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Original Article

The diagnostic value of polysomnography in obstructive sleep apnea-hypopnea syndrome patients with hypertension and secondary atherosclerosis

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Abstract: Objective: To investigate the utility of polysomnography (PSG) in diagnosing obstructive sleep apnea-hypopnea syndrome (OSAHS) patients with hypertension who develop secondary atherosclerosis, and to identify effective indicators for predicting arterial sclerosis. Methods: 140 OSAHS patients with hypertension diagnosed in Jiujiang NO. 1 People's Hospital from January 2021 to December 2023 were enrolled in this retrospective study and divided into two groups based on the presence of arteriosclerosis: the control group (without arteriosclerosis) and the research group (with arteriosclerosis). Univariate analysis, Pearson correlation analysis, and multivariate logistic regression analysis were used to identify independent factors affecting arteriosclerosis. The diagnostic performance of these factors were evaluated using a receiver operating characteristic (ROC) curve analysis. Results: Compared with the control group, the research group showed significantly higher levels of fasting blood glucose (FBG), triglyceride (TG), low-density lipoprotein (LDL), apnea-hypopnea index (AHI), systolic blood pressure (SBP), and intima-media thickness (IMT), and a significantly lower level of minSpO_2 (P<0.05). In patients with OSAHS and hypertension, FBG, TG, LDL, AHI, SBP, and IMT were positively correlated with secondary atherosclerotic diseases (r=0.273, 0.249, 0.190, 0.294, 0.198, 0.506, all P<0.05), while minSpO_2 was negatively correlated with secondary atherosclerotic diseases (r=-0.199, P<0.05). FBG, TG, LDL, AHI, minSpO_2, SBP, and IMT were identified as independent risk factors for the development of atherosclerosis in patients with OSAHS and hypertension (P<0.05), with the area under the receiver operator characteristic curves of 0.668, 0.647, 0.636, 0.690, 0.636, 0.608, 0.805, and 0.922 for single and combined tests, respectively (P<0.05). Conclusion: The AHI and minSpO_2 from PSG can predict arteriosclerosis. Combining them with FBG, TG, LDL, SBP, and IMT improves the accuracy of risk assessment.

Keywords: Polysomnography, obstructive sleep apnea-hypopnea syndrome, hypertension, atherosclerosis

Introduction

Obstructive sleep apnea hypopnea syndrome (OSAHS) is a chronic sleep disorder characterized by recurrent episodes of complete or partial upper airway obstruction during sleep, leading to breathing pauses or insufficient airflow [1]. Its association with cardiovascular and metabolic diseases has drawn much attention. Epidemiological studies estimate that approximately 936 million people worldwide are affected by OSAHS, with comorbid hypertension observed in nearly 40% of cases [2, 3]. In OSAHS patients with concomitant hypertension, the intermittent hypoxia (IH) activates the nuclear factor kappa-B (NF-κB)/signal transducer and activator of transcription 3 (STAT3) signaling pathway, resulting in increased oxidative stress in vascular endothelial cells and inflammatory factors like interleukin 6 (IL-6) and tumor necrosis factor-α (TNF-α). Consequently, nitric oxide bioavailability decreases, affecting vascular smooth muscle cell proliferation and migration, and accelerating atherosclerosis [4, 5]. OSAHS-related sleep fragmentation triggers hyperactivity of the hypothalamic-pituitary-adrenal (HPA) axis, resulting in elevated nocturnal catecholamine secretion. This disrupts normal circadian blood pressure patterns, leading to structural vascular remodeling characterized by elastin fiber degradation, increased collagen deposition, and progressive atherosclerotic changes [6]. The insidious progression of OSAHS-induced atherosclerosis

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poses particular clinical challenges, as patients typically remain asymptomatic during early disease stages; however, advanced disease frequently manifests as severe cardiovascular and cerebrovascular complications that threaten patient health [7]. Therefore, the development of early warning systems incorporating multimodal physiological parameters is crucial for improving outcomes in hypertensive OSAHS patients at risk for atherosclerosis.

Polysomnography (PSG), the gold standard for OSAHS diagnosis, collects seven types of physiological signals: electroencephalogram (EEG), electro-oculogram (EOG), electromyogram (EMG), nasal airflow, thoracoabdominal movement, oxygen saturation (SpO_2), and limb movement. PSG provides a comprehensive assessment of sleep quality and OSAHS severity by quantifying frequency and duration of respiratory events, as well as their impact on sleep structure [8]. For instance, PSG detects apneas and hypopneas that cause intermittent SpO_2 desaturations and sleep fragmentation, contributing to cardiovascular dysfunction [9]. PSG-monitored alterations in sleep structure, including shifts in sleep-stage distribution and increased awakenings, exhibit strong correlations with OSAHS severity. These metrics are crucial for early identification of atherosclerosis, disease progression assessment, and prognosis prediction [10]. However, current research and clinical practice often focus on isolated PSG parameters, overlooking the interactive effects between multiple parameters. Therefore, this study aims to analyze the clinical characteristics of OSAHS patients with hypertension complicated by arteriosclerosis through PSG examination, identify predictive indicators for secondary arteriosclerosis, and evaluate the efficacy of PSG examination in assessing the risk of arterial stiffness in patients with OSAHS and hypertension.

Materials and methods

Case selection

The sample size calculation was performed using PASS 4.17 software (NCS, Kaysville, Utah, USA), with an unmatched case-control design (ratio =1:2) to investigate the association between OSAHS and hypertension, as well as secondary arteriosclerosis. Based on previous epidemiological data demonstrating a 30% co-occurrence rate of OSAHS among hypertensive patients, with an estimated odds ratio (OR) of 6.44 [11], we set the significance level at 0.05 and statistical power (1-β) at 0.90. Accounting for a potential 10% dropout rate, the required sample sizes were determined to be 88 cases for the control group and 44 cases for the research group (total minimum sample size of 132). A total of 168 patients who underwent PSG in the Jiujiang NO. 1 People's Hospital from January 2021 to December 2023 were selected from this retrospective study. After applying strict inclusion/exclusion criteria (**Figure 1**), 140 eligible hypertensive patients were enrolled. Participants were categorized into the research group and control group based on the presence of arteriosclerosis during hospitalization and 6-month follow-up [12].

Inclusion criteria: (1) Diagnosis of OSAHS according to the *Clinical Practice Guideline for the Diagnosis of Adult Obstructive Sleep Apnea*, defined as an apnea-hypopnea index (AHI) ≥15 events/h with typical symptoms (e.g., daytime sleepiness, nocturnal choking) [13]; (2) Diagnosis of hypertension based on the *2018 Chinese Guidelines for the Prevention and Treatment of Hypertension*, defined as repeated clinic blood pressure ≥140/90 mmHg [14]; (3) Concurrent diagnosis of both hypertension and OSAHS; (4) Age between 18-80 years; (5) Willingness to undergo complete polysomnography and related examinations; and (6) No medication affecting sleep-related breathing or blood pressure regulation had been taken in the 2 weeks before admission.

Exclusion criteria: (1) Patients with severe cardiopulmonary insufficiency; (2) Patients with a history of psychiatric disorders or a hereditary diseases; (3) Patients with a history of substance abuse or alcohol dependence; (4) Patients with a total sleep time <240 minutes during polysomnography; (5) Patients with incomplete clinical data. This study was approved by the Ethics Committee of Jiujiang NO. 1 People's Hospital.

Intervening method

Both patient groups received standard treatments for OSAHS and hypertension. For OSAHS management, therapeutic strategies included

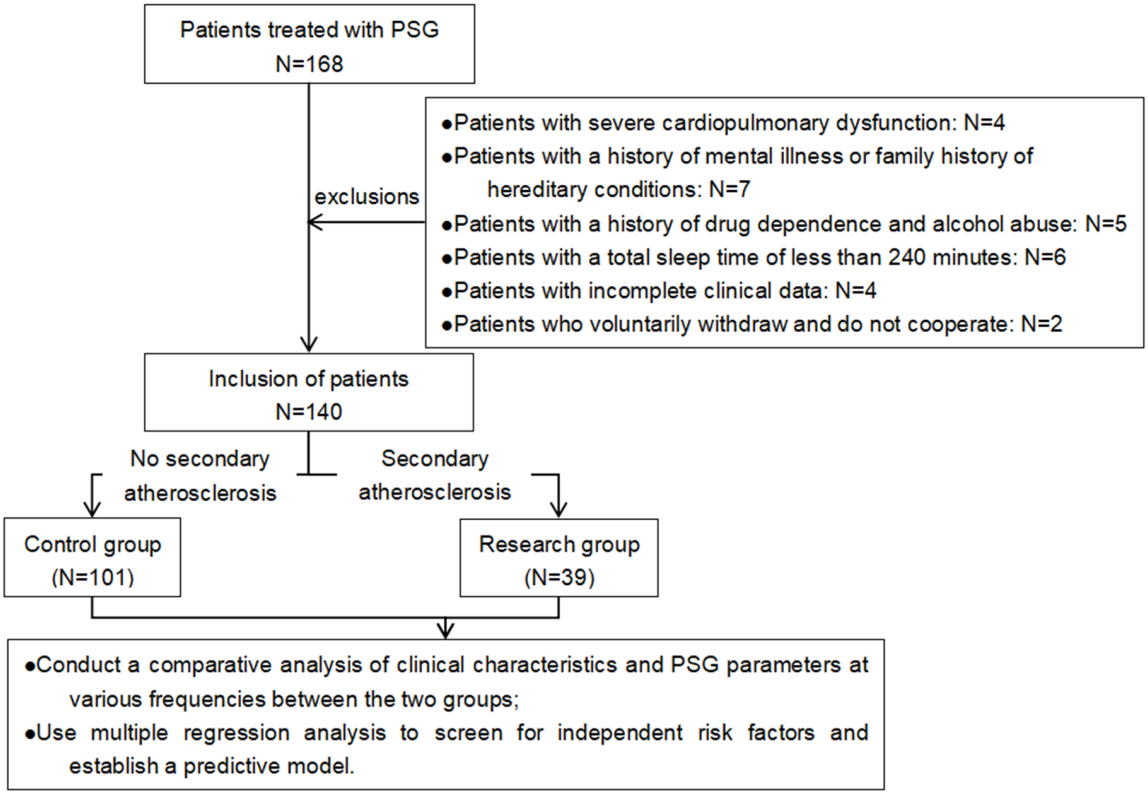

Figure 1. Study flow chart. Note: PSG, polysomnography.

continuous positive airway pressure (CPAP) therapy, oral appliance therapy, or surgical interventions, with individualized treatment plans formulated by specialists based on patients' clinical characteristics. Hypertension management encompassed pharmacological and non-pharmacological approaches. The medication regimen included evidence-based use of diuretics, calcium channel blockers, angiotensin-converting enzyme inhibitors, and other antihypertensive agents, tailored by clinicians according to patients' blood pressure profiles, comorbid conditions, and individual tolerance. Complementary lifestyle modifications consisted of sodium-restricted diet implementation, smoking cessation and alcohol intake limitation, and structured exercise programs, all aimed at optimizing cardiovascular health and achieving blood pressure control. This included systematic antiplatelet therapy for thrombosis prevention and personalized lipid-modifying regimens when indicated, with the dual objectives of maintaining optimal lipid profiles and mitigating atherosclerotic cardiovascular risks through comprehensive metabolic regulation.

Data collection

Clinical data, including age, sex, height, body mass index (BMI), duration of hypertension, diabetes history, and smoking and alcohol consumption histories, were collected for all patients. Fasting blood samples were also collected for laboratory tests, including blood urea nitrogen (BUN), creatinine (Cr), estimated glomerular filtration rate (eGFR), uric acid (UA), fasting blood glucose (FBG), triglycerides (TG), total cholesterol (TC), high-density lipoprotein cholesterol (HDL-C), low-density lipoprotein cholesterol (LDL-C), aspartate aminotransferase (AST), and alanine aminotransferase (ALT). Additionally, patients underwent overnight PSG monitoring, ambulatory blood pressure monitoring, and carotid blood flow measurements.

Biochemical parameters measurement

After a 12-hour overnight fast, 2 ml venous blood samples were collected from patients and centrifuged at 3000 rpm for 10 minutes under 4°C to isolate serum for laboratory index determination. The biochemical parame-

ters, including BUN, Cr, eGFR, UA, TG, TC, HDL-C, LDL-C, AST, and ALT, were measured enzymatically using an AU 5800 Chemistry Analyzer (Beckman Coulter, USA). FBG levels were assessed using the glucose oxidase-peroxidase reagent with a DxC 800 Glucose Analyzer (Beckman Coulter, USA).

PSG monitoring

All patients diagnosed with OSAHS were monitored using PSG with the E-system (Australia Kangdi Company) for a duration of 8 hours, commencing at 22:00 on the night of the examination and concluding at 6:00 the following morning. During the monitoring, the patients' apnea index (AI), hypopnea index (HI), AHI, oxygen desaturation index (ODI), minimum oxygen saturation ($minSpO_2$), and average oxygen saturation ($AvgSpO_2$) were recorded.

Ambulatory blood pressure monitoring

A non-invasive portable ambulatory blood pressure monitor (Mobile-O-Graph PWA, IEM GmbH) was utilized to record blood pressure dynamics from 8:00 to 22:00, measuring every 60 minutes to obtain systolic blood pressure (SBP) and diastolic blood pressure (DBP).

Carotid artery ultrasonography

The intima-media thickness (IMT) was measured using a Vidd-7 device from General Electric Company, employing a probe frequency of 10 MHz.

Statistical treatment

Statistical analyses were conducted using SPSS version 23.0 (SPSS Inc., Chicago, IL, USA). Continuous measurement data that followed a normal distribution were expressed as mean ± standard deviation, and a t-test was employed for group comparisons. For measurement data that did not follow a normal distribution, results were reported as [M (P_{25}, P_{75})], and the nonparametric rank sum test was utilized. Count data were expressed as percentages (%) and compared using the chi-square test (χ^2). The presence or absence of secondary arteriosclerosis served as the dependent variable, while statistically significant factors identified through univariate analysis and Pearson correlation analyses were treated as independent variables in multivariate logistic regression. A nomogram prediction model was developed based on the independent risk factors identified using multivariate analysis. Receiver operating characteristic (ROC) curve analysis was employed to evaluate the predictive performance of the established model. A *P*-value of <0.05 was considered statistically significant.

Results

Patient characteristics

A total of 140 OSAHS patients with hypertension were included in the study. Among these, 39 patients (27.86%) presented secondary arteriosclerosis were classified as the research group, while the remaining 101 patients (72.14%) without arteriosclerosis were classified as the control group, as illustrated in **Figure 1**. No significant differences were observed between the two groups regarding age, gender, BMI, duration of hypertension, history of diabetes, smoking, alcohol consumption, BUN, Cr, eGFR, UA, Tc, HDL-C, ALT, and AST levels (*P*>0.05). However, FBG, TG, and LDL levels were significantly higher in the research group compared to the control group (*P*<0.05), as shown in **Table 1**.

Comparison of the PSG parameters between the two patient groups

There was no significant difference in the levels of AI, HI, ODI, and $AvgSpO_2$ between the two groups (*P*>0.05). However, the AHI in the research group was significantly higher than that in the control group, while the $minSpO_2$ was significantly lower (*P*<0.05), as illustrated in **Figure 2**.

Comparison of ambulatory blood pressure and carotid blood flow index between the two groups

DBP levels were comparable between the two groups (*P*>0.05). However, SBP and IMT were significantly higher in the study group than those in the control group (*P*<0.05), shown in **Figure 3**.

Correlation analysis of secondary arteriosclerosis with clinical parameters in OSAHS patients with hypertension

Pearson correlation analysis indicated that FBG, TG, LDL, AHI, SBP, and IMT in patients

Table 1. Comparison of baseline characteristics between the two patient groups

Datum		Research group (n=39)	Control group (n=101)	$t/\chi^2/Z$	*P*
Age		49.33±10.51	46.05±9.10	1.832	0.069
Sex	Male	26 (66.67)	53 (52.48)	2.305	0.129
	Female	13 (33.33)	48 (47.52)		
BMI (kg/m^2)		28.26±3.74	26.99±3.89	1.743	0.083
Hypertension disease course		5 (4, 5)	4 (4, 5)	-1.887	0.059
Diabetes mellitus	Yes	21 (53.85)	40 (39.60)	2.321	0.128
	No	18 (46.15)	61 (60.40)		
History of smoking	Yes	14 (35.90)	33 (32.67)	0.131	0.717
	No	25 (64.10)	68 (67.33)		
History of alcohol consumption	Yes	18 (46.15)	37 (36.63)	1.069	0.301
	No	21 (53.85)	64 (63.37)		
BUN (mmol/L)		5.16±1.74	4.86±1.03	1.272	0.205
Cr (μmol/L)		69.08±9.41	68.56±9.82	0.285	0.776
eGFR (mL/min/1.73 m^2)		111.07±18.72	108.47±15.97	0.820	0.414
UA (μmol/L)		403.45±36.96	396.80±32.46	1.045	0.298
FBG (mmol/L)		4.95 (4.31, 5.7)	4.21 (3.80, 4.87)	-3.068	0.002
TG (mmol/)		1.89 (1.66, 2.21)	1.78 (1.47, 1.98)	-2.689	0.007
TC (mmol/L)		4.72±0.81	4.44±0.75	1.929	0.056
HDL (mmol/L)		1.07 (0.92, 1.26)	0.97 (0.86, 1.15)	-1.936	0.053
LDL (mmol/L)		3.16±0.68	2.75±0.56	3.608	<0.001
ALT (U/L)		22.48±3.32	23.63±4.11	-1.568	0.119
AST (U/L)		25.47±4.08	26.59±4.84	-1.271	0.206

Note: BMI, body mass index; BUN, blood urea nitrogen; Cr, creatinine; eGFR, estimated glomerular filtration rate; UA, uric acid; FBG, fasting blood glucose; TG, triglyceride; TC, total cholesterol; HDL-C, high-density lipoprotein cholesterol; LDL-C, low-density lipoprotein cholesterol; ALT, alanine aminotransferase; AST, aspartate aminotransferase.

with OSAHS and hypertension were positively correlated with secondary arteriosclerotic diseases (r=0.273, 0.249, 0.294, 0.190, 0.198, 0.252; all $P<0.05$). In contrast, $minSpO_2$ was negatively correlated with secondary arteriosclerotic disease (r=-0.199, $P<0.05$), as shown in **Table 2**.

Multivariate analysis of secondary arteriosclerosis in OSAHS patients with hypertension

Variables demonstrating statistical significance in univariate and correlation analyses (FBG, TG, LDL, AHI, $minSpO_2$, SBP, and IMT) were incorporated into a multivariate logistic regression model for identifying risk factors for atherosclerosis in patients with concurrent OSAHS and hypertension. The results confirmed that all these parameters were independent risk factors for atherosclerosis in OSAHS patients with hypertension ($P<0.05$), as detailed in **Table 3**.

Construction of a nomogram for predicting secondary atherosclerosis in OSAHS patients with hypertension

A nomogram prediction model for secondary atherosclerosis in OSAHS patients with hypertension was constructed incorporating FBG, TG, LDL, AHI, $minSpO_2$, SBP, and IMT, as shown in **Figure 4**. The relative contribution of each factor, ranked by descending importance, was: LDL, FBG, $minSpO_2$, and IMT.

Predictive performance of each risk factor and their combination for secondary arteriosclerosis in OSAHS patients with hypertension

The ROC curve was employed to evaluate the efficacy of various risk factors in predicting arteriosclerosis among OSAHS patients with hypertension. The areas under the curve (AUC) for FBG, TG, LDL, AHI, $minSpO_2$, SBP, IMT, and their combination were 0.668, 0.647, 0.636, 0.690, 0.636, 0.608, 0.805, and 0.922,

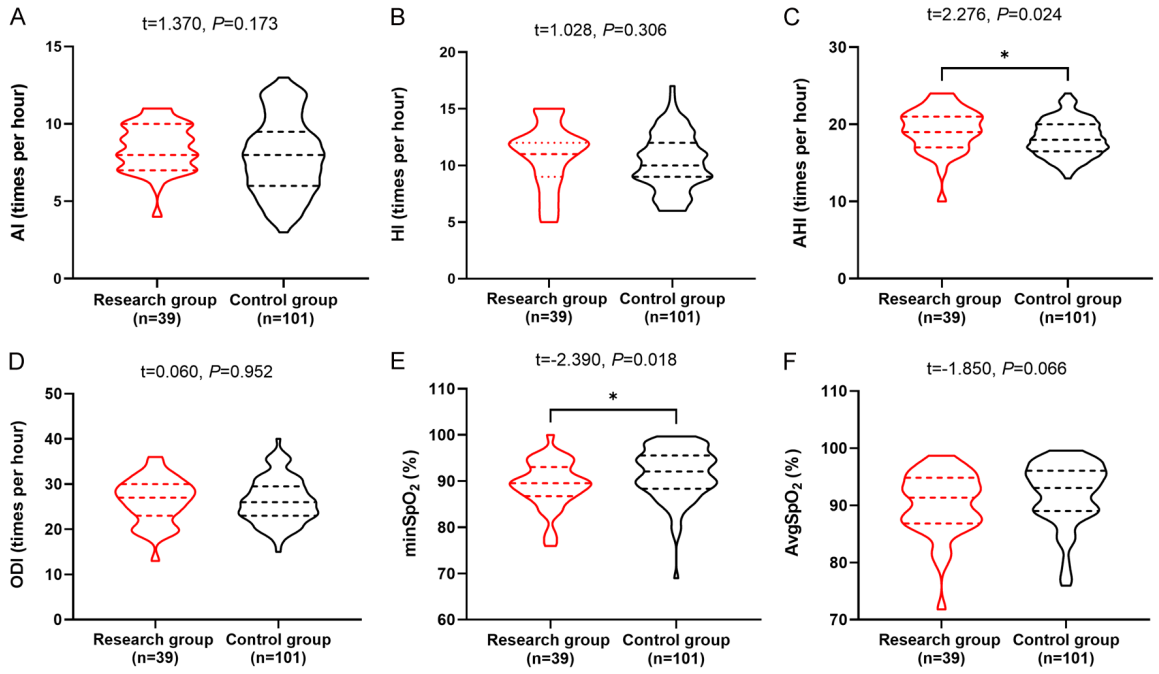

Figure 2. Comparison of PSG parameters between the two groups of patients. Note: (A) AI, (B) HI, (C) AHI, (D) ODI, (E) $minSpO_2$, (F) $AvgSpO_2$; AI, apnea index; HI, hypopnea index; AHI, apnea-hypopnea index; ODI, oxygen desaturation index; $minSpO_2$, minimum oxygen saturation; $AvgSpO_2$, average oxygen saturation. *P<0.05.

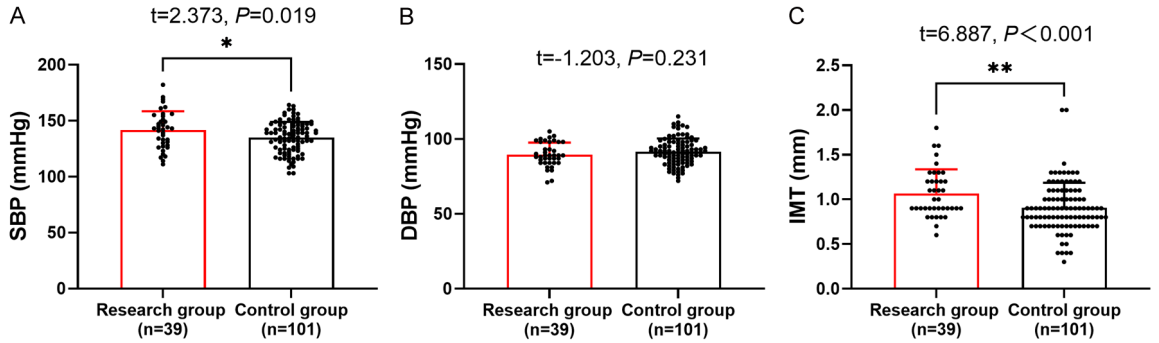

Figure 3. Comparison of ambulatory blood pressure and intima-media thickness between the two groups. Note: (A) SBP, (B) DBP, (C) IMT. SBP, systolic blood pressure; DBP, diastolic blood pressure; IMT, intima-media thickness. *P <0.05, **P <0.001.

respectively (**Figure 5**). The nomogram incorporating all these risk factors demonstrated the highest predictive performance, with the sensitivity of 79.5% and the specificity of 92.1% (**Table 4**).

Discussion

Hypertension is a prevalent comorbidity in OSAHS patients, with reported incidence ranging from 50% to 60% [15]. This association likely originates from intermittent hypoxemia and disrupted sleep architecture. The recurrent hypoxemic episodes trigger sympathetic nervous system hyperactivation, resulting in vasoconstriction and consequent elevated blood pressure [16]. Additionally, sleep fragmentation interferes with normal circadian blood pressure regulation, particularly blunting the physiological nocturnal blood pressure dip, which may elevate cardiovascular risk [17]. Research indicates that this OSAHS-hypertension comorbidity is closely linked to an increased risk of arterial stiffness [18].

Table 2. Correlation analysis of secondary arteriosclerosis with clinical parameters in patients

Variable	Statistical value	FBG	TG	LDL-C	AHI	SBP	IMT	$minSpO_2$
Secondary arteriosclerotic	*r*	0.273	0.249	0.294	0.190	0.198	0.252	-0.199
	P	0.001	0.003	<0.001	0.024	0.019	0.003	0.018

Note: FBG, fasting blood glucose; TG, triglyceride; LDL-C, low-density lipoprotein cholesterol; SBP, systolic blood pressure; IMT, intima-media thickness; AHI, apnea-hypopnea index; $minSpO_2$, minimum oxygen saturation.

Table 3. Multivariate logistic regression analysis of arteriosclerosis in OSAHS patients with hypertension

Variables	β	*SE*	Wald χ^2	*P*	OR (95% CI)
FBG	0.830	0.278	8.924	0.003	2.293 (1.330~3.952)
TG	1.768	0.706	6.263	0.012	5.858 (1.467~23.393)
LDL-C	0.297	0.102	8.453	0.004	1.345 (1.101~1.643)
AHI	1.576	0.490	10.360	0.001	4.836 (1.852~12.627)
$minSpO_2$	-0.140	0.048	8.655	0.003	0.870 (0.792~0.954)
SBP	0.043	0.016	6.991	0.008	1.044 (1.011~1.077)
IMT	2.813	0.895	9.887	0.002	16.667 (2.886~96.267)

Note: FBG, fasting blood glucose; TG, triglyceride; LDL-C, low-density lipoprotein cholesterol; AHI, apnea-hypopnea index; $minSpO_2$, minimum oxygen saturation; SBP, systolic blood pressure; IMT, intima-media thickness.

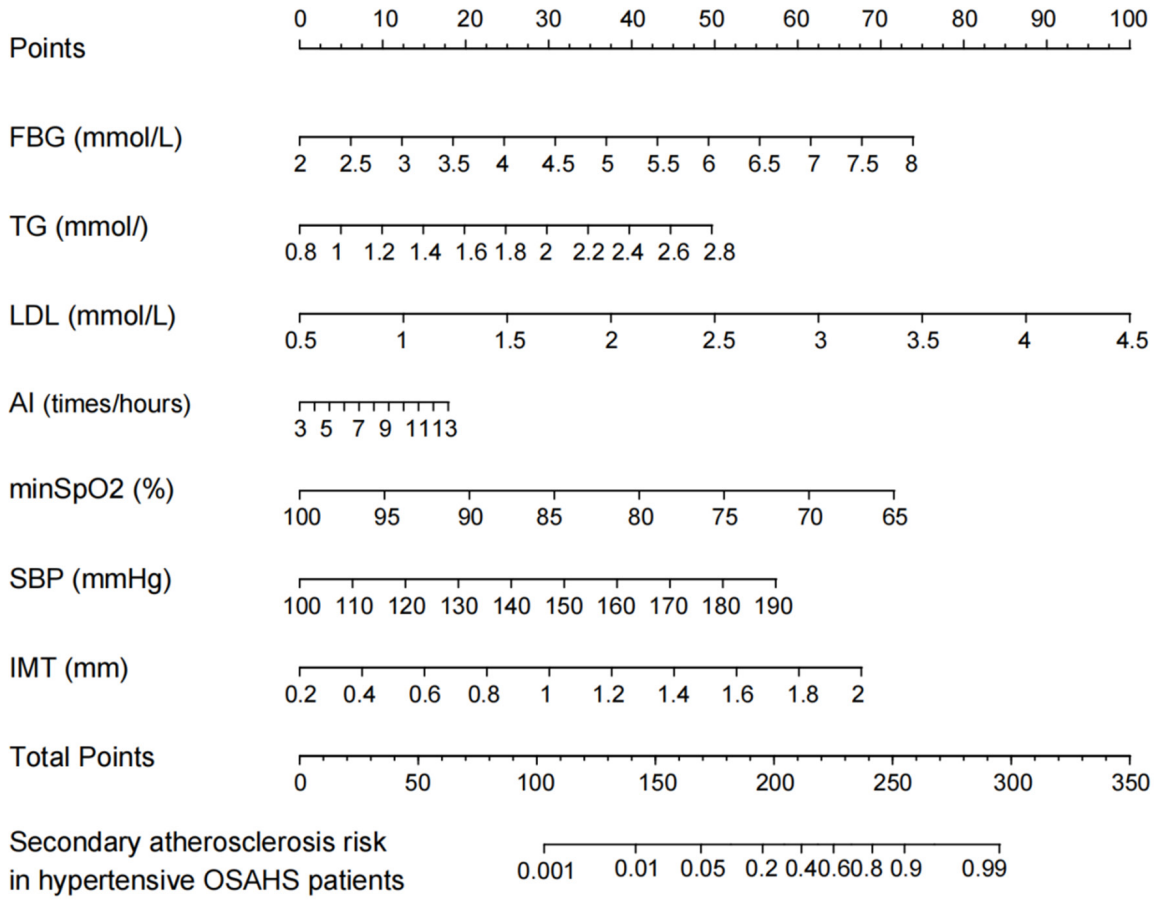

Figure 4. Nomogram model for risk prediction of secondary arteriosclerosis in OSAHS patients with hypertension. Note: FBG, fasting blood glucose; TG, triglyceride; LDL-C, low-density lipoprotein cholesterol; AHI, apnea-hypopnea index; $minSpO_2$, minimum oxygen saturation; SBP, systolic blood pressure; IMT, intima-media thickness; OSAHS, obstructive sleep apnea hypopnea syndrome.

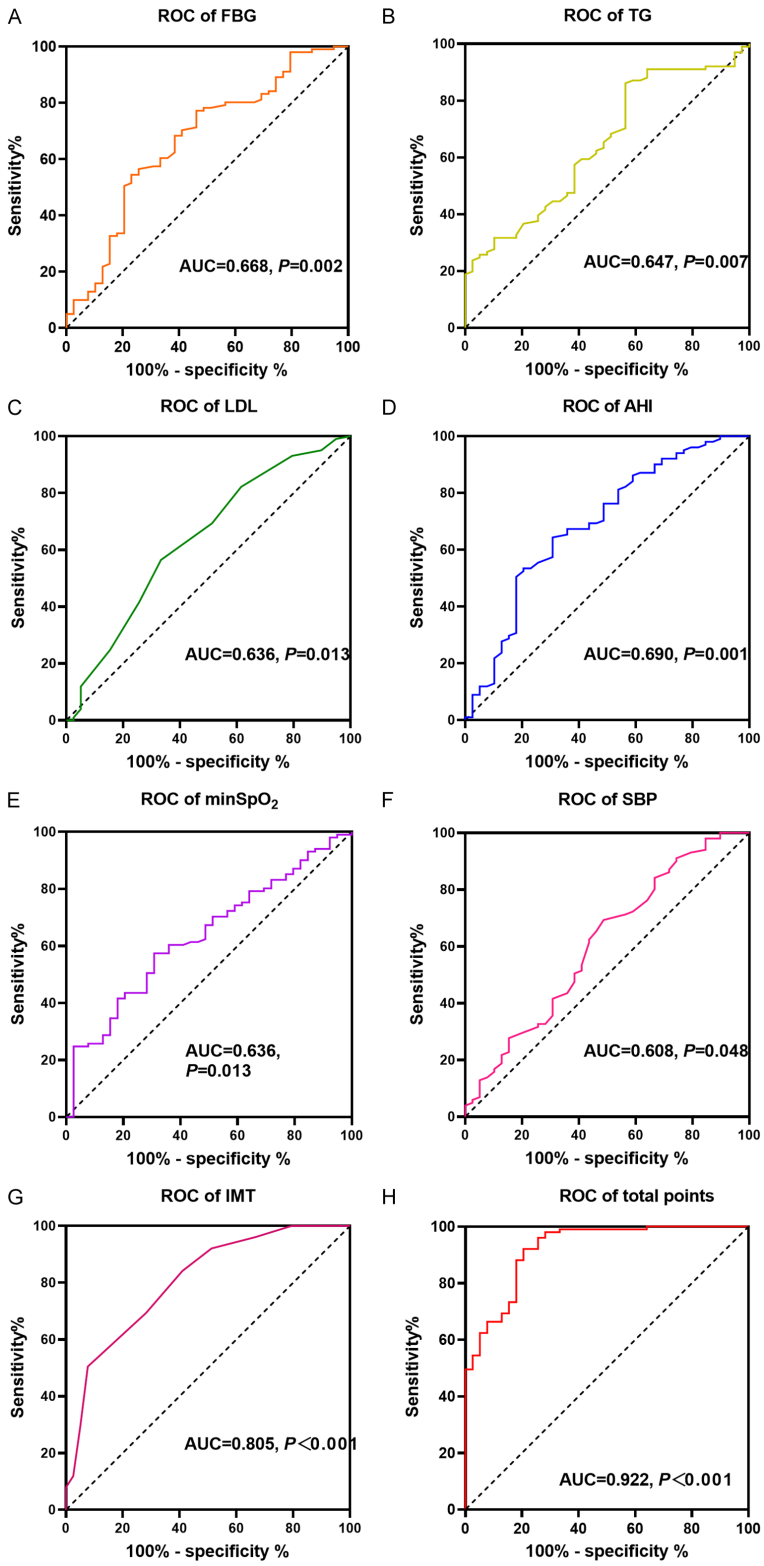

Figure 5. ROC curves for FBG (A), TG (B), LDL (C), AHI (D), $minSpO_2$ (E), SBP (F), IMT (G), and total risk score (H) in predicting secondary arteriosclerosis in OSAHS patients with hypertension. Note: FBG, fasting blood glucose; TG, triglyceride; LDL-C, low-density lipoprotein cholesterol; AHI, apnea-hypopnea index; $minSpO_2$, minimum oxygen saturation; SBP, systolic blood pressure; IMT, intima-media thickness; ROC, receiver operating characteristic.

Arteriosclerosis develops progressively through three key pathological processes, including vascular endothelial dysfunction, proliferation of vascular smooth muscle cells, and lipid deposition [3]. On one hand, intermittent hypoxia and hypercapnia resulting from OSAHS can exacerbate vascular endothelial damage, promote the release of inflammatory factors and oxidative stress products, and accelerate the progression of arterial stiffness [2]. On the other hand, hypertension can itself cause damage to vascular endothelial cells, enhance lipid deposition and inflammatory responses, and contribute to the formation of atherosclerotic plaques [19]. Therefore, for OSAHS patients with hypertension, comprehensive interventions beyond conventional antihypertensive and ventilatory therapies are imperative. Targeting sleep quality optimization and hypoxemia correction may potentially attenuate arteriosclerosis progression and improve long-term cardiovascular outcomes.

Polysomnography (PSG) is widely adopted for the diagnosis of obstructive sleep apnea-hypopnea syndrome (OSAHS) in clinical practice. However, its potential for assessing the risk of arterial stiffness has not been fully explored. This study aims to investigate the association between PSG parameters and arterial stiffness in OSAHS patients with hypertension.

The findings revealed that the prevalence of arteriosclerosis among OSAHS patients with hypertension was 27.86%, indicating a relatively high risk

Table 4. ROC curve analysis of various factors and their combination in predicting secondary arteriosclerosis in OSAHS patients with hypertension

Variable	AUC	*SE*	*P*	95% CI	Sensitivity	Specificity
FBG	0.668	0.053	0.002	0.564~0.771	76.9%	68.6%
TG	0.647	0.051	0.007	0.547~0.747	43.6%	86.1%
LDL-C	0.636	0.054	0.013	0.531~0.742	66.7%	56.4%
AHI	0.690	0.052	0.001	0.588~0.792	69.2%	66.4%
$minSpO_2$	0.636	0.050	0.013	0.538~0.734	57.4%	73.4%
SBP	0.608	0.055	0.048	0.500~0.716	51.3%	79.4%
IMT	0.805	0.042	<0.00 1	0.724~0.887	59.0%	56.8%
Total risk score	0.922	0.025	<0.00 1	0.872~0.971	79.5%	92.1%

Note: FBG, fasting blood glucose; TG, triglyceride; LDL-C, low-density lipoprotein cholesterol; AHI, apnea-hypopnea index; $minSpO_2$, minimum oxygen saturation; SBP, systolic blood pressure; IMT, intima-media thickness; ROC, receiver operating characteristic; AUC, area under the curve.

of arteriosclerosis in this population. This heightened risk is primarily attributed to OSAHS-related nocturnal intermittent hypoxia. Repeated episodes of hypoxia triggers cardiovascular and central chemoreceptors, activating sympathetic nerves, promoting peripheral vasoconstriction, and consequently elevating blood pressure [20]. Additionally, hypoxia-reoxygenation cycles lead to excessive ROS generation, which increases the production of vasculitis-inducing substances. This process not only perpetuates elevated blood pressure levels but may also exacerbate endothelial dysfunction, thereby worsening the condition of arteriosclerosis [21]. This study also identified a close association between arteriosclerosis and several patient parameters, including FBG, TG, LDL, AHI, $minSpO_2$, SBP, and IMT. FBG, TG, and LDL are critical indicators of metabolic syndrome, and their abnormal elevations can lead to vascular endothelial damage and lipid deposition, thereby accelerating atherosclerosis progression [22, 23]. AHI and $minSpO_2$ are critical indicators of OSAHS severity, with higher AHI reflecting more frequent apnea and hypopnea events, and lower $minSpO_2$ indicating inadequate oxygen absorption during sleep due to apnea and hypoventilation episodes [24, 25]. Huang et al. [26] suggested that low $minSpO_2$ levels may enhance sympathetic nerve activity while diminishing parasympathetic activity, potentially leading to cardiac variability dysfunction. SBP represents the pressure exerted by blood on arterial walls during heart contractions. Chronic elevation in SBP causes mechanical damage to arterial walls, promoting lipid deposition and plaque formation, which contribute to arteriosclerosis and stenosis [27]. IMT, a biomarker for cardiovascular risk, has been shown to correlate with an increased risk of arteriosclerosis [28]. Multifactor logistic regression identified these factors as independent risk factors for the development of arteriosclerosis, and Pearson correlation analysis revealed that increases in FBG, TG, LDL, AHI, SBP, and IMT were positively correlated with an elevated risk of arteriosclerosis, while a decrease in $minSpO_2$ was negatively correlated with arteriosclerosis risk, aligning partially with previous results [29, 30]. These results underscore the importance of early intervention in OSAHS patients with hypertension. Effective strategies include controlling blood glucose and lipid levels, enhancing sleep quality, and increasing nocturnal oxygen saturation, all of which are crucial for preventing and mitigating arteriosclerosis progression.

This study assessed the predictive performance of various factors and models to evaluate their practical application value in forecasting secondary arteriosclerosis in OSAHS patients with hypertension. ROC curve analysis revealed that the IMT and total score exhibited the highest AUC values of 0.805 and 0.922, respectively, indicating their high accuracy in predicting arteriosclerosis. As a key marker of arteriosclerosis, IMT is easily measurable and offers significant predictive value. The total risk score, integrating multiple risk factors, provides a more comprehensive assessment. The AUC values for AHI and $minSpO_2$ derived from PSG were 0.690 and 0.636, respectively. While these values are slightly lower than those of IMT and the total risk score, they still suggest a

reasonable predictive capability. This indicates that AHI and minSpO$_2$ can serve as valuable tools for assessing the risk of arteriosclerosis in OSAHS patients with hypertension. However, AHI and minSpO$_2$ alone do not fully capture the risk of arteriosclerosis and should be considered in conjunction with other clinical indicators, such as FBG, TG, LDL, SBP, and IMT, for a more comprehensive risk assessment.

This study has limitations, such as small sample size and a single-center study, which may limit the generalizability of the results. Future research will optimize the prediction model, increase the sample size, and integrate more clinical data and biomarkers to improve the accuracy of atherosclerosis prediction. Additionally, the relationships between various PSG parameters and atherosclerosis across different populations will be explored to identify specific prediction patterns. Meanwhile, long-term follow-up data will be utilized to better understand the dynamic relationship between atherosclerosis progression and PSG monitoring indicators. The goal is to provide more precise tools to identify and manage atherosclerosis risk in OSAHS patients with hypertension, improving their prognosis and quality of life.

In summary, a comprehensive assessment incorporating FBG, TG, LDL, SBP, and IMT is essential for enhancing the prediction accuracy for atherosclerosis risk in OSAHS patients with hypertension. Future research will focus on refining the predictive model by integrating additional clinical data and biomarkers, thereby improving the precision of arteriosclerosis predictions.

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Disclosure of conflict of interest

None.

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