

**ASSOCIATION OF OBSTRUCTIVE SLEEP APNEA SEVERITY WITH
NOCTURNAL OXYGENATION AND ANTHROPOMETRIC PARAMETERS IN
PATIENTS WITH CORONARY ARTERY DISEASE**

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Obstructive sleep apnea (OSA) worsens the course of coronary artery disease (CAD) through recurrent nocturnal desaturation, increased sympathetic tone, and endothelial dysfunction, yet it often remains undiagnosed: nocturnal symptoms are masked by the underlying disease and sleep testing is not part of the routine work-up.

To determine the frequency and severity of OSA among CAD patients who underwent respiratory monitoring, and to assess the association of the apnea–hypopnea index (AHI) with nocturnal oxygenation, anthropometric and cardiovascular parameters.

A cross-sectional exploratory analysis of a retrospective clinical database was conducted from January 2025 to June 2026. Of 67 patients, 45 underwent respiratory monitoring, and 38 with confirmed CAD and available AHI data were included. Events were scored according to American Academy of Sleep Medicine criteria and were predominantly obstructive. Variable-specific complete-case analysis was used, with n reported for each result; estimated echocardiographic values were excluded. Patients were stratified by AHI (<5, n=11; 5–14.9, n=5; 15–29.9, n=13; ≥ 30 h⁻¹, n=9), with the main comparison being AHI <15 (n=16) versus ≥ 15 (n=22). After the Shapiro–Wilk test, groups were compared using Welch’s t-test or Mann–Whitney U test, and four groups using ANOVA or Kruskal–Wallis; categorical variables were analyzed by Fisher’s exact test. Spearman correlation, Firth logistic regression, and ROC analysis were also performed. No multiple-comparison correction was applied; p<0.05 was considered statistically significant.

Mean age was 60.81 ± 9.66 years (n=31), with 14/29 men (48.3%); median BMI was 33.00 [30.50–37.50] kg/m², all patients had hypertension, and 12/31 had diabetes. OSA (AHI ≥ 5) occurred in 27/38 (71.1%), while AHI ≥ 15 was observed in 22/38 (57.9%). Compared with AHI <15, the AHI ≥ 15 group had higher BMI (36.00 vs 30.85 kg/m²; p=0.039) and body weight (100.0 vs 85.0 kg; p=0.012), but lower minimum nocturnal SpO₂ (77.5% vs 85.0%; p<0.001) and mean nocturnal SpO₂ (92.40% vs 94.35%; p=0.025). Four-group analysis also showed differences in BMI (p=0.033) and minimum SpO₂ (p=0.009). AHI correlated negatively with minimum SpO₂ ($\rho=-0.599$; p<0.001). Body weight showed moderate discrimination for AHI ≥ 15 (AUC=0.756, 95% CI 0.594–0.918; cut-off ≥ 90 kg; sensitivity 89.5%, specificity 66.7%; p=0.002), while BMI had AUC=0.721 (p=0.016). After adjustment for age and sex, BMI was not independently associated with AHI ≥ 15 (OR=1.110; 95% CI 0.957–1.287; p=0.167). No significant differences were found in other clinical, metabolic, or echocardiographic variables (p=0.148–1.000). Uric acid was higher in the AHI ≥ 15 group (7.05 ± 1.24 vs 5.05 ± 1.75 mg/dL; p=0.012).

In this sample, OSA was detected in 71.1% and moderate-to-severe OSA in 57.9% of patients, supporting targeted screening. Patients with AHI ≥ 15 had higher body weight and BMI and lower nocturnal SpO₂. A body weight ≥ 90 kg showed moderate discriminatory ability and may serve as a simple screening marker, but requires external validation. The small sample, uneven missing data, lack of multiple-comparison correction, and cross-sectional design limit causal inference; the uric acid difference remains exploratory.