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Retraction

Retracted: Effect of Hyperbaric Oxygen Therapy on Sleep Quality, Drug Dosage, and Nerve Function in Patients with Sleep Disorders after Ischemic Cerebral Stroke

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This article has been retracted by Hindawi following an investigation undertaken by the publisher [1]. This investigation has uncovered evidence of one or more of the following indicators of systematic manipulation of the publication process:

(1) Discrepancies in scope

(2) Discrepancies in the description of the research reported

(3) Discrepancies between the availability of data and the research described

(4) Inappropriate citations

(5) Incoherent, meaningless and/or irrelevant content included in the article

(6) Manipulated or compromised peer review

The presence of these indicators undermines our confidence in the integrity of the article's content and we cannot, therefore, vouch for its reliability. Please note that this notice is intended solely to alert readers that the content of this article is unreliable. We have not investigated whether authors were aware of or involved in the systematic manipulation of the publication process.

Wiley and Hindawi regrets that the usual quality checks did not identify these issues before publication and have since put additional measures in place to safeguard research integrity.

We wish to credit our own Research Integrity and Research Publishing teams and anonymous and named external researchers and research integrity experts for contributing to this investigation.

The corresponding author, as the representative of all authors, has been given the opportunity to register their agreement or disagreement to this retraction. We have kept a record of any response received.

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[1] J. Wang, C. Wang, X. Wu, T. Ma, and X. Guo, "Effect of Hyperbaric Oxygen Therapy on Sleep Quality, Drug Dosage, and Nerve Function in Patients with Sleep Disorders after Ischemic Cerebral Stroke," *Emergency Medicine International*, vol. 2022, Article ID 8307865, 6 pages, 2022.

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Research Article

Effect of Hyperbaric Oxygen Therapy on Sleep Quality, Drug Dosage, and Nerve Function in Patients with Sleep Disorders after Ischemic Cerebral Stroke

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Objective. To explore the effects of hyperbaric oxygen therapy (HOT) on sleep quality, drug dosage, and nerve function in patients with sleep disorders after ischemic cerebral stroke (ICS). *Methods*. A total of 120 patients with acute ICS and sleep disorders who came to our hospital for treatment from January 2019 to October 2021 were selected and divided into control and observation groups according to the random numbering method, with 60 cases in each group. Both groups were treated with sertraline and eszopiclone for treating insomnia. The control group was given routine treatment for ICS, and the observation group was additionally treated with HOT in addition to the control group. The sleep quality, the use of sleep medication, the neurological function score, and the levels of serum tumor necrosis factor-α (TNF-α), endothelin (ET), and neuropeptide Y (NPY) before and after treatment were compared between the two groups. *Results.* The levels of TNF-α, ET and NPY were not significantly different between the two groups of patients before treatment ($P > 0.05$), and all of the above indicators decreased significantly in both groups after treatment, with the observation group being lower than the control group ($P < 0.05$). There was no significant difference in the sleep quality scores of PSQI, ESS, and SBQ between the two groups before treatment ($P > 0.05$), and the above indicators decreased significantly in both groups after treatment, with the observation group being lower than the control group ($P < 0.05$). There was no significant difference in the dose of sleep medication used in the first day of treatment between the two groups ($P > 0.05$), and the amount of sleep medication used in the observation group was significantly less than that in the control group after 14 d of treatment ($P < 0.05$). There was no significant difference in the NIHSS scores between the two groups before treatment ($P > 0.05$), and the scores of both groups decreased after treatment, and the scores of the observation group were significantly lower than those of the control group ($P < 0.05$). *Conclusion.* Compared with routine treatment, the addition of HOT to treat patients with sleep disorders after ICS can significantly improve their sleep quality, reduce dosage of sleep drugs, reduce inflammatory level of brain tissue and nerve function damage, and improve their prognosis. *Trial Registration.* This study was registered in the EA2019056

1. Preface

Ischemic stroke, also known as cerebral infarction, is a localized necrotic lesion of brain tissue caused by a narrowing or occlusion of the blood supplying arteries to the brain that triggers insufficient blood supply to the brain [1]. Ischemic stroke has a high morbidity and mortality rate and a poor prognosis, and it often occurs in middle-aged and elderly people [2]. In recent years, with the change of people's lifestyle, the number of cases has been increasing year by year, which seriously threatens the physical and mental health of patients. Ischemic stroke is caused by obstruction of cerebral blood flow and the pathophysiological changes of ischemia, hypoxia, and necrosis of the brain parenchyma, which in turn impairs cognitive and other neurological functions of the patient. The current treatment modalities

Table 1: Comparison of the general data of the two groups of patients.

Indicator		Control group ($n = 60$)	Observation group ($n = 60$)	Statistical value	P-value
Gender (male/female)	—	33/27	32/28	0.034	0.855
Age (years)	—	64.43 ± 8.64	64.41 ± 8.63	0.013	0.990
Co-morbidities	Hypertension	24	23	0.047	0.977
	Diabetes mellitus	19	20	—	—
	Hyperlipidemia	17	17	—	—
Lesion site	Thalamus	5	3	2.606	0.626
	Cerebellum	7	4	—	—
	Cerebral hemispheres	16	19	—	—
	Basal ganglia	18	15	—	—
	Brainstem	14	19	—	—
Education level	Illiterate	3	5	1.016	0.907
	Elementary school	14	12	—	—
	Junior high school	27	26	—	—
	High school	13	15	—	—
	University	3	2	—	—

for ischemic stroke mainly focus on thrombolysis, anticoagulation, and reduction of blood viscosity [3, 4]. Some ischemic stroke patients are often accompanied by varying degrees of anxiety and depression, which seriously affect the patient's sleep quality and is very unfavorable for the patient's treatment and recovery [5]. Dexzopiclone is a common sedative-hypnotic drug commonly used for various types of insomnia symptoms [6]. Sertraline is a commonly used antidepressant and anxiety drug in clinical practice, which can effectively relieve the bad psychological state of patients and contribute to the sleep quality of patients [7].

In addition to the above treatment, how to improve the patient's neurological impairment, restore the patient's motor function, and improve the patient's prognosis is also the focus of clinical attention. Hyperbaric oxygen therapy (HOT) is currently an effective way to promote the prognosis of patients with ischemic stroke, which can effectively increase the oxygen acquisition of brain tissue by inhaling high concentrations of oxygen, thereby promoting the recovery of injured neurological functions, improving blood circulation, enhancing brain metabolism, and contributing to the recovery of neurological functions [8, 9]. In this study, hyperbaric oxygen was used to treat patients with sleep disorders after ischemic stroke, and its effects on sleep quality, drug dosage, and neurological function were analyzed. The reports are as follows.

2. Materials and Methods

2.1. General Information. From January 2019 to October 2021, 120 patients with acute ischemic stroke sleep disorder who came to our hospital for treatment were selected as research subjects. According to the random numbering method, they were divided into the control group and observation group, with 60 cases in each group. There was no statistical difference in the data between the two groups ($P > 0.05$), Table 1.

Diagnostic criteria: (1) focal neurological deficits and, in a few cases, global neurological deficits; (2) unlimited duration or persistence of symptoms or signs for more than 24 h; (3) exclusion of vascular causes; (4) exclusion of cerebral hemorrhage by cranial CT or MRI.

Inclusion criteria: (1) The diagnostic criteria for ischemic stroke were met [10], and confirmed by head CT or MRI; (2) Had not taken similar drugs or drugs antagonistic to this drug recently; (3) All patients were first-time patients stroke patients; (4) informed and voluntary participation. Exclusions: (1) Patients with malignant tumors; (2) Mental disorders, unable to communicate normally; (3) Poor compliance. (4) Complicated with organic diseases such as heart, liver, and kidney.

2.2. Methods

2.2.1. The Control Group Was Given Conventional Treatment. The patient was hospitalized immediately after diagnosis, and given normal pressure oxygen inhalation, intracranial pressure reduction, antiplatelet drugs, thrombolysis, neurotrophic solution, symptomatic and supportive treatment, etc. Aspirin enteric-coated tablets (Shenyang Aojina Pharmaceutical Co., Ltd., approved by H20065051, 100 mg/time, once/d) and nimodipine (Hubei Sihuan Pharmaceutical Co., Ltd., approved by H20030026, 60 mg/time, 2 times/d) were given for the treatment of ischemic stroke. Critically ill patients were given drugs to prevent infection, antiarrhythmia and maintain water-electrolyte balance, sedation and antiepilepsy according to the patient's symptoms, as well as appropriate rehabilitation instructions for two consecutive weeks.

2.2.2. The Observation Group Received Hyperbaric Oxygen Therapy on the Basis of the Control Group. The patients in the observation group started to receive hyperbaric oxygen therapy after the vital signs stabilized on the 3rd day of admission. A single hyperbaric chamber was used, with partial pressure of oxygen at 2.0 mPa, pressurized to 0.2 Mpa, and stabilized for 20 min. The chamber was washed at high flow rate for 10 min at the high pressure plane, and then gradually decompressed and discharged, 60 min/time, 1

time/d, for two weeks, and early rehabilitation training was also instructed.

2.2.3. Treatment Options for Sleep Disorder Medications. The two groups of patients were given the same sleep drug sertraline (Pfizer Pharmaceutical Co., Ltd., approved by the State Drug H10980141, 50 mg*30 tablets) and dexrazopiclone (Jiangsu Tasly Diyi Pharmaceutical Co., Ltd., approved by the State Drug H20070069, 3 mg*6 tablets) for the treatment of sleep disorders. Dexrazopiclone 3 mg/d, 1 time/d and sertraline 50 mg/d, 1 time/d were started and the dose was adjusted according to the patient's sleep status.

2.2.4. Observation Indicators

(1) To compare the sleep quality of the two groups of patients before and after treatment. All patients were evaluated by specialized medical staff within 24 hours of admission and were collected by a sleep disorder scale. The Pittsburgh Sleep Quality Index Inventory (PSQI) was used to assess sleep quality, consisting of 7 factors (sleep quality, time to fall asleep, sleep duration, sleep efficiency, sleep disorder, hypnotic medication, and daytime function), with a total score range of 0–21. A total PSQI score ≥7 can be used as a criterion for diagnosing sleep disorders, and a higher score indicates poorer sleep quality. Epworth (ESS) was used to evaluate the degree of daytime sleepiness, and the ESS scale consisted of 8 items, each item was scored 0~3 (never napping was scored as 0, rarely napping was scored as 1, sometimes napping was scored as 2, and often napping was scored as 3), and the total score was 24, ESS>10 was assessed as daytime sleepiness, the higher the score, the more serious the patient's sleepiness. The STOP-Bang questionnaire (SBQ) was used to assess sleep apnea, which included whether snoring was loud, whether daytime fatigue was present, whether sleep apnea was observed, whether hypertension was present, and gender, age, BMI, and neck circumference. The presence of sleep apnea was determined by an SBQ score ≥3.

(2) To compare the dosage of sleep drug sertraline before and after treatment in the two groups of patients.

(3) The serum levels of tumor necrosis factor-α (TNF-α), endothelin (ET), and neuropeptide Y (NPY) were compared between the two groups of patients before and after treatment. 8 mL of fasting venous blood was collected from patients, centrifuged at 3500 r/min for 10 min at 5°C, and the upper layer of plasma was stored in a refrigerator at −40°C and assayed by radioimmunoassay technique.

(4) Comparison of the changes of neurological function between the two groups before and after treatment. NIHSS was used to evaluate the neurological function of the patients. The scores included the level of consciousness, language, articulation, movement of limbs, sensation, and facial paralysis.

2.2.5. Statistical Analysis. Statistical software SPSS 22.0 was used to analyze the data. The count data was expressed as rate, and the χ^2 test was used. The measurement data was expressed as $(x \pm s)$, and the t-test was used. $P < 0.05$ was considered statistically significant.

3. Results

3.1. Comparison of the General Data of the Two Groups of Patients. The general data of the patients in the two groups were compared, and the results showed that the differences between the two groups were not significant $P > 0.05$ in the data of gender, age, co-morbidities, lesion sites, and education level, as shown in Table 1.

3.2. Comparison of the Contents of TNF-α, ET, and NPY in the Two Groups of Patients. There was no significant difference in the contents of TNF-α, ET, and NPY between the two groups before treatment ($P > 0.05$). The levels of TNF-α, ET, and NPY decreased in both groups after treatment, with the above indexes in the observation group being significantly lower than those in the control group ($P < 0.05$), as shown in Table 2.

3.3. Comparison of sleep quality between the two groups of patients. There was no significant difference in the scores of PSQI, ESS, and SBQ between the two groups before treatment ($P > 0.05$). The scores of PSQI, ESS, and SBQ decreased in both groups after treatment, with the scores of the above-mentioned scales in the observation group being significantly lower than those in the control group ($P < 0.05$), as shown in Table 3.

3.4. Comparison of the Dosage of Sleep Medication before and after Treatment between the Two Groups of Patients. There was no significant difference in the dosage of sleep medication between the two groups on the first day of treatment ($P > 0.05$). After 14 days of treatment, the dosage of sleep medication in the observation group was significantly lower than that in the control group ($P < 0.05$), as shown in Table 4.

3.5. Comparison of the NIHSS Scores of the Two Groups of Patients. There was no significant difference in the NIHSS scores between the two groups before treatment ($P > 0.05$), the scores of both groups decreased after treatment, and the scores of the observation group were significantly lower than those of the control group, and the differences between the two groups were statistically different ($P < 0.05$), as shown in Table 5.

Table 2: Comparison of the levels of TNF-α, ET, and NPY in the two groups of patients ($\bar{x} \pm s$, pg/mL).

Group	Number of cases	TNF-α		ET		NPY	
		Before treatment	After treatment	Before treatment	After treatment	Before treatment	After treatment
Observation group	60	87.61 ± 5.54^a	34.41 ± 4.76^a	228.31 ± 9.56	154.26 ± 8.11^a	181.48 ± 7.53	97.49 ± 6.86^a
Control group	60	87.58 ± 5.49	59.77 ± 4.83^a	228.29 ± 9.61	198.65 ± 8.23^a	180.52 ± 7.49	126.17 ± 7.73^a
t	—	0.030	28.967	0.011	29.759	0.700	21.495
P	—	0.976	0.000	0.991	0.000	0.485	0.000

Note. $aP < 0.05$ compared with before treatment.

Table 3: Comparison of sleep quality between the two groups of patients ($\bar{x} \pm s$, points).

Group	Number of cases	PSQI		ESS		SBQ	
		Before treatment	After treatment	Before treatment	After treatment	Before treatment	After treatment
Observation group	60	7.44 ± 3.92	5.23 ± 3.02^a	7.62 ± 4.32	5.19 ± 4.11^a	3.26 ± 1.69	2.18 ± 0.67^a
Control group	60	7.51 ± 4.01	6.41 ± 3.11	7.57 ± 4.29	6.71 ± 4.15	3.24 ± 1.67	2.98 ± 1.02
t		0.097	2.108	0.064	2.016	0.065	5.078
P		0.923	0.037	0.949	0.046	0.948	0.000

Note. $aP < 0.05$ compared with before treatment.

Table 4: Comparison of sleep medication dosage changes before and after treatment in the two groups of patients ($\bar{x} \pm s$, mg).

Group	Number of cases	1st day of treatment	14 days of treatment
Observation group	60	53.00 ± 1.37	28.00 ± 0.56^a
Control group	60	53.00 ± 1.21	37.00 ± 0.64^a
t	—	0.000	81.976
P	—	1.000	0.000

Note. $aP < 0.05$ compared with before treatment.

4. Discussions

Ischemic stroke is a common clinical cardiovascular and cerebrovascular disease, and patients are often accompanied by varying degrees of sleep disorders, which affect the recovery process of patients and are not conducive to the improvement of prognosis [11]. Patients have various forms of sleep disorders, including difficulty in falling asleep, insomnia, and sleep apnea. Long-term sleep disorders can lead to poor mental concentration, daytime sleepiness, anxiety, and depression [12]. At present, sleep disorders are generally treated with drugs, but long-term use of drug therapy can produce toxic side effects on the body [13], HOT is a noninvasive treatment method and is widely used in neurology [14]. In this study, HOT was used to treat ischemic stroke patients with sleep disorders, in order to improve the sleep quality of patients, reduce the dosage of sleep drugs, and improve the neurological function of patients.

In this study, it was found that the levels of TNF-α, ET, and NPY were not significantly different between the two groups before treatment, and the levels of these indicators decreased in both groups after treatment, and the observation group was significantly lower than the control group. TNF-α is a tumor necrosis factor with strong biological activity, which can activate leukocytes and promote the release of various inflammatory mediators; ET has a vasoconstrictive function and can be released in large quantities in a state of ischemia and hypoxia, and is an endogenous injury-causing factor. NPY is a neuropeptide that is widely distributed in the central and peripheral nervous systems and can stimulate the occurrence of inflammatory responses [15]. The reduction of inflammatory factors such as TNF-α, ET, and NPY after treatment suggests that hyperbaric oxygen therapy contributes to the clearance of serum inflammatory factors in ischemic stroke, reduces the inflammatory response, and improves cerebral blood circulation. HOT can improve patients' cerebral hypoxia, increase the partial pressure of blood oxygen in cerebral vessels, promote cerebral blood circulation, improve cerebral metabolism, reduce

Table 5: Comparison of NIHSS scores between the two groups ($\bar{x} \pm s$, points).

Group	Number of cases	Before treatment	After treatment
Observation group	60	7.21 ± 4.35	4.78 ± 2.45^a
Control group	60	7.18 ± 4.27	5.89 ± 3.21^a
t	—	0.038	2.129
P	—	0.970	0.035

Note. $aP < 0.05$ compared with before treatment.

intracranial pressure, alleviate cerebral edema, accelerate the rate of removal of oxygen free radicals and inflammatory factors in the body, and reduce their secondary damage to the brain tissue [16]. It was found that there was no significant difference in the scores of PSQI, ESS, and SBQ between the two groups before treatment, and the scores of the above scales decreased in both groups after treatment, with the observation group significantly lower than the control group, and the use of sleep medication in the observation group was significantly lower than the control group after treatment. PSQI, ESS, and SBQ are common clinical scales used to assess patients' sleep quality, and the results of the study showed that adding hyperbaric oxygen therapy to conventional treatment can improve patients' sleep quality and reduce dependence on sleep medication.

The pathogenesis of sleep disorders in patients with ischemic stroke is complex. In recent years, there have been many studies [17, 18] on the pathogenesis of sleep disturbance after ischemic stroke at home and abroad, and it is believed that the neurons in the brain may be damaged, the brain edema compresses the surrounding brain tissue, and the brain areas involved in sleep regulation are severely damaged, blocking the conduction of the specific reticular upward activation system, thus triggering the disruption of sleep structure. HOT can improve the state of brain hypoxia, increase the blood oxygen content of brain tissue, restore blood supply balance, improve the state of intracranial hypertension, accelerate the scavenging of free radicals, reduce secondary damage to brain tissue, eliminate the vicious cycle of brain tissue hypoxia-edema-hypoxia, can promote the reconstruction of deficient neurological function, and improve the quality of sleep [7, 19]. At the same time, hyperbaric oxygen therapy reduces the inflammatory response of the blood vessels in the brain, decreases the interference of neuropathic factors in the sleep of patients, improves the quality of their sleep, reduces dependence on drugs, and decreases the use of drugs [20]. NHISS scores decreased in both groups before and after treatment compared with those before treatment, and were lower in the observation group. The possible reason for the analysis is that insufficient energy metabolism of brain cells due to severe ischemia and hypoxia is the main cause of triggering neurological function damage, while HOT can rapidly increase the oxygen content of blood, improve hemodynamics, promote oxygen metabolism of brain cells, accelerate the regeneration and repair of capillaries, help the recovery of the internal environment of damaged neurons, and reduce the damage to patients' neurological function [21].

In conclusion, the treatment of patients with sleep disorders after ischemic stroke with HOT on the basis of conventional treatment has good efficacy, can significantly improve the sleep quality of patients, reduce the use of sleep medication, high safety, reduce the inflammatory level of brain tissue, improve the neurological impairment of patients, and is worthy of clinical promotion. The clinical registration number is EA2019056.

Data Availability

The data can be obtained from the author upon reasonable request.

Ethical Approval

This study was approved by the ethics committee of our hospital. The number is L2019096.

Conflicts of Interest

The authors declare that the research was conducted in the absence of any commercial or financial relationships that could be construed as a potential conflict of interest.

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