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# Analysis of Sleep Quality and Its Influencing Factors in Patients with Metabolic Syndrome: A Retrospective Cross-Sectional Study

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# Abstract

### Background

To investigate the objective sleep characteristics in patients with metabolic syndrome (MS) and explore the associated influencing factors.

### Methods

A retrospective cross-sectional study evaluated 219 patients (122 non-MS, 97 MS) undergoing polysomnography. Clinical, metabolic, and sleep parameters were analyzed.

### Results

The MS group exhibited significantly deteriorated metabolic and inflammatory profiles. Polysomnography revealed impaired sleep architecture in MS patients, marked by reduced total sleep time, sleep efficiency (SE), and N2/N3 stages, alongside increased wake after sleep onset, N1 stage, REM stage, apnea-hypopnea index (AHI), and micro-arousal index (all $P < 0.05$). Multivariate analysis demonstrated that body mass index (BMI) is an independent risk factor for concurrent severe obstructive sleep apnea (OSA) in MS patients. Additionally, age and homocysteine (Hcy) were identified as independent determinants of SE (all $P < 0.05$).

### Conclusions

MS patients demonstrate profound sleep architecture abnormalities and sleep-disordered breathing, notably sleep fragmentation and diminished deep sleep. BMI independently predicts severe OSA in this population


# Introduction

Metabolic syndrome (MS) is a complex constellation of metabolic disorders, encompassing dyslipidemia, hypertension, obesity, and impaired glucose metabolism, which significantly elevates the risk of chronic conditions such as cardiovascular and cerebrovascular diseases [1]. Driven by economic growth and rising living standards, the widespread adoption of sedentary lifestyles alongside high-calorie, high-fat diets has led to a continuous surge in MS prevalence, establishing it as a major public health concern [2]. Research indicates that patients with MS frequently suffer from sleep disorders, particularly obstructive sleep apnea (OSA). These two conditions interact bidirectionally through mechanisms involving obesity, insulin resistance, intermittent hypoxia, and inflammatory responses [3,4]. However, current literature predominantly focuses on subjective sleep assessments or isolated OSA, leaving a critical gap in our understanding of the objective sleep architecture and its associated factors in patients with MS. Polysomnography (PSG), recognized as the gold standard for evaluating sleep architecture and sleep-disordered breathing events, objectively reflects a patient's true sleep profile [5,6]. Therefore, this study utilizes PSG to evaluate the objective sleep characteristics of patients with MS and

further analyze their associated factors, aiming to provide an evidence base for the early identification and comprehensive management of sleep-related issues in this population.

## Materials and Methods

# 1.1 Study Population

This retrospective cross-sectional study enrolled 219 hospitalized patients who underwent polysomnography (PSG) monitoring at the Affiliated Chaohu Hospital of Anhui Medical University between January 2020 and August 2024. The inclusion criteria were as follows: (1) age ≥ 18 years; (2) availability of complete metabolic parameter records required for the diagnosis of metabolic syndrome (MS) within a valid timeframe in the electronic medical records (defined as within 3 months before or after the date of the PSG examination); and (3) completion of overnight PSG monitoring at our institution (monitoring duration ≥ 6 hours with intact and reliable data). The exclusion criteria included: (1) incomplete PSG monitoring data (e.g., monitoring duration < 6 hours, missing key indicators) or incomplete electronic medical records; and (2) presence of severe organ failure (e.g., New York Heart Association [NYHA] class IV heart failure, end-stage renal disease requiring dialysis), malignancies, or other acute illnesses that significantly affect metabolism and sleep. Based on the presence or absence of MS, the patients were categorized into an MS group (n = 97) and a non-MS group (n = 122). This retrospective study was approved by the Ethics Committee of the Affiliated Chaohu Hospital of Anhui Medical University (Approval No. KYXNF202511-005), and the requirement for written informed consent was waived.

# 1.2 Diagnostic Criteria and Data Collection

## 1.2.1 Diagnostic Criteria for Metabolic Syndrome (MS)

The diagnosis of MS was based on the criteria established by the Chinese Diabetes Society (CDS)[7]. Patients meeting three or more of the following criteria were diagnosed with MS: (1) Body mass index (BMI) ≥ 25 kg/m²; (2) Fasting plasma glucose (FPG) ≥ 6.1 mmol/L, or previously diagnosed diabetes mellitus currently receiving treatment; (3) Hypertension: blood pressure ≥ 140/90 mmHg, or previously diagnosed hypertension currently receiving treatment; (4) Dyslipidemia: triglycerides (TG) ≥ 1.7 mmol/L, and/or high-density lipoprotein cholesterol (HDL-C) ≤ 0.9 mmol/L (in males) or < 1.0 mmol/L (in females).

## 1.2.2 Clinical and Biochemical Data Collection

General patient demographics and clinical characteristics were collected, including age, sex, height, weight, smoking status, alcohol consumption, and histories of hypertension and diabetes mellitus.

Fasting venous blood samples were drawn from all patients in the morning (after an overnight fast of > 8 hours) to measure fasting plasma glucose (FPG), lipid profiles, complete blood counts, uric acid, homocysteine (Hcy), and high-sensitivity C-reactive protein (hs-CRP). The triglyceride-glucose (TyG) index, a well-established surrogate marker for insulin resistance and metabolic risk, was calculated based on the laboratory results. Prior to calculation, the laboratory-measured FPG and triglyceride (TG) values in mmol/L were converted to mg/dL using standard conversion factors (multiplied by 18 for FPG and 88.5 for TG). The TyG index was then calculated using the following formula:

$$\mathrm{TyG} = \ln\left[\frac{\mathrm{TG(mg/dL)} \times \mathrm{FPG(mg/dL)}}{2}\right].$$

# 1.3 Polysomnography (PSG) Monitoring

All patients underwent overnight monitoring during natural sleep using the Compumedics Grael polysomnography system (Australia). Electrodes were placed according to the standard sleep diagnostic montage to simultaneously record physiological signals, including electroencephalogram (EEG), electrooculogram (EOG), submental electromyogram (EMG), electrocardiogram (ECG), thoracoabdominal respiratory movements, and oxygen saturation. Oronasal airflow was monitored via dual channels using a nasal pressure transducer and an oronasal thermal sensor.Upon completion of the monitoring, recordings with good overnight signal quality were manually scored by professional sleep technologists. Sleep staging and associated event analyses were performed in accordance with the scoring manual of the American Academy of Sleep Medicine (AASM). Hypopnea events were scored using the standard criteria, defined as a reduction in airflow accompanied by an oxygen desaturation of ≥ 4%. The core objective parameters extracted included: total sleep time (TST), sleep efficiency (SE), the percentage of TST spent in each sleep stage (N1, N2, N3, and rapid eye movement [REM] stages), apnea-hypopnea index (AHI), lowest oxygen saturation ($LSpO_2$), and arousal index (ArI).

# 1.4 Statistical Analysis

Statistical analyses were performed using SPSS version 26.0 (IBM Corp., Armonk, NY, USA). Continuous variables were assessed for normality using the Shapiro-Wilk test. Normally distributed continuous data were expressed as mean ± standard deviation (SD) and compared between groups using the independent samples t-test. Non-normally distributed continuous variables were presented as median and interquartile range (IQR) and compared using the Mann-Whitney U test. Categorical variables were expressed as frequencies and percentages, with intergroup comparisons conducted using the Pearson Chi-square test. Within the MS group, Spearman rank correlation analysis was performed to evaluate the associations between key PSG parameters (sleep efficiency, AHI, and proportion of N3 stage) and metabolic profiles. To identify independent predictors, binary logistic regression analysis was employed for severe OSA, while multiple linear regression analysis was used to investigate the independent determinants of objective sleep efficiency. Notably, when constructing the multivariate regression models, variables demonstrating a potential association in the univariate analysis ($P < 0.20$), along with

clinically pivotal variables selected a priori based on established cardiovascular and metabolic pathophysiology (such as Hcy and hs-CRP), were forced into the models to rigorously adjust for potential confounding effects. A two-sided P-value < 0.05 was considered statistically significant.

## Results

# 2.1 Comparison of Baseline Characteristics Between the MS and Non-MS Groups

A total of 219 subjects were enrolled in this study, comprising 122 in the non-MS group and 97 in the MS group(Table 1).

**[Insert Table 1 here]**

There were no statistically significant differences in age, low-density lipoprotein cholesterol (LDL-C), or total cholesterol (TC) levels between the two groups (all P > 0.05). Compared with the non-MS group, the MS group exhibited significantly higher proportions of males, hypertension, diabetes mellitus, smoking, and alcohol consumption. Furthermore, the MS group demonstrated significantly elevated body mass index (BMI), fasting plasma glucose (FPG), triglycerides (TG), high-sensitivity C-reactive protein (hs-CRP), homocysteine (Hcy), neutrophil-to-lymphocyte ratio (NLR), TyG index, and uric acid (UA), alongside decreased levels of high-density lipoprotein cholesterol (HDL-C) (all P < 0.05).

# 2.2 Comparison of Polysomnography (PSG) Parameters Between the Two Groups

The comparison of polysomnography (PSG) parameters between the two groups is presented in Table 2.

**[Insert Table 2 here]**

Compared with the non-MS group, the MS group exhibited decreased total sleep time (TST) and sleep efficiency (SE), alongside prolonged wake after sleep onset (WASO) (all P < 0.05). Regarding sleep architecture, the MS group showed an increased proportion of the N1 stage, decreased proportions of the N2 and N3 stages, and an elevated proportion of the REM stage (all P < 0.05). In terms of sleep-disordered breathing and arousal-related parameters, the MS group demonstrated an elevated apnea-hypopnea index (AHI), a decreased lowest oxygen saturation (LSpO2), and an increased arousal index (ArI) (all P < 0.05). Further analysis based on the severity of obstructive sleep apnea (OSA) revealed a statistically significant difference in the distribution of OSA severity between the two groups ($\chi^2$ = 64.547, P < 0.001); the proportion of severe OSA was markedly higher in the MS group than in the non-MS group (61.9% vs. 14.8%).

## 2.3 Correlation Analysis Between Sleep Parameters and Clinical Indicators in Patients with Metabolic Syndrome

Within the MS group (n = 97), Spearman rank correlation analysis was performed to evaluate the associations of core PSG parameters (AHI, SE, and the proportion of N3 stage) with demographic characteristics, metabolic parameters, and inflammatory markers (Table 3).

**[Insert Table 3 here]**

BMI was positively correlated with AHI (r = 0.456, P < 0.001). Age was negatively correlated with SE (r = -0.259, P = 0.011) and the proportion of N3 stage (r = -0.206, P = 0.046). Among the metabolic parameters, triglycerides (TG) showed a positive correlation with SE (r = 0.221, P = 0.033). The correlations between the remaining clinical indicators and the aforementioned core PSG parameters did not reach statistical significance (all P > 0.05).

## 2.4 Independent Determinants of Sleep Quality and Respiratory Events in Patients with Metabolic Syndrome

To identify independent risk factors for concurrent severe OSA (defined as AHI ≥ 30 events/h), a binary logistic regression model was constructed. Variables demonstrating potential statistical significance in the preliminary univariate screening (BMI and history of diabetes mellitus), alongside a priori demographic covariates forced into the model for adjustment (age and sex), were included as independent variables (Table 4).

**[Insert Table 4 here]**

After adjusting for relevant confounding factors, BMI was identified as an independent risk factor for severe OSA (odds ratio [OR] = 1.194, 95% confidence interval [CI]: 1.067–1.338, P = 0.002). Age, sex, and history of diabetes mellitus did not exhibit statistically significant associations with severe OSA (all P > 0.05).

A multiple linear regression model was constructed with sleep efficiency (SE) as the dependent variable, incorporating age, sex, BMI, TG, history of diabetes mellitus (DM), hs-CRP, and Hcy as independent variables (Table 5).

**[Insert Table 5 here]**

The results demonstrated that age and Hcy were independent determinants of SE in patients with MS. Specifically, age was negatively associated with SE (β = -0.310, *P* = 0.019), and Hcy also exhibited a negative association with SE (β = -0.306, *P* = 0.017). Conversely, the associations of sex, BMI, TG, DM, and hs-CRP with SE did not reach statistical significance (all *P* > 0.05).

# Discussion

This study utilized polysomnography (PSG) to evaluate the objective sleep quality of patients with metabolic syndrome (MS) and further investigated the associated factors within the MS population. Our results indicate that MS patients exhibit significantly higher levels of obesity, glucose and lipid metabolic disturbances, TyG index, hs-CRP, NLR, Hcy, and uric acid compared to the non-MS group. This suggests that MS represents not merely a cluster of metabolic disturbances but a state of intense chronic low-grade inflammation and oxidative stress[8]. Previous research has established that such systemic inflammation and metabolic dysregulation are critical drivers of cardiovascular and cerebrovascular diseases[1–8]. Our findings further corroborate that this pathophysiological state also fosters severe sleep impairments.

Against this background of metabolic and inflammatory abnormalities, the objective sleep architecture of patients was significantly disrupted. Compared with the non-MS group, the MS group demonstrated decreased sleep efficiency (SE), prolonged wake after sleep onset (WASO), an increased proportion of light sleep (N1 stage), and a marked decline in deep sleep (N3 stage). N3 sleep plays a pivotal role in maintaining metabolic homeostasis, clearing cerebral metabolic waste, and facilitating physiological repair[6]. Consequently, the reduction in N3 sleep and the increase in the arousal index are not only objective hallmarks of sleep fragmentation and quality deterioration but may also, in turn, exacerbate metabolic disturbances. Clinical controlled studies have similarly confirmed that MS patients exhibit more pronounced N1 stage prolongation and N3 stage shortening compared to non-MS subjects, aligning with our PSG findings[5–10]. Furthermore, recent research has demonstrated significant links between sleep architecture parameters—specifically reduced deep sleep and increased arousals—and metabolic indicators[5], suggesting a vicious cycle between sleep structure damage and metabolic abnormalities.

Beyond sleep architecture disruption, our study confirmed that MS patients suffer from more severe sleep-disordered breathing. The MS group exhibited an elevated AHI, decreased minimum oxygen saturation, and a remarkably high prevalence of severe OSA (61.9%). Recent clinical studies and large-scale population-based research consistently show that as OSA severity increases, the prevalence of metabolic syndrome rises significantly [12], with worsening AHI and hypoxemia being closely associated with insulin resistance and the deterioration of metabolic components such as FPG, TG, and uric acid[9, 11, 13] .

Our correlation analysis showed that BMI was positively correlated with AHI and identified BMI as an independent risk factor for severe OSA, further validating the mechanism where obesity drives sleep-disordered breathing through anatomical narrowing and adipokine abnormalities[3, 4, 10]. Notably, although triglycerides (TG) initially showed a positive correlation with SE, this paradoxical result likely stemmed from confounding effects. Patients with severe OSA and poor sleep quality often take lipid-lowering medications, which lowers their TG levels; additionally, younger individuals may have higher

sleep efficiency but poorer lifestyles leading to elevated TG. Indeed, in the multiple linear regression model, the independent effect of TG on SE vanished after adjusting for age and BMI, confirming that the initial correlation was a "spurious correlation" influenced by age and clinical medication use.

Further multivariate regression confirmed that, beyond age-related decline, significantly elevated baseline homocysteine (Hcy) is a core independent risk factor for impaired SE in MS patients. This suggests that endothelial dysfunction and oxidative stress, represented by Hcy, may be critical pathophysiological links mediating sleep quality decline in MS. Systematic reviews indicate that Hcy levels in OSAHS patients are significantly higher than in healthy controls and correlate with AHI severity[14]. Mechanistically, Hcy induces vascular endothelial apoptosis and microcirculatory ischemia while acting as a potent inducer of reactive oxygen species (ROS) and oxidative stress[15]. In the context of MS, high Hcy-driven central micro-injury and oxidative imbalance likely lower the cerebral arousal threshold, causing frequent microarousals and fragmented sleep.

## Conclusion

In conclusion, objective sleep impairment in MS patients results from the combined effects of obesity-related airway obstruction, metabolic disturbances, and impaired vascular endothelial function. Our results directly support the role of obesity in exacerbating respiratory events and the potential link between Hcy-related oxidative stress and diminished sleep quality[16]. Early PSG assessment and comprehensive intervention should be prioritized for MS patients with high BMI and elevated Hcy levels. Limitations of this study include its single-center cross-sectional design, which precludes causal inference, the lack of systematic data on lipid-lowering medications, and the relatively limited sample size. Future large-scale prospective longitudinal studies are needed to further elucidate the bidirectional regulation among metabolism, inflammation, and objective sleep architecture.

## Declarations

# Ethics approval and consent to participate:

The study was conducted in accordance with the Declaration of Helsinki. Ethical approval was obtained from the Ethics Committee of the Affiliated Chaohu Hospital of Anhui Medical University (Approval No. KYXNF202511-005). Due to the retrospective nature of this study, the requirement for informed consent was waived by the Ethics Committee of the Affiliated Chaohu Hospital of Anhui Medical University.

# Consent for publication:

Not applicable.

# Availability of data and materials:

The datasets generated and analysed during the current study are not publicly available due to hospital policy restrictions but are available from the corresponding author on reasonable request.

# Competing interests:

This study is supported by the Major Natural Science Research Project of Anhui Provincial Universities(No.2023AH040088), Discipline Co-construction Project of Anhui Medical University .The authors declare that they have no other competing interests.

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# Authors' contributions:

Bowen CHANG(First Author): Conceptualization, Data Curation、Methodology, Investigation, Formal Analysis, Investigation、Writing - Original Draft;

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Xianli PAN: Data Curation , Investigation

ZengFeng SU、(Corresponding Author):Conceptualization, Funding Acquisition, Resources, Supervision, Writing - Review & Editing.

All authors read and approved the final manuscript."

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# Tables

Table 1
Comparison of baseline characteristics between the MS and non-MS groups

| Variables | Non-MS (n = 122) | MS(n = 97) | Statistic | P |
|---|---|---|---|---|
| Age | 54.82 ± 17.75 | 57.33 ± 12.86 | t = − 1.213 | 0.227 |
| Male, n(%) | 51(41.8%) | 70(72.2%) | $\chi^2$ = 20.147 | < 0.001 |
| BMI(kg/m²) | 22.71 ± 3.35 | 28.76 ± 4.36 | t = − 11.110 | < 0.001 |
| FPG(mmol/L) | 4.90(4.60–5.39) | 5.70(5.10–7.30) | Z = − 6.812 | < 0.001 |
| TG(mmol/L) | 1.14(0.83–1.51) | 2.16(1.53–2.97) | Z = − 8.047 | < 0.001 |
| HDL-C(mmol/L) | 1.21(1.05–1.44) | 0.90(0.77–1.01) | Z = − 7.973 | < 0.001 |
| LDL-C(mmol/L) | 2.41 ± 0.85 | 2.19 ± 0.77 | t = 1.890 | 0.060 |
| TC(mmol/L) | 4.27 ± 1.07 | 4.19 ± 0.98 | t = 0.556 | 0.579 |
| TyG | 8.5(8.1–8.7) | 9.2(8.9–9.6) | Z = − 9.272 | < 0.001 |
| UA(μmol/L) | 309(241–371) | 365(295–433) | Z = − 4.481 | < 0.001 |
| hs-CRP(mg/L) | 0.8(0.6–1.9) | 2.3(1.1–3.8) | Z = − 4.218 | < 0.001 |
| Hcy(μmol/L) | 11.0(9.4–14.4) | 12.6(10.8–16.1) | Z = − 2.971 | 0.003 |
| NLR | 1.9(1.4–2.7) | 2.1(1.6–3.0) | Z = − 2.186 | 0.029 |
| Hypertension, n(%) | 37(30.3%) | 76(78.4%) | $\chi^2$ = 49.898 | < 0.001 |
| DM, n(%) | 10(8.2%) | 41(42.3%) | $\chi^2$ =35.114 | < 0.001 |
| Smoking, n(%) | 10(8.2%) | 36(37.1%) | $\chi^2$ = 27.231 | < 0.001 |
| Drinking, n(%) | 8(6.6%) | 35(36.1%) | $\chi^2$ = 29.852 | < 0.001 |

Notes:Data are presented as mean ± standard deviation (SD), median (interquartile range, IQR), or n (%). Differences between groups were evaluated using the independent samples t-test, Mann-Whitney U test, or Pearson

Table 2
Comparison of polysomnography parameters between the MS and non-MS groups

| **Variables** | **Non-MS (n = 122)** | **MS(n = 97)** | **Statistic (Z/$\chi^2$)** | **P** |
|---|---|---|---|---|
| TST(min) | 452.0 (404.0, 485.5) | 420.8 (357.4, 451.0) | -3.401[a] | < 0.001 |
| SE | 0.87 (0.78, 0.92) | 0.79 (0.68, 0.87) | -4.466[a] | < 0.001 |
| WASO(min) | 44.0 (23.0, 77.5) | 85.8 (50.6, 131.0) | -4.914[a] | < 0.001 |
| Stage N1 (%) | 14.6 (8.9, 21.9) | 28.9 (19.0, 44.0) | -6.882[a] | < 0.001 |
| Stage N2 (%) | 59.5 (49.3, 68.7) | 47.3 (36.1, 56.0) | -5.336[a] | < 0.001 |
| Stage N3 (%) | 9.4 (1.2, 19.6) | 4.2 (0.2, 14.2) | -2.280[a] | 0.023 |
| Stage REM (%) | 11.9 (6.9, 18.3) | 16.1 (10.2, 20.8) | -3.039[a] | 0.002 |
| AHI (events/h) | 8.7 (4.2, 18.3) | 37.3 (20.0, 67.0) | -8.386[a] | < 0.001 |
| $LSpO_2$(%) | 90.0 (85.0, 92.0) | 81.5 (70.0, 87.3) | -6.910[a] | < 0.001 |
| ArI (events/h) | 12.2 (6.5, 18.3) | 28.0 (16.3, 48.9) | -6.596[a] | < 0.001 |
| OSA severity,n (%) | | | 64.547[b] | < 0.001 |
| Normal (AHI < 5) | 38 (31.1%) | 2 (2.1%) | | |
| Mild (5 ≤ AHI < 15) | 44 (36.1%) | 18 (18.6%) | | |
| Moderate (15 ≤ AHI < 30) | 22 (18.0%) | 17 (17.5%) | | |
| Severe(AHI ≥ 30) | 18 (14.8%) | 60 (61.9%) | | |

Notes: Data are expressed as median (interquartile range, IQR) or n (%). [a]Analyzed using the Mann-Whitney U test (Z value is presented). [b] Analyzed using the Pearson Chi-square test .

Table 3
Correlations between core PSG parameters and clinical, metabolic, and inflammatory markers in the MS group

| Variables | AHI(r, P) | SE(r, P) | N3(r, P) |
|---|---|---|---|
| Age | -0.117,0.252 | -0.259,0.011 | -0.206,0.046 |
| BMI | 0.456, < 0.001 | -0.122, 0.245 | -0.018, 0.868 |
| FPG | 0.074, 0.472 | -0.026, 0.800 | -0.055, 0.598 |
| TG | 0.048,0.648 | 0.221,0.033 | -0.132,0.210 |
| TyG | 0.093,0.376 | 0.180,0.085 | -0.177,0.091 |
| UA | 0.092,0.378 | 0.014,0.895 | -0.119,0.258 |
| hs-CRP | -0.015,0.907 | -0.100,0.430 | 0.214,0.090 |
| Hcy | -0.079,0.463 | -0.154,0.149 | -0.075,0.490 |
| NLR | 0.141,0.173 | -0.093,0.374 | -0.085,0.415 |

Table 4
Binary logistic regression analysis of independent risk factors for severe OSA in patients with MS

| Variables | B | SE | Wald$\chi^2$ | P | OR(Exp (B)) | CI |
|---|---|---|---|---|---|---|
| BMI | 0.187 | 0.061 | 9.292 | 0.002 | 1.194 | (1.067, 1.338) |
| Age | -0.012 | 0.019 | 0.406 | 0.524 | 0.988 | (0.951, 1.027) |
| Sex (Male | 0.375 | 0.510 | 0.541 | 0.462 | 1.455 | (0.476, 4.400) |
| DM | 0.648 | 0.486 | 1.776 | 0.183 | 1.911 | (0.806, 4.516) |
| Constant | -4.389 | 2.411 | 3.315 | 0.069 | 0.012 | (0.0001, 1.508) |
| Notes: The dependent variable is the presence of severe OSA (AHI ≥ 30 events/h). | | | | | | |

Table 5
Multiple linear regression analysis of independent determinants for sleep efficiency in patients with MS

| Variables | B | β | t | P | VIF |
|---|---|---|---|---|---|
| Constant | 1.144 | — | 13.914 | < 0.001 | — |
| Age | -0.003 | -0.310 | -2.394 | 0.019 | 1.155 |
| Hcy | -0.007 | -0.306 | -2.433 | 0.017 | 1.091 |
| Sex (Male) | 0.006 | 0.051 | 0.407 | 0.685 | 1.109 |
| BMI | 0.002 | 0.165 | 1.256 | 0.212 | 1.201 |
| TG | 0.004 | 0.101 | 0.817 | 0.416 | 1.050 |
| DM | 0.018 | 0.134 | 1.077 | 0.285 | 1.060 |
| hs-CRP | -0.001 | -0.116 | -0.925 | 0.358 | 1.078 |
| Notes: B: unstandardized coefficient; β: standardized coefficient; VIF: variance inflation factor. | | | | | |