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Original Article

Obstructive sleep apnea contributes to coronary artery disease development and poor prognosis

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Abstract: Objective: To explore the correlation between obstructive sleep apnea (OSA) and the incidence of coronary heart disease (CHD), as well as the impact of OSA severity on the short-term outcomes of CHD patients. Methods: A retrospective analysis was conducted on 78 CHD patients admitted to Zhejiang Provincial People's Hospital from January 2022 to December 2024, with another 78 subjects who underwent health checkups in the same hospital during the same period enrolled as the control group. The prevalence of OSA and baseline data were compared between the two groups. Meanwhile, CHD patients were divided into the mild-to-moderate (n = 26) and severe (n = 19) OSA groups according to OSA severity. Baseline data of patients in different subgroups were compared, and all patients were followed up. The incidence of adverse cardiovascular events was recorded and analyzed. Results: The CHD group had higher incidences of OSA, hypertension, dyslipidemia and diabetes compared to the control group (all $P < 0.05$). Multivariate analysis identified these factors as independent risk factors for CHD. A predictive model constructed based on these factors showed good performance with an AUC of 0.638. Further analysis revealed that patients with severe OSA had higher levels of peripheral hemoglobin and IL-6, higher incidence of coagulation dysfunction and diabetes mellitus than those with mild-to-moderate OSA (all $P < 0.05$). Conclusion: OSA is closely associated with the occurrence of CHD, and the predictive model established by combining OSA with other baseline data has favorable predictive efficacy for CHD. In addition, OSA severity exerts a notable impact on the prognosis of CHD patients.

Keywords: Coronary heart disease, obstructive sleep apnea, predictive model, disease severity, short-term prognosis

Introduction

Obstructive sleep apnea (OSA) is a prevalent disorder characterized by recurrent upper airway collapse during sleep, which leads to reduced ventilation or apnea. Its clinical manifestations include nocturnal sleep-disordered breathing, diurnal somnolence, and fatigue, all of which severely impair patients' quality of life [1, 2]. In recent years, with the changes in lifestyles and the rising prevalence of obesity, the incidence of OSA is increasing year by year, making it a major public health concern. Studies have demonstrated that OSA is not only an independent risk factor for cardiovascular diseases, but also closely associated with the onset and progression of coronary heart disease (CHD). However, the specific underlying mechanisms and clinical implications of this association remain to be fully elucidated [3, 4].

As one of the leading fatal diseases globally, CHD is associated with various pathophysiological processes, including sustained sympathetic activation, systemic inflammation and oxidative stress, dysregulation of glucose and lipid metabolism, hypercoagulable states, and myocardial ischemia-reperfusion injury [5, 6]. Recent studies further suggest that OSA may exacerbate these pathological processes through

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mechanisms such as intermittent hypoxia, sleep structure disruption, and pronounced fluctuations in intrathoracic pressure, thereby directly contributing to the onset and progression of CHD. However, current research on the relationship between OSA and CHD largely focuses on single mechanisms or cross-sectional analyses [7, 8]. There is a lack of comprehensive predictive models that integrate OSA with patients' baseline clinical characteristics, and a systematic conclusion regarding the impact of OSA severity on the prognosis of CHD patients at different stages has not yet been reached. Therefore, this study aims to explore the correlation between OSA and the occurrence of CHD through retrospective analysis and to construct a risk prediction model for CHD combining OSA with other baseline indicators, further analyzing the effect of different OSA severity levels on short-term cardiovascular adverse events in CHD patients. The innovation of this study lies not only in evaluating the role of OSA in CHD occurrence from a multifactorial integrated perspective but also in being the first to investigate the association between OSA severity and short-term prognosis of CHD in the local population. It is anticipated to provide more targeted evidence-based guidance for early identification of high-risk patients and implementation of personalized interventions in clinical practice.

Materials and methods

General data

A retrospective analysis was conducted on clinical data of 78 patients with CHD admitted to Zhejiang Provincial People's Hospital from January 2022 to December 2024. Meanwhile, 78 subjects who underwent routine health checkups in the same hospital during the same period was selected as the control group. The prevalence of OSA and baseline data were observed and compared between the two groups. At the same time, patients were divided into the mild-to-moderate OSA group (n = 26) and severe OSA group (n = 19) according to the OSA severity.

This study was approved by the Ethics Committee of the Zhejiang Provincial People's Hospital (Affiliated People's Hospital, Hangzhou Medical College).

Inclusion criteria: 1. Age > 18 years; 2. Diagnosis of CHD in accordance with relevant guidelines [9]; 3. Diagnosis of OSA in accordance with its guideline standards [10]; 4. Complete clinical data; 5. First-time diagnosis of CHD.

Exclusion criteria: 1. Sleep-disordered breathing caused by other etiologies; 2. A history of asthma; 3. Other valvular heart disease or congenital heart disease; 4. A history of prior treatment for CHD; 5. Thyroid dysfunction; 6. Hepatic, renal or other organ dysfunction; 7. Incomplete clinical data; 8. Pregnant or lactating women.

Methods

Biochemical testing: General patient information, including baseline data, liver and kidney function indicators, disease course, inflammatory factor levels, was retrieved from the electronic medical record system.

Laboratory test protocols: Fasting venous blood samples were collected from the patients upon admission. A fully automated hematology analyzer (Sysmex XN-9000) was used for complete blood count to measure white blood cell (WBC) count. Serum interleukin-6 (IL-6) levels were detected via chemiluminescent immunoassay using an analyzer (Roche Cobas e801). C-reactive protein (CRP) was measured by immunoturbidimetry with an analyzer (Beckman Coulter AU5800). Liver and kidney function and blood lipid levels were assessed using a biochemical analyzer (Roche Cobas c702). Specific indicators included alanine aminotransferase (ALT) and aspartate aminotransferase (AST) for liver function evaluation; albumin, serum creatinine (SCr) and blood urea nitrogen (BUN) for renal function assessment; and total cholesterol, high-density lipoprotein (HDL), and low-density lipoprotein (IDL) for lipid profile analysis. All test procedures were strictly performed in accordance with the standard operating procedures provided by the reagent manufacturers.

Polysomnography (PSG): Sleep monitoring was conducted using a Compumedics polysomnography system (Australia). All subjects underwent a 7-hour unattended polysomnography test, during which conventional parameters including peripheral oxygen saturation, pulse rate, respiratory rate, snoring intensity, and oronasal airflow were simultaneously recorded and monitored. Monitoring data were analyzed

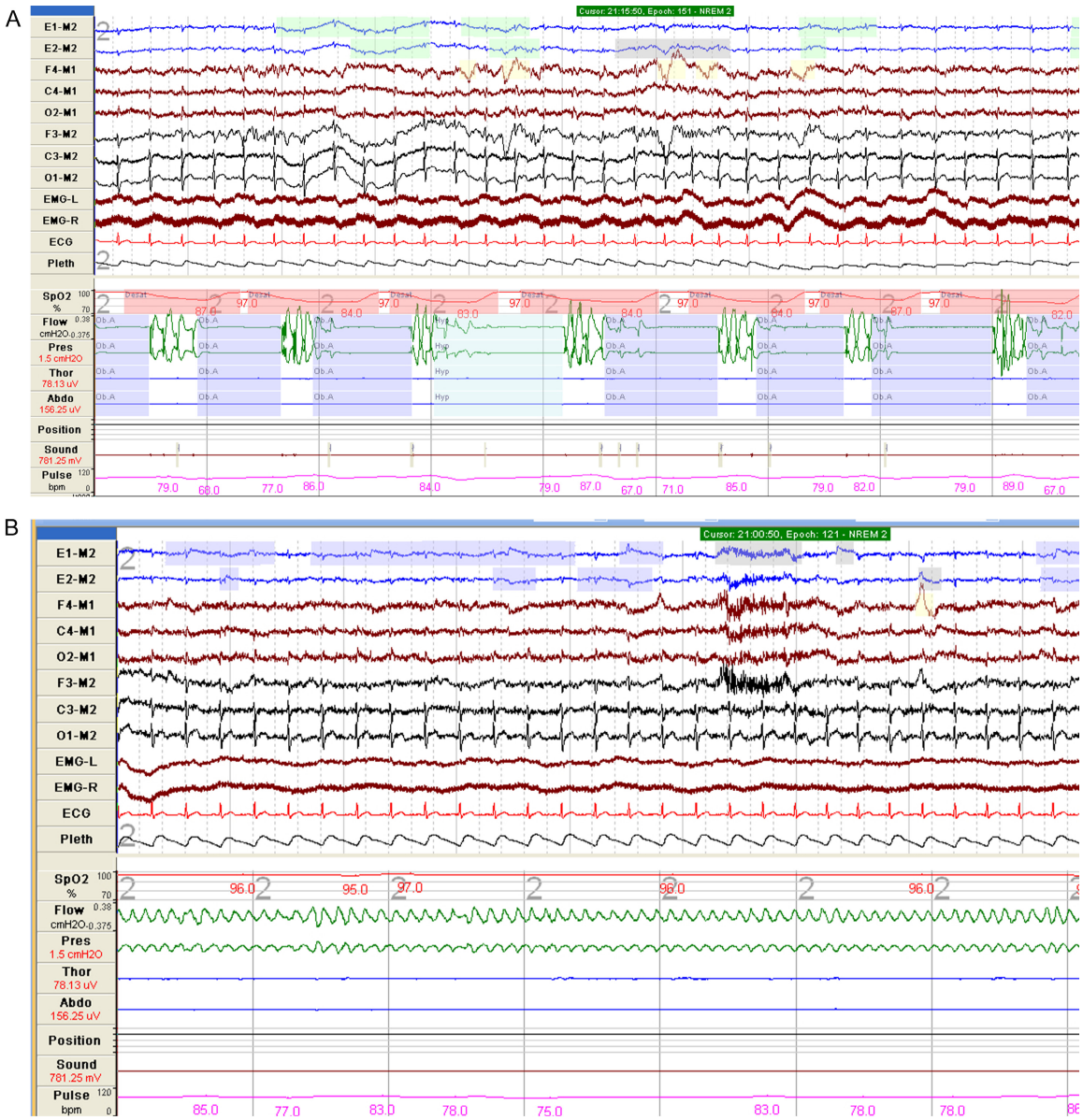

Figure 1. Polysomnography of sleep apnea in the two groups. A. Polysomnography for healthy individuals; B. Polysomnography for OSA patients. OSA: Obstructive Sleep Apnea.

using Remlogic software, and diagnoses were made in accordance with relevant guidelines (**Figure 1**).

Coronary angiography: Coronary angiography of the left and right coronary arteries was performed via peripheral arteries (radial or femoral arteries) using a GE Innova 2100 digital subtraction angiography system (USA). Standard Jundkins technique was used for multi-angle angiography during the procedure. The angiographic results were independently analyzed by two senior cardiologists, and the Gensini score of the coronary arteries was calculated accordingly.

Observation indicators

Primary observation indicators: To compare the incidence of OSA between CHD and control groups, and to establish a predictive model for the differences in baseline data between the two groups; to compare the relationship between different degrees of OSA lesions and the incidence of postoperative adverse events in patients.

Secondary observation indicators: To compare the expression differences between different degrees of OSA lesions and baseline data of patients with CHD.

Table 1. Comparison of baseline data between coronary heart disease and control groups

Items	Control Group (n = 78)	Coronary Heart Disease Group (n = 78)	Statistical Value	*p*-value
Age (years)	56.4 ± 10.2	55.1 ± 9.8	0.784	0.437
Male, n (%)	45 (57.7%)	48 (61.5%)	0.253	0.618
BMI (kg/m^2)	24.8 ± 3.1	25.2 ± 3.4	0.763	0.448
Smoking history, n (%)	32 (41.0%)	29 (37.2%)	0.247	0.618
Alcohol consumption history, n (%)	28 (35.9%)	25 (32.1%)	0.276	0.599
OSA, n (%)	25 (32.1%)	45 (57.7%)	10.892	0.001
Hypertension, n (%)	30 (38.5%)	50 (64.1%)	10.584	0.001
Dyslipidemia, n (%)	28 (35.9%)	47 (60.3%)	9.483	0.002
Diabetes mellitus, n (%)	15 (19.2%)	30 (38.5%)	7.136	0.008

Note: OSA: Obstructive Sleep Apnea; BMI: Body Mass Index.

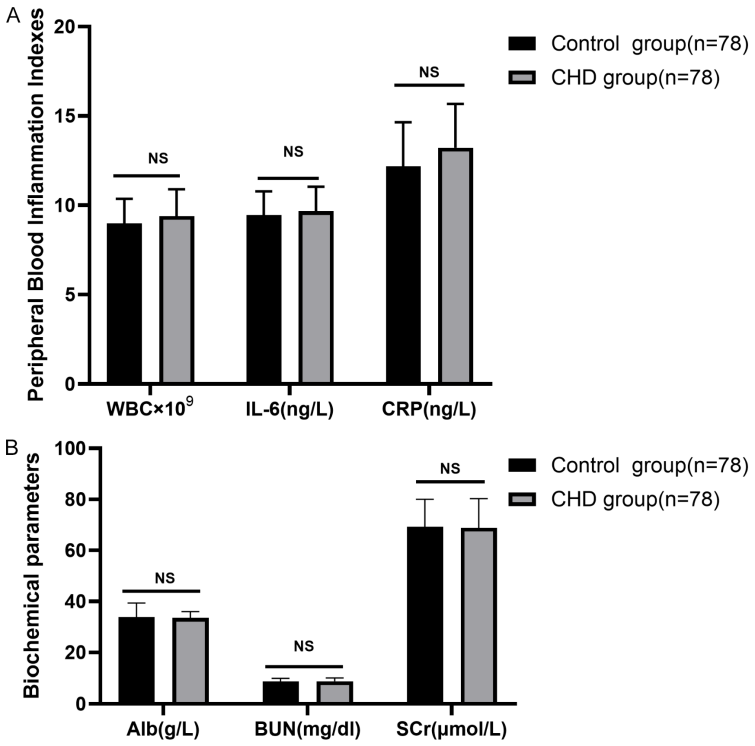

Figure 2. Comparison of peripheral serum inflammatory indicators and liver and kidney function indicators. A. Peripheral serum inflammatory indicators. B. Liver and kidney function indicators. WBC, white blood cell; CRP, C-reactive protein; IL-6, interleukin-6; Alb, albumin; SCr, serum creatinine; BUN, blood urea nitrogen. Statistical significance is indicated as follows: NS, not significant.

Table 2. Variable assignment

Variables	Assignment
Obstructive Sleep apnea	Yes = 0; No = 1
Hypertension	Yes = 0; No = 1
Diabetes mellitus	Yes = 0; No = 1
Dyslipidemia	Yes = 0; No = 1

Statistical analysis

SPSS 23.0 statistical software was used for data analysis. Normalized continuous data were expressed as "Mean ± SD". Independent samples t-tests were used for intragroup comparisons of independent, normally distributed, and homogeneous variance data between the two groups. Chi-square tests were used for comparisons of categorical data [n (%)]. Multivariate analysis was performed using multivariate logistic regression analysis, with forward stepwise regression used for variable selection. The significance level for variable entry into the model was set at 0.05, and that for variable removal from the model was set at 0.10. The study explored the influencing factors of CHD and established a predictive model for CHD. ROC curves were used to evaluate the model's effectiveness. $P < 0.05$ was considered statistically significant.

Results

Comparison of baseline data between CHD and control groups

The results of this study showed no statistically significant differences between the two groups

Table 3. Multivariate logistic regression analysis of influencing factors for coronary heart disease

Variables	B Value	Standard Error	Wald χ^2 Value	*p*-value	OR Value	95% Confidence Interval (95% CI)
OSA	0.721	0.385	3.507	0.041	2.057	(1.968, 4.371)
Hypertension	1.102	0.378	8.496	0.004	3.010	(1.434, 6.321)
Dyslipidemia	0.963	0.382	6.356	0.012	2.620	(1.238, 5.545)
Diabetes mellitus	0.684	0.412	2.756	0.047	1.982	(1.084, 4.444)
Constant	-1.735	0.362	22.971	< 0.001	0.176	-

Note: OSA: Obstructive Sleep Apnea.

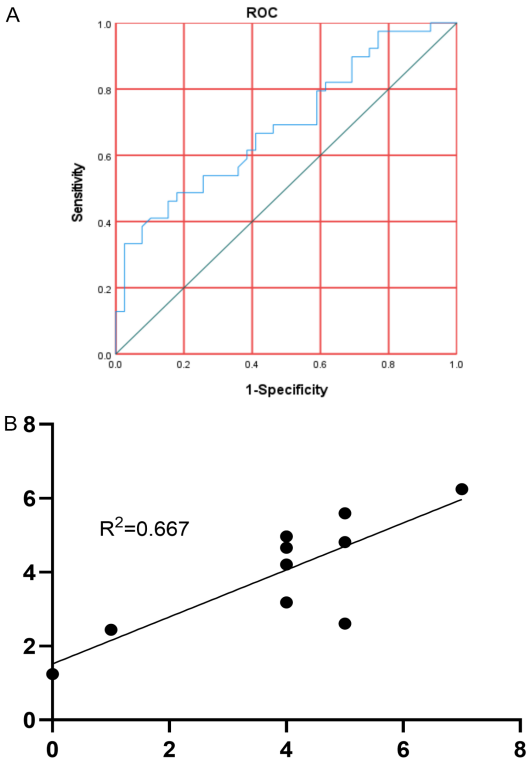

Figure 3. ROC and calibration curves of the coronary heart disease predictive model. A. ROC curve. B. Calibration curve.

in terms of age, sex, BMI, smoking history, and alcohol consumption history (all P > 0.05). However, there were statistically significant differences in the incidence of OSA, hypertension, dyslipidemia, and diabetes (all P < 0.05). See **Table 1**.

Comparison of peripheral blood inflammatory factors and liver and kidney function between CHD and control groups

No statistically significant differences were noted in the levels of peripheral blood inflammatory factors, SCr, BUN, and albumin between CHD patients and healthy controls (all P > 0.05). See **Figure 2**.

Multivariate analysis of factors associated with the occurrence of CHD

Multivariate logistic regression analysis identified OSA, hypertension, diabetes mellitus, and dyslipidemia as influencing factors for the development of CHD. See **Tables 2**, **3**.

Establishment and performance evaluation of the CHD predictive model

The predictive model constructed using OSA, hypertension, diabetes mellitus, and dyslipidemia achieved an area under the curve (AUC) of 0.638 with a 95% confidence interval (CI) of 0.516-0.762. The model formula was as follows: Logit(P) = -1.735 + 0.721 × OSA + 1.102 × hypertension + 0.963 × dyslipidemia + 0.684 × diabetes mellitus.

Meanwhile, the Hosmer-Lemeshow test yielded a chi-square value of 2.604e-030 and P = 0.999, indicating good model stability (see **Figure 3A**). Further model analysis confirmed that this model exhibited excellent predictive performance for CHD (see **Figure 3B**).

Establishment and validation of the nomogram predictive model for CHD

A Nomogram predictive model for CHD was established incorporating OSA, hypertension, diabetes mellitus, and dyslipidemia. The score corresponding to each predictive factor for an individual patient can be obtained from the Nomogram. The total score is calculated by summing the scores of all predictive factors, and the corresponding predictive probability represents the likelihood of the patient developing CHD (**Figure 4**). Additionally, a calibration curve was plotted for the model, which showed good calibration for predicting CHD and a high degree of consistency between the predicted results and the actual observations (**Figure 5**). Finally, the decision curve analysis (DCA) re-

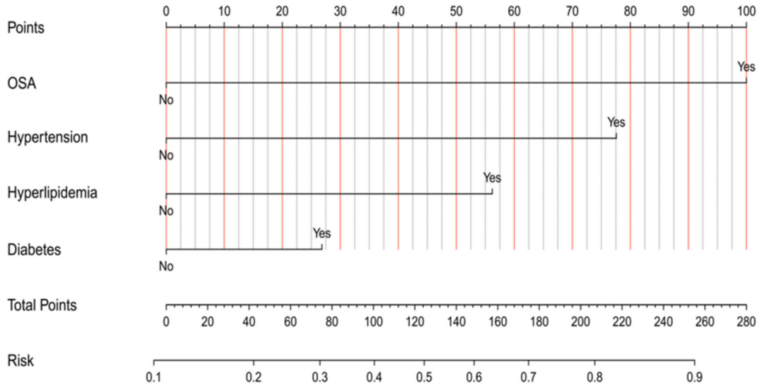

Figure 4. Nomogram predictive model for coronary heart disease. OSA: Obstructive Sleep Apnea.

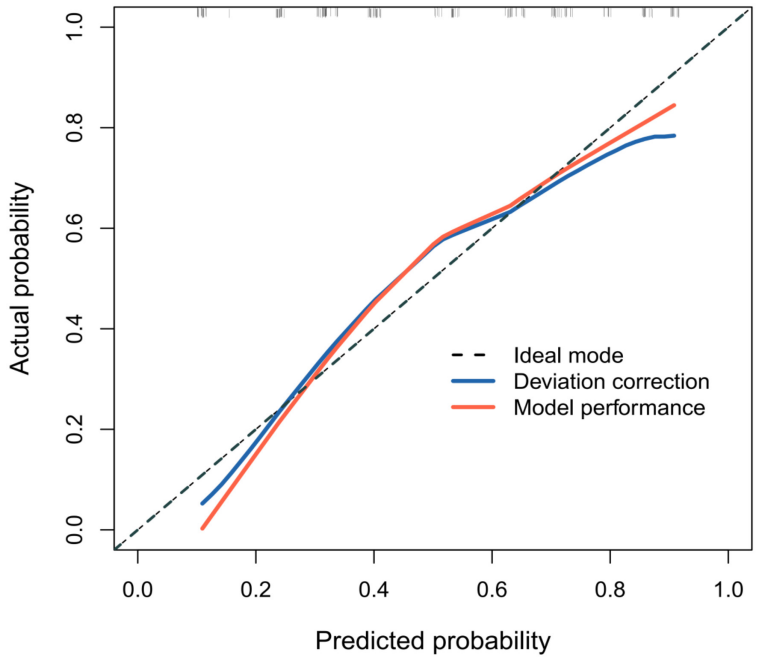

Figure 5. Calibration plot of the predictive model for coronary heart disease.

sults of the predictive model revealed a significant positive net benefit, confirming the favorable clinical utility of the model for CHD prediction (**Figure 6**).

Comparison of baseline data between mild-to-moderate and severe OSA groups

Among the 78 CHD patients, 45 were complicated with OSA, including 26 cases of mild-to-moderate OSA and 19 cases of severe OSA. Further analysis revealed no statistically significant differences between the two groups in terms of age, sex, BMI, smoking history, alcohol consumption history, hypertension, dyslipidemia, and the incidence of diabetes (all P > 0.05). See **Table 4**.

Comparison of peripheral hemoglobin, coagulation function, inflammatory factors, and incidence of diabetes between mild-to-moderate and severe OSA groups

The levels of peripheral hemoglobin, incidence of coagulation dysfunction, IL-6 levels, and prevalence of diabetes mellitus in the severe OSA group were significantly higher than those in the mild-to-moderate OSA group (all P < 0.05). See **Table 5**.

Comparison of Gensini score and total myocardial ischemia burden between mild-to-moderate and severe OSA groups

The Gensini score and total myocardial ischemia burden in the severe OSA group were significantly higher than those in the mild-to-moderate OSA group (P < 0.05). See **Figure 7**.

Comparison of incidence of adverse events between mild-to-moderate and severe OSA groups

The incidence of adverse events during follow-up was higher in the severe OSA group than in the mild-to-moderate group (P < 0.05). See **Table 6**.

Discussion

CHD is a common cardiovascular disorder and also the internal medicine disease with the highest mortality rate from a single cause [11, 12]. Relevant guidelines classify CHD into five subtypes, namely myocardial ischemia, acute coronary syndrome, myocardial infarction, ischemic heart failure, and sudden cardiac death [13]. Epidemiological surveys show that the incidence and mortality of CHD are gradually increasing worldwide, and it has become

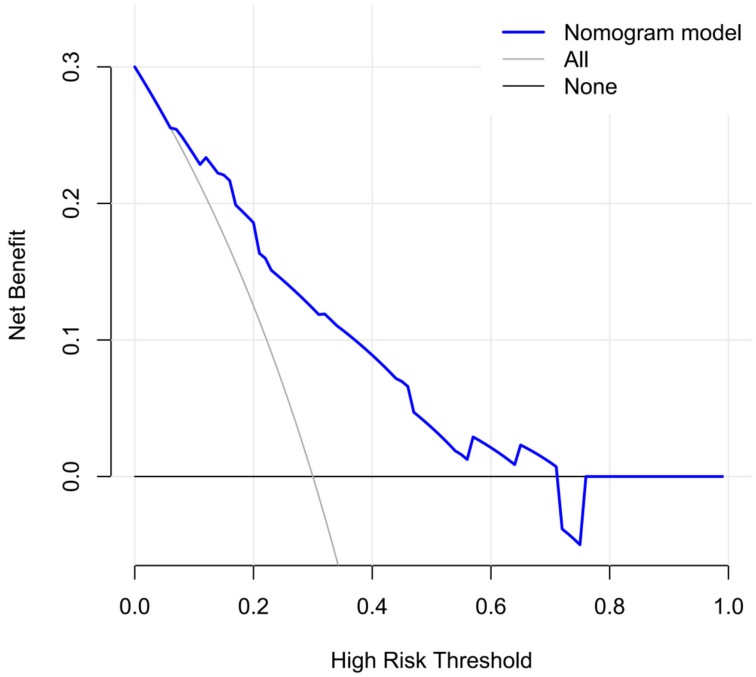

Figure 6. Decision curve analysis of the predictive model for coronary heart disease.

an important disease type threatening the health of the population [14]. Therefore, early diagnosis, treatment, and the implementation of tertiary prevention are of great significance for improving the long-term prognosis of CHD patients and alleviating social burdens. Current studies have confirmed that CHD is closely associated with patients' baseline characteristics; for instance, hypertension, diabetes mellitus, and dyslipidemia are all established influencing factors for CHD [15]. The latest research shows that OSA is also intrinsically linked to the occurrence and development of CHD, highlighting the importance of great significance to study the potential risk factors of CHD and establish a predictive models [16].

Previous studies have identified hypertension, diabetes mellitus, and dyslipidemia as risk factors for CHD [17, 18]. This study also confirmed that the incidence of hypertension, diabetes mellitus, and dyslipidemia in patients with CHD as well as the incidence of OSA were higher than those in the healthy control group. The underlying mechanisms may be explained as follows: First, all four factors mentioned above can cause insulin resistance and metabolic syndrome. Specifically, insulin resistance directly elevates blood glucose levels, which may progress to diabetes mellitus; it also activates the sympathetic nervous system, promotes sodium and water retention in the kidneys, and is accompanied by dyslipidemia, all of which ultimately accelerates coronary atherosclerosis. Second, hypertension causes high-velocity blood flow to damage vascular endothelial cells, hyperglycemia exerts direct toxic effects on endothelial cells, and hyperlipidemia leads to lipid deposition beneath the vascular endothelium, triggering oxidative stress and inflammatory responses. Meanwhile, OSA induces intermittent hypoxia and sleep disturbance, which damage the vascular intima and ultimately contribute to coronary atherosclerosis. Third, OSA impairs blood pressure control, exacerbates glucose and lipid metabolism disorders, and thus accelerates the occurrence and progression of CHD, which corroborates the conclusions of previous studies [19-22]. Meanwhile, the predictive model constructed based on these factors exhibited favorable predictive value in the present study, further verifying the research conclusion that patients' baseline data can effectively predict the occurrence of CHD [23, 24].

Previous studies have confirmed that the main potential mechanisms by which OSA induces CAHD include hypercoagulable state, coagulation dysfunction, and elevated levels of inflammatory factor interleukin-6 (IL-6) [25]. The present study found that patients with severe OSA had higher levels of peripheral hemoglobin, a higher incidence of coagulation dysfunction, and elevated IL-6 levels compared with those with mild-to-moderate OSA; additionally, the severity of coronary artery lesions and myocardial ischemia burden were greater in the severe OSA group. The specific mechanisms are elaborated as follows: Chronic intermittent hypoxia caused by OSA can stimulate the proliferation and differentiation of bone marrow hematopoietic stem cells and increase the secretion of erythropoietin, leading to an

Table 4. Comparison of general data between mild-to-moderate and severe OSA groups

Items	Mild-Moderate Group (n = 26)	Severe Group (n = 19)	Statistical Value	*p*-value
Age (years)	58.2 ± 8.7	56.1 ± 9.4	0.783	0.438
Male, n (%)	14 (53.8%)	11 (57.9%)	0.080	0.777
BMI (kg/m^2)	25.1 ± 2.9	24.6 ± 3.3	0.546	0.588
Smoking history, n (%)	11 (42.3%)	7 (36.8%)	0.144	0.704
Alcohol consumption history, n (%)	9 (34.6%)	5 (26.3%)	0.366	0.545
Hypertension, n (%)	10 (38.5%)	6 (31.6%)	0.229	0.632
Dyslipidemia, n (%)	12 (46.2%)	7 (36.8%)	0.392	0.531
Diabetes mellitus, n (%)	5 (19.2%)	3 (15.8%)	0.092	0.762

Note: OSA: Obstructive Sleep Apnea; BMI: Body Mass Index.

Table 5. Comparison of clinical indicators between mild-to-moderate and severe OSA groups

Items	Mild-Moderate Group (n = 26)	Severe Group (n = 19)	Statistical Value	*p*-value
Hemoglobin level (g/L)	132.5 ± 14.2	118.3 ± 16.5	3.127	0.003
Abnormal coagulation function, n (%)	5 (19.2%)	10 (52.6%)	6.040	0.014
Inflammatory factor IL-6 (pg/mL)	12.4 ± 3.8	28.6 ± 10.2	7.542	< 0.001
Diabetes mellitus, n (%)	4 (15.4%)	9 (47.4%)	5.828	0.016

Note: OSA: Obstructive Sleep Apnea.

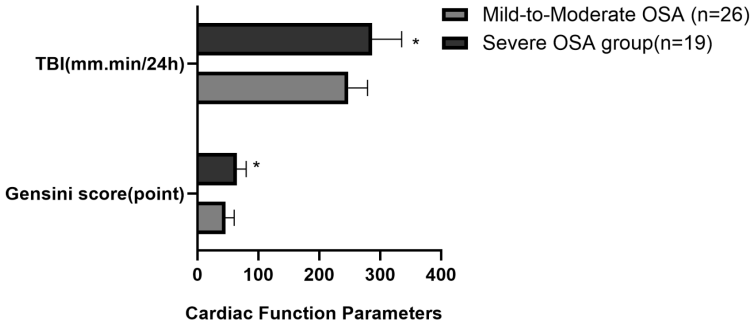

Figure 7. Assessment of coronary blood flow and ischemia degree between mild-to-moderate and severe OSA groups. Compared with mild-to-moderate OSA group, *P < 0.05. OSA: Obstructive Sleep Apnea.

increase in red blood cell count and a hypercoagulable state in OSA patients. Intermittent hypoxia can also upregulate IL-6 levels; IL-6 then induces the liver to produce C-reactive protein and fibrinogen, promotes the expression of adhesion molecules in endothelial cells, drives the transformation of monocytes into macrophages, and stimulates the proliferation and migration of vascular smooth muscle cells. These processes result in the instability of coronary plaques and even thrombosis, exacerbating the severity of coronary artery lesions. Furthermore, intermittent hypoxemia and hypercapnia caused by OSA directly stimulate carotid chemoreceptors, leading to persistent sympathetic activation, elevated catecholamine levels, increased blood pressure and heart rate, augmented myocardial oxygen consumption, and aggravated myocardial ischemia, thereby worsening the condition. Moreover, the cardiac toxicity of catecholamines can directly induce coronary artery spasm and even myocardial infarction, which is consistent with previous reports. In terms of the impact of OSA on patient prognosis, the present study found that the incidence of adverse cardiovascular events was higher in patients with severe OSA than that in those with mild-to-moderate OSA, which further suggests a direct correlation between OSA and patient prognosis. The potential mechanisms may involve the following aspects: Severe OSA excessively activates the sympathetic nervous system, resulting in increased nocturnal blood pressure and heart rate, elevated myocardial oxygen consumption, and thus an increased risk of adverse cardiovascular events. Second, severe hypoxia in the body can trigger systemic

Table 6. Comparison of incidence of adverse events between mild-to-moderate and severe OSA groups

Group	Myocardial Infarction	Revascularization	Heart Failure	Unstable Angina Pectoris	Total
Mild-Moderate Group (n = 26)	1	1	0	1	3/26
Severe Group (n = 19)	2	1	3	1	7/19
X^2					4.067
P					0.043

Note: OSA: Obstructive Sleep Apnea.

inflammatory responses, promoting the massive release of inflammatory factors such as IL-6, which leads to plaque instability, coronary artery spasm, and even acute myocardial infarction. Finally, severe OSA directly induces platelet aggregation and impairs the fibrinolytic system, which can directly trigger adverse events such as myocardial infarction and stroke, consistent with the conclusions of previous studies [26, 27].

This study has several limitations. First, as a retrospective study, it is subject to selection bias and information bias. Second, this is a single-center study with a relatively limited sample size, which may reduce the generalizability of the research results. Additionally, the lack of external data precluded external validation, which may further diminish the reliability of the CHD predictive model and warrant further research for verification. Furthermore, the follow-up period in this study was relatively short, making it impossible to track the impact of OSA comorbidity on the long-term quality of life of CAHD patients. Longer follow-up periods are needed to address this gap in future studies.

Conclusion

This retrospective study confirmed that OSA is an important independent risk factor for the occurrence and prognosis of CHD. OSA, together with hypertension, diabetes mellitus, and dyslipidemia, participates in pathological processes such as insulin resistance, vascular endothelial injury, and systemic inflammatory responses, synergistically promoting the occurrence and progression of coronary atherosclerosis. The CHD risk prediction model constructed based on the above factors exhibited good discriminative efficacy, providing a practical tool for the early identification of high-risk populations.

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Disclosure of conflict of interest

None.

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