [25]

Pulmonary Hypertension

OSAS is relatively common in patients with Pulmonary Hypertension (PH), especially in those with chronic thromboembolic pulmonary hypertension or PH associated with lung disease or hypoxia. The patients with OSAS were mostly male, older, and had lower daytime arterial oxygen pressure. Lower daytime arterial oxygen pressure is a risk factor for OSAS in older male patients with PH. This suggests that careful screening for OSAS in patients with PH, particularly those who are older, male, and have lower daytime arterial oxygen pressure, may be beneficial. As always, individual patient care should be guided by a healthcare professional. [26]

Metabolic disorders

OSAS and obesity have a bidirectional relationship. Obesity, particularly abdominal obesity, is a major risk factor for developing OSAS. Conversely, OSAS itself may promote weight gain and make weight loss more difficult. [27] What is more, OSAS has been independently associated with insulin resistance, even after adjusting for obesity and other confounding factors. The severity of OSAS correlates with the degree of insulin resistance. Intermittent hypoxia's effect on adipose tissue induces insulin resistance, marking the first step in the OSAS-diabetes link. Over the last few years, the relationship between OSAS and various metabolic disorders, especially type 2 diabetes (T2D), has become more evident. Studies have reported that the prevalence of prediabetes, assessed by insulin resistance and glucose intolerance, is higher in OSAS patients than in controls, with estimates ranging from 20% to 67%. Several cross-sectional studies have shown that OSAS impairs insulin sensitivity and glucose tolerance. It has been demonstrated that OSAS is independently related to the development of insulin resistance, with the oxygen desaturation index being the main determining factor. [28]

Several potential mechanisms have been proposed to explain the association between OSAS and insulin resistance. Firstly, OSAS is characterized by recurrent episodes of hypoxia (low oxygen levels) and reoxygenation. This intermittent hypoxia can lead to oxidative stress and inflammation, both of which can impair insulin signaling and result in insulin resistance. OSAS often results in fragmented sleep. Poor sleep quality and short sleep duration have been

associated with alterations in glucose metabolism and increased risk of insulin resistance. [29] The repeated episodes of apnea in OSAS lead to increased sympathetic nervous system activity, resulting in the release of stress hormones such as cortisol and adrenaline, which can interfere with insulin action and further contribute to insulin resistance. OSAS has also been associated with increased levels of inflammatory cytokines and adipocyte-derived factors, such as adiponectin, leptin, and resistin. These factors can contribute to systemic inflammation and insulin resistance. Furthermore, changes in Hypothalamic–Pituitary–Adrenal (HPA) axis activity have been observed in patients with OSAS. The HPA axis plays a crucial role in the regulation of glucose metabolism, and alterations in its activity can lead to insulin resistance. [28] It's important to note that these mechanisms are not mutually exclusive and likely interact in complex ways to contribute to the development of insulin resistance in individuals with OSAS. [28], [29]

OSAS may contribute to the development of dyslipidemia, a disorder of lipid metabolism. Studies have found that OSAS is associated with higher levels of triglycerides and LDL cholesterol, as well as lower levels of HDL cholesterol. [28] Dyslipidemia and redox imbalance are potential mechanisms linking OSAS with the development of vascular diseases. OSAS patients exhibited significantly higher levels of triacylglycerols, total cholesterol, and LDL-cholesterol compared to healthy controls. Among the LDL and HDL subfractions, OSAS patients showed significantly lower levels of atheroprotective LDL1 and large HDL subfractions, and significantly higher levels of atherogenic small dense LDL3–7 and HDL8–10 subfractions. Lipoperoxide levels in patients with OSAS were significantly elevated compared to those in healthy individuals. The lipoprotein pro-atherogenic phenotype was found in individuals with OSAS, characterized by increased levels of atherogenic lipoprotein subfractions and reduced levels of atheroprotective subfractions. [30] These findings suggest a complex interplay between OSAS and metabolic disorders. However, further research is needed to fully understand these relationships and their clinical implications.

Neurocognitive disorders

The impact of OSAS extends beyond cardiovascular health to neurocognitive functioning. A study published in BMC Psychiatry in 2024 conducted a two-sample bidirectional Mendelian randomization (MR) analysis to investigate the causal relationship between OSAS and a range of neurocognitive characteristics. The study found that OSAS

significantly increases the volume of the hippocampus. Nominally causal effects of OSAS on brain structures, such as the thickness of the temporal pole, amygdala structure change, and cerebellum white matter change covering lifespan, were observed. The same study also detected bidirectional causal links between brain cortical structure, brain subcortical structures, cognitive performance, and the risk of OSAS. Patients with OSAS may suffer from insomnia, excessive daytime sleepiness, tiredness, inattention, or headaches due to frequent occurrences of blocked airways. These cognitive deficits can affect academic and occupational performance, as well as daily functioning and quality of life. [31]

Another review summarized recent findings on the association between OSAS and processes related to Alzheimer's dementia (AD) and Parkinson's disease (PD). Associations between OSAS and alterations in grey and white matter, brain diffusivity, and deficits in memory, attention, and executive control were reported. Furthermore, OSAS was correlated with higher risks of developing AD and PD and associated pathophysiology. Treatment was found to alleviate but not reverse some of the damage. [32]

Association between OSAS and psychiatric disorders

The relationship between OSAS and psychiatric disorders is complex and multifaceted, warranting a comprehensive understanding for effective management. OSAS is common among patients with psychiatric disorders, particularly depression and posttraumatic stress disorder. The overlap of symptoms such as sleep issues, mood changes, and vegetative symptoms between OSAS and psychiatric disorders, as well as side effects of psychiatric medications, can make the recognition of OSAS in patients with psychiatric disorders challenging. Therefore, clinicians should not assume that all sleep-related symptoms are consequences of psychiatric illness or medication but should instead be cognizant of the potential for coexisting OSAS that requires treatment. Recognizing and managing OSAS in patients with psychiatric disorders are critical to improve response to treatment, quality of life, and overall health. [33]

Quality of Life and Psychosocial Conseequences

OSAS is a prevalent chronic disease that significantly impacts a patient's perception of health and quality of life (QoL). Patients with sleep apnea experience sleep-disrupting symptoms and daytime sleepiness/fatigue, which adversely impact physical, psycho-cognitive, and social aspects of their lives in complex, interactive ways. This can result in impaired social and relational functioning, including strained interpersonal relationships, social isolation, and loneliness. OSAS can also contribute to mood disorders such as depression and anxiety, as well as decreased libido and sexual dysfunction. The psychosocial consequences of OSAS can significantly impact an individual's mental health, relationships, and overall satisfaction with life. The use of CPAP therapy and other interventions to effectively manage OSAS can lead to improvements in sleep quality, daytime functioning, and overall quality of life. [34]

Treatment

The treatment landscape for OSAS is diverse, offering a range of options that can be tailored to the individual patient's needs and circumstances. The current standard of treatment for OSAS is Continuous Positive Airway Pressure (CPAP). CPAP therapy is highly effective in suppressing respiratory disturbances during sleep and improving several clinical manifestations. [35] However, not all patients tolerate CPAP well, necessitating the exploration of alternative therapies. The European Respiratory Society has provided guidelines on non-CPAP therapies for OSAS. These include gastric bypass surgery, custom-made dual-block mandibular advancement devices, hypoglossal nerve stimulation, myofunctional therapy, maxillo-mandibular osteotomy, carbonic anhydrase inhibitors, and positional therapy. Each of these therapies offers unique benefits and may be more suitable for certain patients based on their specific circumstances. [36] Behavioral interventions are another crucial aspect of OSAS treatment. These interventions include lifestyle changes such as weight loss, avoidance of alcohol and sedatives, smoking cessation, and positional therapy. These changes can significantly improve OSAS symptoms and overall health. Oral appliances, custom-made devices that fit in the mouth like a sports mouthguard or orthodontic retainer, are another treatment option. Nasal expiratory positive airway pressure, a treatment involving a small disposable device worn over the nostrils during sleep, is also used. Negative pressure interventions, which use negative pressure to help keep the airways open, offer another treatment avenue. Various surgical options are available as well, depending on the specific

anatomical abnormalities contributing to OSAS. [37] Surgical interventions, such as uvulopalatopharyngoplasty (UPPP) or maxillomandibular advancement (MMA), may be considered for patients who do not respond to conservative treatments. [38] Lastly, pharmaceutical interventions are being explored for the treatment of OSAS. While several medications have been studied, only a handful have demonstrated encouraging outcomes, such as the combination of noradrenergic and antimuscarinic drugs. While not yet a mainstay of treatment, drug therapies could offer additional options in the future. [39]

The treatment of OSAS is multifaceted and should be individualized, taking into account the severity of the patient's OSAS, their general health, and their personal preferences. It's also crucial to consider the potential benefits and risks of each treatment option. As our understanding of OSAS continues to evolve, so too will the treatment options available to patients. It is essential for healthcare providers to stay abreast of these developments to provide the best possible care for their patients. [40]

Conclusion

Obstructive Sleep Apnoea Syndrome (OSAS) is a medical condition that holds significant importance due to its wide-ranging health implications. This condition not only disrupts sleep patterns and leads to intermittent hypoxia but also has a profound impact on various bodily systems, particularly the cardiovascular system. The strong association between OSAS and cardiovascular issues, such as coronary artery disease, heart failure, arrhythmias, stroke, and hypertension, underscores the critical importance of early identification and treatment of this sleep disorder.

By comprehending the health impacts of OSAS, healthcare professionals can provide comprehensive care and improve outcomes for individuals grappling with this condition. It is crucial for healthcare providers to understand the intricate relationship between OSAS and its associated health complications to ensure effective management and treatment strategies. However, further investigation is necessary to delve deeper into the mechanisms underlying the connection between OSAS and its associated health complications. This will enable the development of more efficient treatments and interventions that can effectively address the multifaceted nature of OSAS and its impact on overall health and quality of life. Therefore, ongoing research and collaboration among healthcare professionals, researchers, and scientists are essential to advance our understanding of OSAS and its implications. By expanding our

knowledge in this field, we can enhance patient care, optimize treatment approaches, and ultimately improve the quality of life for individuals affected by OSAS.

Author's contribution

Conceptualization, Wiktoria Julia Krzesłowska and Kamila Szewczyk; methodology, Weronika Hołownia, Kamila Szewczyk, Paulina Pytel; software, Szymon Wiśniewski, Bartłomiej Szewczyk; check, Weronika Hołownia, Szymon Wiśniewski; formal analysis, Wiktoria Julia Krzesłowska and Paulina Pytel; investigation, Bartlomiej Szewczyk; resources, Szymon Wiśniewski, Bartłomiej Szewczyk, Weronika Hołownia; data curation, Weronika Hołownia, Szymon Wiśniewski; writing - rough preparation, Kamila Szewczyk; writing - review and editing, Kamila Szewczyk; project administration, Wiktoria Julia Krzesłowska, Bartłomiej Szewczyk.visualization, Kamila Szewczyk, Weronika Hołownia; supervision, Kamila Szewczyk, Paulina Pytel;All authors have read and agreed with the published version of the manuscript.

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