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UDC: 616.2-008.4::612.821.7
DOI: https://doi.org/10.2298/VSP210727093J

Correlation between oxidative stress and cognitive impairment in patients with obstructive sleep apnea-hypopnea syndrome

Korelacija između oksidativnog stresa i kognitivnog oštećenja kod bolesnika sa sindromom opstruktivnog poremećaja disanja (apneje i hipopneje) tokom spavanja

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Abstract

Background/Aim. It is necessary to find relevant oxidative stress markers for predicting the severity of obstructive sleep apnea-hypopnea syndrome (OSAHS), a sleep disorder-related respiratory disease. The aim of the study was to investigate if there is a correlation between oxidative stress and cognitive impairment in OSAHS patients. **Methods.** A total of 220 patients were divided into the group of snoring patients, the group with mild to moderate OSAHS, and the group with severe OSAHS according to polysomnography (PSG). Apnea-hypopnea index (AHI), oxygen desaturation index (ODI), and baseline data were monitored. Oxidative stress indices were measured by colorimetry from blood samples taken early in the morning. The patients were then divided into the group with normal cognition and cognitive impairment group based on mini-mental state examination (MMSE) and Montreal cognitive assessment (MoCA). Independent risk factors for cognitive impairment were analyzed by multivariate logistic regression. The correlation between oxidative stress and cognitive impairment was analyzed by Pearson's method. Receiver operating characteristic (ROC) curves made it possible to analyze the efficiency of oxidative stress combined with detection for assessing cognitive impairment in OSAHS patients. **Results.** The snoring group, mild to moderate OSAHS group, and severe OSAHS group had significantly different snoring loudness, body mass index (BMI), AHI, ODI, MoCA, and MMSE scores, and levels of malondialdehyde (MDA), glutathione peroxidase (GSH-Px), and superoxide dismutase (SOD) ($p < 0.05$). The cognitive impairment group and group with normal cognition had different BMI, GSH-Px, MDA, SOD, neuroglobin, hypoxia-inducible factor, AHI, and lowest nocturnal oxygen saturation ($p < 0.05$ or $p < 0.01$) levels. BMI, GSH-Px, MDA, SOD, neuroglobin, hypoxia-inducible factor, AHI, and lowest nocturnal oxygen saturation were independent risk factors for cognitive impairment. The MoCA and MMSE scores of cognitive impairment had positive correlations with GSH-Px and SOD but negative correlations with MDA ($p < 0.05$). The area under the ROC curve of GSH-Px, MDA, and SOD and their combination for prediction of cognitive impairement were 0.670, 0.702, 0.705, and 0.836, respectively. **Conclusion.** Oxidative stress may be the biochemical basis of cognitive impairment in OSAHS patients.

Key words:
cognitive dysfunction; oxidative stress; sleep apnea, obstructive; sleep apnea syndromes.

Apstrakt

Uvod/Cilj. Neophodno je pronaći odgovarajuće markere oksidativnog stresa za predviđanje težine sindroma opstruktivne apneje-hipopneje u snu (SOAHS), respiratorne bolesti povezane sa poremećajem spavanja. Cilj rada je bio da se istraži da li postoji korelacija između oksidativnog stresa i kognitivnog oštećenja kod bolesnika sa SOAHS. **Metode**. Ukupno 220 bolesnika podeljeno je na grupu koja hrče, grupu sa blagim do umerenim SOAHS i grupu sa teškim SOAHS, izmereno primenom polisomnografije (PSG). Praćeni su indeks apneje-hipopneje (IAH), indeks desaturacije kiseonikom (IDK) i osnovni podaci. Indeksi oksidativnog stresa mereni su metodom kolorimetrije iz uzoraka krvi uzetih u ranim jutarnjim časovima. Bolesnici su dalje bili podeljeni na osnovu *mini-mental state examination* (MMSE) i *Montreal cognitive assessment* (MoCA) procena u grupu sa normalnom kognicijom i grupu sa kognitivnim oštećenjima. Nezavisni faktori rizika od kognitivnog oštećenja analizirani su multivarijantnom logističkom regresijom. Korelacija između oksidativnog stresa

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i kognitivnih oštećenja analizirana je Pirsonovom metodom. Krive *receiver operating characteristic* (ROC) omogućile su analizu efikasnosti oksidativnog stresa u kombinaciji sa procenom kognitivnog oštećenja kod bolesnika sa SOAHS. **Rezultati**. Grupa koja je hrkala, grupa sa blagim do umerenim oblikom SOAHS i grupa sa teškim oblikom SOAHS imale su značajno različite jačine hrkanja, različite vrednosti indeksa telesne mase (ITM), IAH, IDK, MoCA i MMSE i različite nivoe malondialdehida (MDA), glutation peroksidaze (GSH-Px) i superoksid dismutaze (SOD) ($p < 0,05$). Grupa sa kognitivnim oštećenjem i grupa sa normalnom kognicijom imale su različite vrednosti ITM, GSH-Px, MDA, SOD, neuroglobina, hipoksijom izazvanog faktora, IAH i najnižu noćnu zasićenost kiseonikom ($p < 0,05$ ili $p < 0,01$). Nezavisni faktori rizika od kognitivnog oštećenja bili su ITM, GSH-Px, MDA, SOD, neuroglobin, hipoksijom izazvan faktor, AHI i najniža noćna zasićenost kiseonikom. Rezultati skorova kognitivnog oštećenja MoCA i MMSE pozitivno su korelisali sa GSH-Px i SOD, ali je korelacija sa MDA bila negativna ($p < 0,05$). Površina ispod ROC krive za GSH-Px, MDA, SOD i njihove kombinacije za predviđanje kognitivnog oštećenja iznosila je 0,670, 0,702, 0,705 i 0,836, redom. **Zaključak**. Oksidativni stres može biti biohemijska osnova kognitivnog oštećenja kod bolesnika sa OSAHS.

Ključne reči:
kognicija, poremećaji; stres, oksidativni; apneja u snu, opstruktivna; apneja u snu, sindromi.

Introduction

Obstructive sleep apnea-hypopnea syndrome (OSAHS) is a type of sleep disorder-related respiratory disease whose influence on sleep quality and mood is well known. It is featured by increased resistance in the upper respiratory tract that occurs repeatedly during sleep, resulting in apnea, hypopnea, and sleep fragmentation at night [1]. OSAHS is prominently associated with the increased risk of death and occurrence of diseases such as cardiovascular (CV) and cerebrovascular (CEV) diseases (CVD and CEVD, respectively), metabolic disorders, and neurocognitive impairment, among which neurocognitive impairment has been paid progressively more attention to by relevant scholars in recent years [2]. Patients with OSAHS suffer from defects in attention span, distraction, sustained attention, working memory, visual space, and executive function. Cognitive impairment has always been a non-negligible clinical manifestation of patients with OSAHS. The underlying mechanisms of neurocognitive impairment related to sleep apnea may include intermittent hypoxemia, sleep fragmentation, neuroinflammation, CEV changes, and ischemic preconditioning [3]. It has been revealed by some research that sleep disorders can induce oxidative stress, and progressively more attention has been paid to the relationship between oxidative stress and nervous system damage [4]. Oxidative stress is induced generally because the production of oxygen free radicals in the body exceeds the body's endogenous antioxidant capacity (such as reduction of sleep time), resulting in the process of tissue damage, which is essential for the development of neurodegenerative diseases. The association of mechanisms of OSAHS-related cognitive impairment with inflammatory response, oxidative stress, and brain injury has been confirmed [5]. However, there are still controversies over a series of criteria of oxidative stress, the role of the new biomarkers in OSAHS patients, and the best biomarker for cognition [6]. Glutathione, superoxide dismutase (SOD), malondialdehyde (MDA), and advanced oxidation protein products are recognized serum biomarkers of oxidative stress [7]. However, not all the results of studies can confirm the presence of oxidative stress damage in patients with OSAHS, which may be related to the efficacy of the oxidative stress index adopted. Hence, it is of important clinical value to search for oxidative stress markers able to predict the severity of sleep apnea.

The purpose of the present study is to evaluate the cognitive function of patients with OSAHS, group the patients according to their cognitive status, and make a comparison to identify the factors affecting cognitive impairment in OSAHS patients. In addition, the correlations between oxidative stress indices and cognitive impairment were evaluated to provide a reliable basis for early detection and follow-up treatment of cognitive impairment in OSAHS patients.

Methods

A total of 220 OSAHS patients diagnosed and treated in our hospital from January 2016 to June 2018 were enrolled. All of them were confirmed by polysomnography (PSG) of 7hrs sleep at night as meeting the diagnostic criteria in the Guidelines for diagnosis and treatment of OSAHS (revised in 2011). According to PSG results the patients were divided into snoring group (n = 60), mild to moderate OSAHS group (n = 80), and severe OSAHS group (n = 80). Among the subjects, there were 150 males and 70 females, aged 20–60 years old. The general information and laboratory test data of the patients were obtained by consulting the relevant case data of patients in our department and treatment sites. This study was reviewed and approved by the Medical Ethics Committee, and all patients and their family members signed the informed consent.

Inclusion criteria were: (1) patients who were first diagnosed with OSAHS and received no OSAHS-related surgery, (2) those whose main complaints were sleep snoring and sleep suffocation, (3) those who meet the diagnostic criteria for OSAHS according to the Guidelines for diagnosis and treatment of OSAHS [8], and (4) those who were able to cooperate and finish the mini-mental state examination (MMSE) and Montreal cognitive assessment (MoCA) (Chinese versions). Exclusion criteria were: (1) patients with a history of CEVD or other severe organ diseases, (2) those with vascular and other dementias, Parkinson's disease, or other neuropsychiatric diseases such as anxiety and depression, or (3) those dependent on alcohol or diazepam drugs, or with communication disorders.

All the patients were asked in detail about their medical history to reconfirm whether they met the inclusion criteria, and general data such as height, weight, neck circumference, and years of education were obtained. Body mass index (BMI) was calculated based on height and weight as follows: BMI = weight (kg)/height^2 (m^2). Epworth sleepiness scale (ESS) was adopted for evaluating daytime sleepiness, which involved a total of 8 scenes, including sitting and reading, watching television, sitting inactively in a public place, riding as a passenger in a car for an hour, lying down to rest in the afternoon when circumstances permit, sitting and talking to someone, sitting quietly after lunch, and sitting in a car while stopping for a few minutes in traffic. A higher score indicated more severe daytime sleepiness.

Alice LE PSG system (Philips Respironics) was applied for monitoring the PSG of the subjects overnight, and the monitoring time was ≥ 7 hrs. The recorded data were automatically analyzed by the G3 software system and reviewed frame by frame by the same professional sleep physician. In addition, two indices were monitored: the apnea-hypopnea index (AHI) – the number of apneas or hypopneas recorded during the study per hour of sleep (generally expressed as the number of events per hour) and oxygen desaturation index (ODI). Desaturation episodes are generally described as a decrease in the mean oxygen saturation of ≥ 4% (over the last 120 sec) that last for at least 10 sec [9]. After the patients' conditions were stable, MMSE and MoCA scores were evaluated by the same physician in a quiet room. MMSE was used to evaluate cognitive function involving memory, orientation, attention and calculation, recall, and language abilities. There were 30 questions in total, with a total score of 30 points (1 point for each correct answer and 0 points for each wrong answer). The higher the score was, the better the cognitive function would be. MoCA can be used as a simple and sensitive neurocognitive assessment tool to detect cognitive impairment in patients with OSAHS, which should be completed within 10 min according to the evaluation criteria. It evaluates the functions of 7 cognitive regions of patients, including visual space and execution, naming, attention, language, abstraction, memory and delayed recall, and time and place orientation. A total score ≥ 26 points suggests normal cognition. It is a screening tool for mild cognitive impairment.

In the early morning, 3 mL of fasting blood was drawn from the median cubital vein of each patient in the three groups, loaded in an anticoagulant tube, and centrifuged at 3,000 rpm for 15 min. Then the supernatant was collected and stored in a refrigerator at -20 °C for later detection. According to the instructions of corresponding kits (Beijing Zhongshan Golden Bridge Biotechnology Co., Ltd., China), the activities of SOD and serum glutathione peroxidase (GSH-Px) were detected by colorimetry, and the level of MDA was measured by the thiobarbituric acid method.

SPSS 25.0 software (IBM Corp, Armonk, NY, USA) was used for statistical analysis. All data were tested for homogeneity of variance and normal distribution, and the normally distributed measurement data were expressed as mean ± standard deviation. An independent sample *t*-test was conducted for comparison between groups, and F-test was adopted for comparison among multiple groups. The measurement data that did not conform to normal distribution were represented as median (P_{25}, P_{75}), and the Kolmogorov-Smirnov test was used for comparison between groups. The qualitative data were expressed as frequency or percentage, and the χ^2 test was performed for comparison between groups. In combination with clinical factors, variables with significant differences in univariate analysis were taken as candidate variables and introduced into the multivariate logistic regression model of cognitive impairment to establish a prediction model aiming to predict the probability of cognitive impairment in patients with OSAHS. The predictive value of oxidative stress-related indices for prediction was analyzed by the receiver operating characteristic (ROC) curve. The area under the ROC curve (AURC) of each index was calculated, and the diagnostic efficiency was higher when AURC was closer to 1. AURC had low predictive accuracy when it was 0.5–0.7, it was relatively accurate when AURC was 0.7–0.9, and it was highly accurate when AURC exceeded 0.9. AURC of 0.5 indicated that the diagnostic method was completely ineffective and had no diagnostic value. Sensitivity = number of true positive cases/(number of true positive cases + number of false negative cases) × 100%. Specificity = number of true negative cases/(number of true negative cases + number of false positive cases) × 100%. The difference was considered statistically significant when $p < 0.05$.

Results

There were no significant differences in the general data such as gender, age, years of education, neck circumference, and ESS score among the snoring group, mild to moderate OSAHS group, and severe OSAHS group ($p > 0.05$), suggesting that the groups were comparable in the follow-up indices. Significant differences were observed in snoring loudness, BMI, AHI, ODI, MoCA, and MMSE scores, and levels of MDA, GSH-Px, and SOD among the three groups of patients. Compared with the snoring group, the mild to moderate OSAHS group had slightly higher snoring loudness, BMI, AHI, ODI, and level of MDA ($p > 0.05$), and the severe OSAHS group had significantly higher values ($p < 0.05$). Besides, compared with the snoring group, the SOD and GSH-Px levels and MoCA and MMSE scores of the mild to moderate OSAHS group were slightly lower ($p > 0.05$), and those of severe OSAHS group were significantly lower ($p < 0.05$) (Table 1).

There were no statistically significant differences in age and gender between the cognitive impairment group and the normal cognitive group ($p > 0.05$). GSH-Px, MDA, neuroglobin, hypoxia-inducible factor, and AHI were significantly higher, while BMI, SOD, and lowest nocturnal oxygen saturation were significantly lower in the cognitive impairment group than those in the normal cognitive group ($p < 0.05$ or $p < 0.01$).

Establishment of prediction model: Multivariate analysis was performed based on the factors influencing cognitive impairment in the 220 OSAHS patients in our hospital. With the presence of cognitive impairment as the dependent variable (normal cognitive group = 0, cognitive impairment group = 1), the variables with statistically significant differences shown in

univariate analysis were included in the analysis. The results showed that BMI, GSH-Px, MDA, SOD, neuroglobin, hypoxia-inducible factor, AHI, and lowest nocturnal oxygen saturation were independent risk factors for cognitive impairment in patients with OSAHS (Figure 1).

The results of Pearson's correlation analysis demonstrated that MoCA and MMSE scores of cognitive impairment in OSAHS patients had positive correlations with GSH-Px and SOD and negative associations with MDA ($p < 0.05$) (Table 2).

ROC curve analysis revealed that when the cut-off values of GSH-Px, MDA, and SOD were 251.07 U, 9.46 nmol/mL, and 98.32 mmol/L, respectively, the AURC were 0.670, 0.702, and 0.705, respectively, showing predictive values for cognitive impairment. Moreover, the AURC of the three parameters combined for prediction was 0.836 ($p < 0.001$), indicating that the diagnostic value of the combination of the three indices is higher (Table 3, Figure 2).

Table 1

Baseline clinical data

Parameter	Snoring group (n = 60)	Mild to moderate OSAHS group (n = 80)	Severe OSAHS group (n = 80)	*p*
Age (years)	46.3 ± 10.5	50.0 ± 10.3	51.5 ± 12.5	0.154
Male/female	41/19	53/27	56/24	0.878
Years of education (years)	10.7 ± 2.4	11.02 ± 2.56	10.94 ± 2.63	0.115
Snoring loudness (dB)	6.45 ± 1.48	6.98 ± 1.77	8.03 ± 1.74	0.002
Neck circumference (cm)	39.82 ± 3.68	40.03 ± 3.06	39.45 ± 4.13	0.097
ESS score	7.06 (3.15, 10.28)	7.25 (2.94, 10.33)	4.26 (3.15, 10.58)	0.329
BMI (kg/m^2)	25.51 ± 3.69	27.02 ± 4.34	28.25 ± 4.83	0.009
AHI (times/h)	1.81 ± 0.83	13.99 ± 3.06	53.68 ± 17.92	< 0.001
ODI (times/h)	7.25 (3.24, 11.25)	29.21 (14.54, 43.22)	64.53 (41.52, 82.58)	< 0.001
MoCA score	26.72 ± 0.53	24.86 ± 1.32	24.01 ± 1.00	0.014
MMSE score	29.58 ± 1.06	28.26 ± 1.84	27.32 ± 2.95	0.021
MDA (nmol/mL)	7.52 ± 1.80	10.64 ± 1.73	12.00 ± 2.01	0.003
GSH-Px (U)	283.49 ± 30.52	238.92 ± 31.67	229.67 ± 28.13	0.011
SOD (U/L)	117.16 ± 20.35	99.57 ± 18.62	86.21 ± 10.83	0.009

Results are given as number of patients or mean ± standard deviation or median (25th percentile, 75th percentile). OSAHS − obstructive sleep apnea-hypopnea syndrome; ESS − Epworth sleepiness scale; BMI – body mass index; AHI – apnea-hypopnea index; ODI – oxygen desaturation index; MoCA – Montreal cognitive assessment; MMSE – mini-mental state examination; GSH-Px – glutathione peroxidase; MDA − malondialdehyde; SOD − superoxide dismutase.

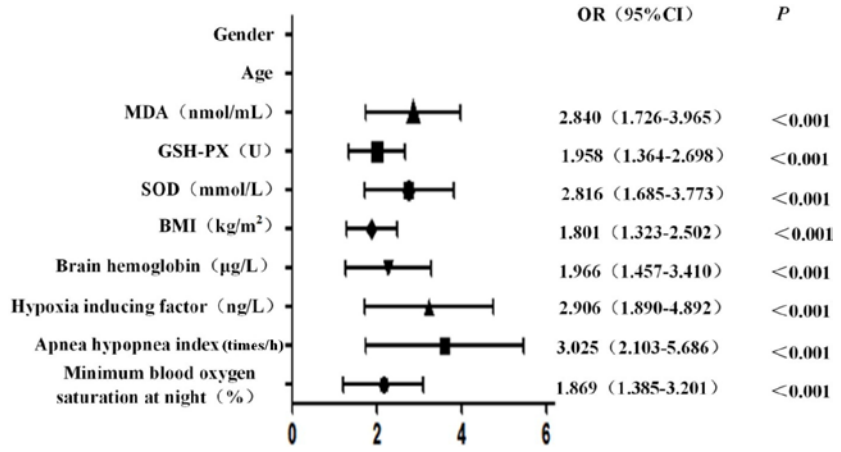


Fig. 1 – Forest plot of prediction model of cognitive impairment in patients with OSAHS. OSAHS − obstructive sleep apnea-hypopnea syndrome; MDA − malondialdehyde; GSH-PX – glutathione peroxidase; SOD − superoxide dismutase; BMI – body mass index; OR – odds ratio; CI – confidence interval.

Table 2

Correlation between cognitive impairment and oxidative stress

Score	GSH-Px		MDA		SOD	
	r	*p*	*r*	*p*	*r*	*p*
MoCA	0.421	0.013	-0.536	< 0.001	0.354	0.013
MMSE	0.625	0.004	-0.378	0.031	0.213	0.047

MoCA – Montreal cognitive assessment; MMSE – mini-mental state examination. GSH-PX – glutathione peroxidase; MDA − malondialdehyde; SOD − superoxide dismutase; *r* – Pearson's correlation coefficient.

Table 3

Predictive values of oxidative stress indices

Diagnostic index	AUC	95% CI	Cut-off	*p*	Sensitivity (%)	Specificity (%)
GSH-Px (U)	0.670	0.591–0.748	251.07	< 0.001	63.39	75.68
MDA (nmol/mL)	0.702	0.623–0.781	9.46	< 0.001	68.96	75.41
SOD (U/L)	0.705	0.629–0.782	98.32	< 0.001	67.53	76.27
Combination	0.836	0.781–0.889	–	< 0.001	70.16	83.24

MDA – malondialdehyde; GSH-Px – glutathione peroxidase; SOD − superoxide dismutase.; AUC – area under curve; CI – confidence interval.

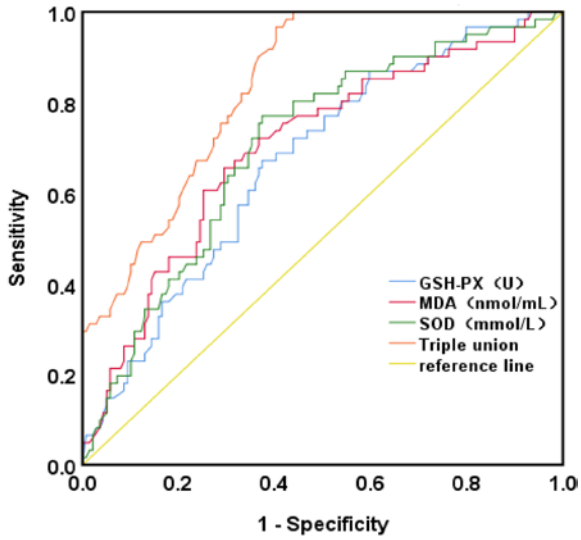


Fig. 2 – ROC curves for predictive values of oxidative stress indices for cognitive impairment in patients with obstructive sleep apnea-hypopnea syndrome.
GSH-PX – glutathione peroxidase; MDA − malondialdehyde; SOD − superoxide dismutase.

Discussion

OSAHS, a chronic disease characterized by recurrent partial or complete airflow obstruction during sleep, is increasingly becoming the cause of morbidity and death. During sleep, the muscle regulation function of the nervous system to the upper airway is decreased, and the upper airway becomes narrow, which can cause a partial or complete collapse of the airway, resulting in hypoventilation. If the airway is completely obstructed, it will lead to apnea [10]. OSAHS can induce damage to multiple systems of the whole body, and when the nervous system is injured, the major manifestation is cognitive impairment in the patients. Metabolic disorders and endocrine and neurological dysfunctions are the pathophysiological causes of OSAHS. The major inducing factor of OSAHS is concentric obesity with familial aggregation [11]. Besides daytime sleepiness and lack of cognitive ability, the clinical manifestations of OSAHS also include CV, respiratory, and nervous system injuries. Moreover, OSAHS is also an independent risk factor for morbidity and death from CVD, hypertension, and CEVD (such as atherosclerosis, arrhythmia, and ischemic heart disease), seriously affecting people's health and quality of life [12]. Some patients with severe cognitive impairment caused by OSAHS may develop Alzheimer's disease [13]. It is believed that the increased risk of CVD, CEVD, respiratory failure, and cognitive impairment is mediated by several mechanisms, such as sympathetic activation and oxidative stress [14]. It is speculated that the cause of oxidative stress in patients may be related to intermittent hypoxia, increased sympathetic excitability, and sleep disorder. There are few reports on the possible role of oxidative stress in the pathogenesis of cognitive impairment in patients with OSAHS [1]. Therefore, in this study, the indices of oxidative stress were measured, factors for cognitive impairment in patients with OSAHS were analyzed, and whether oxidative stress is connected with cognitive impairment in patients with OSAHS was explored.

In recent years, the influencing factors and pathogenesis of cognitive impairment in patients with OSAHS have been extensively studied [15]. The factors influencing cognitive impairment in patients with OSAHS include education, obesity, and intermittent hypoxia at night [16]. Young et al. [17] found that age, BMI, and lowest oxygen saturation are risk factors for cognitive impairment in patients with OSAHS. In this study, 220 patients were enrolled and divided into three groups, i.e., the snoring group, the mild to moderate OSAHS group, and the severe OSAHS group. These results suggest that patients with OSAHS suffer from cognitive impairment, which positively correlates with the severity of OSAHS. According to the analysis of risk factors for cognitive impairment, BMI, GSH-Px, MDA, SOD, neuroglobin,

hypoxia-inducing factor, AHI, and lowest nocturnal oxygen saturation were independent risk factors for cognitive impairment in patients with OSAHS, in line with the results of a previous study [17]. The identification of these high-risk factors is helpful for screening the high-risk factors of patients as soon as possible and reducing the incidence rate of cognitive impairment in patients with OSAHS.

OSAHS is a type of oxidative stress disease. The recurrent process of hypoxia and reoxygenation in patients results in the damage of various cellular structures of the human body, which is the cytological basis of various systemic diseases in patients with OSAHS [18]. The oxidative stress index of MDA content is able to directly reflect the degree of lipid peroxidation in the body and indirectly reflect the degree of cell injury. GSH-Px protects the integrity of cell membrane structure and function, which has a negative correlation with the severity of the disease. The function of SOD is to eliminate excessive oxygen free radicals and prevent tissues and organs from being damaged by free radicals [19]. Therefore, it was speculated that the abnormal oxidative stress level might be the molecular mechanism of cognitive impairment in patients with OSAHS. According to the study of Li et al. [6], MDA and SOD were representative indices of oxidative stress response of biological function for cognitive impairment in patients with OSAHS. OSAHS patients have a higher incidence rate of cognitive impairment, and oxidative stress may be one of the pathogeneses of OSAHS-related cognitive impairment [20]. In this study, there were significant differences in the levels of MDA, GSH-Px, and SOD among groups, which is consistent with previous literature [21] On this basis, the correlations between oxidative stress indices and cognitive impairment status were studied, and it was found that MoCA and MMSE scores of cognitive impairment were positively correlated with GSH-Px and SOD and negatively correlated with MDA. ROC curve analysis of cognitive impairment diagnosed by oxidative stress indices revealed that when the cut-off values of GSH-Px, MDA, and SOD were 251.07 U/mL, 9.46 nmol/mL, and 98.32 mmol/L, respectively, the AURCs were 0.670, 0.702, and 0.705, respectively, showing diagnostic values. When the three indices were adopted to evaluate the cognitive impairment of patients with OSAHS, it was found that each index had high sensitivity and specificity, indicating a certain predictive value. ROC curve analysis of the combination of the three indices showed that the maximum AURC was 0.836, suggesting that the combination of the oxidative stress parameters (GSH-Px, MDA, and SOD) can be used as a better tool to evaluate the severity of cognitive impairment in patients with OSAHS.

Conclusion

The incidence rate of cognitive impairment is high in patients with OSAHS, and there is oxidative stress in these patients. Oxidative stress is an early predictor and indicator of cognitive impairment in these patients, so it is of great significance to evaluate cognitive impairment and guide the treatment of OSAHS patients in clinical practice.

Conflict of interest

None of the authors report any conflict of interest.

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Received on July 27, 2021
Revised on September 17, 2021
Accepted on November 1, 2021
Online First November 2021