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Arterial Hypertension and Obstructive Sleep Apnea Syndrome

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Abstract: In modern cardiology, close attention has been paid to the role of sleep and its disturbances in the development of various somatic pathologies. Particular attention has been paid to obstructive sleep apnea syndrome (OSAS) and its relationship with cardiovascular diseases, particularly arterial hypertension (HTN) of various origins. OSA is a common but rarely diagnosed sleep disorder, affecting 5 to 10% of the population and characterized by cyclical collapse of the upper airway during sleep, leading to partial or complete cessation of breathing for 10 seconds or more. Numerous studies have shown that OSA is an independent risk factor for the development of hypertension. Due to the high prevalence of both hypertension and OSA, these two conditions often occur in the same patient. Approximately 50% of patients with sleep-disordered breathing have hypertension, and approximately 30% of patients with hypertension have OSA. This combination exacerbates the pathological changes inherent in each individual condition. This review can help practicing cardiologists and general practitioners address this comorbidity and understand the relationship between these conditions.

Keywords: Hypertension, Obstructive Sleep Apnea Syndrome, Blood Pressure, Apnea-Hypopnea Index, CPAP Therapy

Introduction

Arterial hypertension (HTN) remains one of the most prevalent cardiovascular disorders worldwide and is a leading modifiable risk factor for morbidity and mortality[1]. Despite the availability of effective pharmacological and non-pharmacological interventions, global blood pressure control remains insufficient, contributing significantly to cardiovascular complications such as stroke, myocardial infarction, and heart failure. The multifactorial nature of hypertension, involving both internal and external determinants, complicates its prevention and management, necessitating continuous investigation into emerging risk factors and associated conditions[2].

In recent years, increasing attention has been directed toward the role of sleep and sleep-related disorders in the development and progression of cardiovascular diseases[3]. Among these, obstructive sleep apnea syndrome (OSAS) has been identified as a significant but often underdiagnosed condition characterized by recurrent episodes of upper airway obstruction during sleep, resulting in intermittent hypoxia and sleep fragmentation. Epidemiological data indicate that OSAS affects approximately 5–10% of the population and frequently coexists with hypertension, suggesting a strong pathophysiological relationship between the two conditions[4].

Previous studies have established OSAS as an independent risk factor for hypertension, with evidence demonstrating a dose-response relationship between the severity of apnea and the likelihood of developing elevated blood pressure. Research such as the Wisconsin Cohort Study has shown that the risk of hypertension increases significantly with higher apnea-hypopnea index (AHI) values. Furthermore, a substantial proportion of patients with resistant hypertension have been found to have undiagnosed OSAS. However, despite these findings, the integration of sleep disorder assessment into routine hypertension management remains limited, indicating a gap between clinical evidence and practice[5].

The present study aims to analyze the relationship between arterial hypertension and obstructive sleep apnea syndrome, focusing on epidemiological associations, pathophysiological mechanisms, and clinical manifestations such as diurnal blood pressure variability. The methodological approach involves a comprehensive review and synthesis of clinical data, observational studies, and meta-analyses addressing the interaction between OSAS and hypertension. Particular attention is given to mechanisms including sympathetic nervous system activation, endothelial dysfunction, systemic inflammation, and hypoxia, which collectively contribute to blood pressure dysregulation. It is expected that this analysis will clarify the role of OSAS as a contributing factor in hypertension development[6].

The anticipated findings suggest that OSAS significantly influences both the onset and progression of hypertension, particularly through alterations in nocturnal blood pressure patterns and increased variability. These results have important clinical implications, highlighting the necessity of early diagnosis and integrated treatment

strategies, including continuous positive airway pressure (CPAP) therapy in combination with antihypertensive medications. Ultimately, improving the understanding of this comorbidity may enhance patient outcomes, reduce cardiovascular risk, and contribute to more effective management of hypertension in clinical practice[7].

Methodology

This study adopts a qualitative, analytical review design to investigate the relationship between arterial hypertension (HTN) and obstructive sleep apnea syndrome (OSAS) by synthesizing existing clinical, epidemiological, and pathophysiological evidenc[8]. The research is based on a systematic examination of published scientific literature, including observational studies, cohort studies, randomized clinical trials, and meta-analyses that address the comorbidity between hypertension and sleep-disordered breathing. Particular emphasis is placed on epidemiological data evaluating the prevalence and coexistence of HTN and OSAS, as well as on studies analyzing the association between the severity of apnea, measured by the apnea-hypopnea index (AHI), and blood pressure levels[9]. The methodological framework incorporates comparative analysis to assess variations in clinical manifestations, especially diurnal and nocturnal blood pressure patterns, including “dipper” and “non-dipper” profiles. In addition, pathophysiological mechanisms underlying the interaction between OSAS and hypertension are examined through the analysis of research focusing on sympathetic nervous system activation, endothelial dysfunction, systemic inflammation, intermittent hypoxia, and neurohumoral regulation. The study also reviews therapeutic approaches, particularly the effectiveness of continuous positive airway pressure (CPAP) therapy and antihypertensive treatment, by analyzing outcomes reported in clinical trials and meta-analyses[10]. Data interpretation is conducted through integrative synthesis, allowing for the identification of consistent patterns and clinically relevant conclusions. This comprehensive methodological approach provides a multidimensional understanding of the role of OSAS in the development and progression of hypertension and supports the formulation of evidence-based clinical implications[11].

Result and Discussion

Hypertension (HTN) is one of the most common cardiovascular diseases. The overall prevalence of hypertension in the adult population is approximately 30-45% and is steadily increasing. To date, a large number of well-tolerated, reliable, and effective interventions have been developed that focus on lifestyle modification and the use of medications for the correction of blood pressure and the treatment of hypertension. However, blood pressure control remains inadequate worldwide and is far from perfect in Europe. Despite existing effective guidelines for the management of patients with hypertension, it remains the leading modifiable cause of cardiovascular and overall mortality worldwide[12]. Therefore, ongoing research into the prevention and treatment of hypertension remains necessary. The development of hypertension is a complex process involving the interaction of numerous factors, both external and internal. The unknown number of these factors, as well as the dynamic nature of many of them, currently precludes the development of a comprehensive concept for the development of this formidable disease. However, with the emergence of new medical approaches and the reassessment of known but previously poorly understood conditions, it has become possible to gain a better understanding of this complex process and subsequently develop methods that will improve the quality of life of patients with this pathology[13].

In recent years, cardiology has focused on the role of sleep and its disturbances in the development of various somatic pathologies. Particular attention is being paid to obstructive sleep apnea syndrome (OSA) and its association with cardiovascular diseases, particularly hypertension of various etiologies. OSA is a common but underdiagnosed sleep disorder, affecting 5 to 10% of the population and characterized by cyclical collapse of the upper airway during sleep, leading to partial or complete cessation of breathing for 10 seconds or more[14].

Epidemiological Association of OSA and Hypertension

Data from numerous studies has demonstrated that OSA is an independent risk factor for the development of hypertension. Due to the high prevalence of both hypertension and OSA, these two pathologies often occur in the same patient. Approximately 50% of patients with sleep-disordered breathing suffer from hypertension, and approximately 30% of patients with hypertension have OSA, which is often undiagnosed. This combination exacerbates the pathological changes inherent in each individual disorder[15].

The Wisconsin Cohort Study found that the odds ratio for hypertension at 4-year follow-up was 1.4 (95% CI 1.1–1.8) for patients with mild OSA (AHI 0.1–4.9) at baseline compared with those without apnea, 2.0 (95% CI 1.3–3.2) for those with moderate OSA (apnea-hypopnea index (AHI) 5.0–14.9), and 2.9 (95% CI 1.5–5.6) for those with severe OSA (AHI ⩾15). These results indicate that the risk of developing hypertension is significantly associated with the underlying severity of OSA in a dose-response relationship. According to a study by Logan et al., in 41 patients with refractory hypertension (BP >140/90 mmHg), which did not respond to therapy with three or more medications, OSA was diagnosed in 83% of cases[16].

According to the 2004 Joint National Committee 7th Report on the Diagnosis, Evaluation, Prevention, and Treatment of High Blood Pressure (JNC 7), OSA is the leading cause of secondary hypertension. This demonstrates the prevalence and clinical significance of hypertension caused by OSA. However, despite these data, many Russian cardiologists and physicians still fail to consider this information in their clinical practice[17].

Diurnal BP Variability in OSA

Physiologically, both normal and elevated blood pressure decrease at night (a "dipping pattern"). In patients with OSA, blood pressure during sleep does not decrease (a "non-dipper") and may even exceed daytime blood pressure (a "night peaker"). More recent studies also confirm these results in the elderly. This blood pressure variability increases the risk of cardiovascular disease and the occurrence of major cardiovascular events during the night and early morning hours. However, studies assessing diurnal variations in biomarkers corresponding to OSA-induced BP changes have been inconclusive. Nevertheless, this line of research remains potentially valuable. A relatively new classification of sleep-related hypertension in OSA has been proposed: a stable type (both nocturnal and morning hypertension) and an overexertion type (morning hypertension only, without nocturnal hypertension). However, further rigorous clinical studies are needed to confirm the variability of these two types of hypertension[18].

Pathophysiological aspects of the relationship between OSA and hypertension

Most researchers assume that OSA is the cause of hypertension, which should be considered a form of secondary hypertension. It is difficult to identify confounding variables and establish a direct causal relationship between OSA and hypertension. The mechanisms by which OSA is thought to cause hypertension include sympathetic nervous system activation, endothelial dysfunction, increased endothelin levels, decreased nitric oxide, development of systemic inflammation, and hypoxia. The greatest peculiarities in BP behavior in OSA are observed during sleep[19]. “Pulsus paradoxus”, i.e., a decrease in inspiratory BP of ≥10 mmHg, occurs during labored breathing with strong respiratory efforts. Importantly, each termination of an episode of obstructive sleep apnea is characterized by a BP peak lasting only a few seconds, followed by a return of BP to or below the baseline level. Therefore, the higher the number of sleep apneas, the greater the number of BP fluctuations. However, the amplitude of BP peaks varies greatly depending on sympathetic reactivity, which in turn is associated with apnea, hypoxemia, the occurrence and strength of brain excitation at the end of each event, the patient's age, and the normotensive/hypertensive state. The speed and amplitude of BP peaks during sleep are fundamental determinants of the degree of BP variability during the night and the average change in BP from day to night[20] .

OSAHS Treatment – Effect on Blood Pressure

Currently, the gold standard for OSAHS treatment is CPAP therapy, which is administered to up to 80% of patients. Symptomatic and pathogenetic treatment of associated conditions is administered concurrently with OSAHS treatment. Information on hypertension therapy in patients with OSAHS is contradictory. In a comparative analysis of amlodipine, atenolol, enalapril, hydrochlorothiazide, and losartan, β-blockers produced a more pronounced reduction in blood pressure. Other authors have found that the use of different groups of blood pressure-controlling medications does not affect blood pressure variability during sleep in patients with OSAHS combined with hypertension, therefore, they argue that treatment of OSAHS is the only means of correcting blood pressure. Most authors believe that CPAP therapy should be used in combination with antihypertensive drugs when treating hypertension in patients with OSA. However, to date, there are no uniform rational recommendations for the management of such patients[21].

Several meta-analyses and randomized trials demonstrate an improvement in blood pressure with the use of CPAP. The most recent meta-analysis of 30 randomized trials included 1900 patients and showed an average decrease in blood pressure of 2.5 mmHg. The effect of lowering blood pressure is especially evident in patients with more severe OSA. Even a moderate decrease in blood pressure is significant when considered in the context of a 1-5 mmHg

decrease in blood pressure achieved by ACE inhibitors, which can reduce the risk of stroke, serious cardiovascular events, and death by 20% to 25% [22].

Conclusion

- Epidemiological data indicate that OSA is a modifiable and widespread factor in the development of hypertension.
- OSA should always be considered in patients with predominantly nocturnal and morning hypertension, especially if refractory to treatment.
- Treatment of hypertension in OSA usually requires both antihypertensive pharmacological therapy and treatment of apnea.
- Control of BP variability can help improve OSA management and reduce the risk of cardiovascular disease.

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