Heliyon 8 (2022) e12351

Contents lists available at ScienceDirect

Heliyon

journal homepage: www.cell.com/heliyon

Research article

Prognostic factors of hyperbaric oxygen therapy for patients with delayed encephalopathy after acute carbon monoxide poisoning

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ARTICLE INFO

Keywords:
Prognostic factors
Delayed encephalopathy after acute carbon monoxide poisoning
Hyperbaric oxygen therapy

ABSTRACT

Delayed encephalopathy after acute carbon monoxide poisoning (DEACMP) is a disease with an incomplete pathological mechanism, long treatment time, and uncertain factors affecting the therapeutic effect. This study explored prognostic factors for DEACMP patients treated with hyperbaric oxygen therapy (HBOT) in 15 hospitals in China. The findings might provide a theoretical basis for further improving the prognosis of DEACMP patients.

In this study, data from 330 patients with DEACMP who were admitted to HBOT centers of 15 hospitals in Hunan Province (China) from June 2015 to June 2020 were retrospectively analyzed, and their medical records related to disease prognosis were collected and followed up by telephone. Univariate and multivariate analyses were used to identify independent risk factors for the prognosis of DEACMP patients after HBOT. Univariate analysis revealed 11 possible prognostic factors. Consistent with univariate analysis, multivariate analysis found that underlying diseases (Odds radio(OR) = 2.886, P = 0.048), hypermyotonia (OR = 5.2558, P = 0.008), and HBOT pressure no less Than 2.3 atm absolute (ATA) ((OR = 7.812, P = 0.004) were identified as independent prognostic factors among 20 variables for poor prognosis of DEACMP patients treated with HBOT in the study. This multicenter retrospective analysis revealed that the adverse prognostic markers for DEACMP patients treated with HBOT might be underlying diseases, hypermyotonia, and an HBOT pressure of 2.3 ATA or higher.

1. Introduction

Delayed encephalopathy after acute carbon monoxide (CO) poisoning (DEACMP) is the most common and severe complication of CO poisoning. Unfortunately, about 20–40% of patients with CO poisoning will eventually develop DEACMP [1]. DEACMP patients suffer a series of neurological and psychiatric disorders, including cognitive dysfunction, motor impairment, extrapyramidal systems dysfunction, and even consciousness disturbance after a brief lucid interval. The pathogenesis of DEACMP is widely discussed, such as the ischemic-hypoxic damage, inflammation and immune cytokine stimulation, cell apoptosis, and neurotoxicity induced by CO poisoning [2], but no clear pathogenesis has

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https://doi.org/10.1016/j.heliyon.2022.e12351
Received 15 August 2022; Received in revised form 22 October 2022; Accepted 7 December 2022

been established. Due to the poorly studied pathogenesis, there is no precise causal treatment modality, and the current clinical treatment is mainly symptomatic.

Hyperbaric oxygen therapy (HBOT) is a therapeutic modality of breathing nearly 100% oxygen in a hyperbaric chamber with a pressure higher than sea level. Due to its definitive therapeutic effects in alleviating ischemic-hypoxic injury [3], reducing the inflammatory response [4], and decreasing cell apoptosis [5], HBOT is at the core of a comprehensive treatment strategy for DEACMP. Several studies have shown that HBOT can effectively relieve the symptoms of DEACMP patients [6] and reduce the white matter lesions of the brain [7]. However, due to the unclear pathogenesis, the long duration, and the high residual rate of symptoms, some patients still cannot obtain satisfactory therapeutic effects after a long period of HBOT-centered comprehensive treatment [8]. Based on the current status of DEACMP research, there is an urgent need to analyze prognostic influencing factors to accurately predict the prognosis of patients with HBOT and improve the treatment outcome as much as possible. In the existing reports, factors associated with the prognosis of DEACMP include age, comorbidities, the lucid interval [6], intracranial lesions revealed by MRI [9], and other combined treatment modalities such as dexamethasone, butalbital, and mesenchymal stem cell transplantation [7]. However, the existing studies have small sample sizes and lack data from multicenter studies. Therefore, we conducted a multicenter retrospective study from January 2021 to April 2021, analyzing the adverse prognosis factors and intending to explore the possible prognostic markers of HBOT in patients with DEACMP.

2. Methods

2.1. Patient source

This study obtained approval from the ethics committee of Xiangya Hospital, Central South University. Clinical and imaging data were retrieved from patients newly diagnosed with DEACMP between June 2015 and June 2020 in the hyperbaric oxygen departments of 15 hospitals in 13 cities. Patient data were collected from January 2021 to April 2021. The actual end date for all data collection work in this study was April 13, 2021.

2.2. Patient inclusion and exclusion

The study team completed the inclusion and exclusion criteria in December 2020, as shown in Table 1.

2.3. Data collection

After reviewing a large amount of literature related to the prognostic factors of DEACMP, researchers began to collect data. This study collected demographic information such as name, ID number, age, gender, and date of the first visit for patients with DEACMP to hyperbaric oxygen departments. The name and ID number were used to identify the same patient at different medical centers. Medical data during acute CO poisoning were as follows: underlying diseases, duration of CO exposure, duration of coma, duration of the lucid interval, and degree of acute CO poisoning. Medical history data during the period of delayed encephalopathy included impairment of consciousness, cognitive dysfunction, incontinence, muscle strength damage, hypermyotonia, limb tremor, site of intracranial injury, and companion diseases. The treatment data were also retrieved, including HBOT pressure, the interval between the first HBOT and the time of CO poisoning, HBOT sessions, combined drugs (dexamethasone, butalbital, edaravone), and rehabilitation therapy. The necessary descriptions of variables were presented in Supplementary Table S1. The data of all cases were handed over to Xiangya Hospital for a unified summary and excluding duplicate cases. Only data collectors and follow-up personnel could access all clinical and image data, preventing patient data leakage.

Table 1. Inclusion and exclusion criteria of this study.

Inclusion criteria	Exclusion criteria
① A clear history of exposure to CO;	① Less than 10 HBOT sessions;
② An obvious lucid interval;	② Died of other diseases at the time of the follow-up;
③ Appearing symptoms of neurological deficits, such as cognitive dysfunction, motor impairment, and extrapyramidal systems symptoms after the lucid interval;	③ Complications affecting neurological function (such as cerebral hemorrhage, cerebral infarction) before or during the treatment of DEACMP;
④ Total duration of illness no less than 6 months;	④ Other interference factors of diagnosis or judgment.
⑤ Accepting follow-up;	⑤ More than two variables missing

2.4. Telephone follow-up

From January 2021 to April 2021, after obtaining verbal consent from patients or guardians, the enrolled patients were followed up by telephone in this study. The follow-up will include current residual symptoms and findings of reviewed head MRI. The efficacy of the patients will be evaluated based on the results of the telephone follow-up.

2.5. Prognostic evaluation criteria

In this study, two doctors in the hyperbaric oxygen department who were proficient in DEACMP were responsible for prognostic evaluation. The treatment efficacy of patients was classified into four major categories according to their residual symptoms, MRI findings, and daily living ability.

(1) Cured: clinical symptoms and signs all disappeared, daily living ability and intelligence returned to normal, and imaging examination results were all free of abnormalities.
(2) Obvious effective: symptoms and signs basically disappeared, and only some secondary symptoms that do not affect the basic daily life remained, such as slow reaction, mild memory loss, personality changes, and daily life was rarely dependent on others, MRI imaging might appear as residual lesions.
(3) Effective: symptoms and signs were partially reduced compared with baseline, daily life relied on others to some extent, and residual lesions appeared on MRI imaging.
(4) Invalid: symptoms and signs were not significantly relieved, and the lesion shrunk less than 20% on MRI imaging, or the patient's condition worsened.

The cured, obvious effective, and effective patients had a better quality of life and a light burden on the family and society. However, patients with no noticeable improvement or aggravated disease after treatment (invalid patients) were the difficulties for clinical work. The invalid patients had different degrees of resistance to HBOT and drug therapy; the clinical outcome was poor, and the quality of life was low. Therefore, cured, obvious effective, and effective patients were judged as patients with good prognoses, and invalid patients were judged as patients with poor prognoses in this study.

2.6. Statistical methods

All data in this study were analyzed using SPSS software (Version 22; SPSS, Inc., Chicago, IL). Continues data was presented as Median and range for non-normally distributed subjects. Missing values were filled using the median (not normally distributed data), or mode (categorical data). In the univariate analysis, because none of the continuous variables in this study met homogeneity of variance and normality, the Wilcoxon rank-sum test was used for continuous and rank variables. The

chi-square test was used for dichotomous variables. For multi-factor analysis, the binary logistic regression test was used in this study, and continuous variables with outliers were transformed into rank variables. The test level was set at $\alpha = 0.05$.

3. Results

3.1. Patient's characteristic

The data collection for this study took four months, and a total of 392 cases were included in 15 hospitals. Sixty-two cases were excluded, of which 24 had more than two missing variables, 8 were duplicated cases between different hospitals, 9 patients had less than 10 HBOT sessions, 17 patients had diseases affecting neurological function before or during treatment (16 cerebral infarction and 1 cerebral hemorrhage), 2 patients had a history of mental illness, 1 patient had congenital deafness, and 1 patient had another toxic gas poisoning. A total of 330 cases were enrolled (The distribution of 330 cases from 15 hospitals is presented in Supplementary Table S2). After independent evaluation by at least two clinicians, there were 301 patients with good prognoses (71 cured, 131 obvious effective, 99 effective) and 29 patients with poor prognoses (2 of them had passed away at the time of follow-up), and the total effective rate of HBOT was 91.21%. The enrollment of cases in this study is shown in Figure 1.

The male-female ratio was 0.88, and the median age was 55 (range 8–85) years of all 330 patients (Table 2). About half of the patients (50.61%) presented severe CO toxicity in the acute phase and underwent a median lucid interval of 13.5 (range 0.5–180) days before developing DEACMP. The primary underlying diseases were hypertension, diabetes mellitus, and coronary heart disease in 330 cases. During a long-term treatment centered on HBOT (median 40, range 10–450 sessions), 75 patients (22.73%) developed companion diseases. The main companion diseases were respiratory infections (7.27%), myocardial damage (1.52%), electrolyte disturbances (1.21%), and upper gastrointestinal bleeding (1.21%). Thankfully, all of the above companion diseases were transient damage and fully recovered. A treatment strategy centered on HBOT was safe for DEACMP patients.

3.2. Univariate analysis of prognosis factors for DEACMP

In this study, univariate analysis was first used to analyze the disease factors and treatment factors affecting the prognosis of DEACMP. The selection principle of variables was based on previous literature reports, clinical experience, the risk factors and clinical symptoms of DEACMP, and the aim to explore the benefit possibility of combining HBOT with drug or rehabilitation therapy. This study analyzed age, underlying diseases, duration of CO exposure, duration of coma during acute CO poisoning, the lucid interval, degree of CO poisoning, symptoms during DEACMP period (including impaired consciousness, incontinence, muscle strength damage, hypermyotonia, and limb tremor), site of intracranial injury, the interval between the first HBOT session and acute CO poisoning, the number of HBOT sessions after the occurrence of DEACMP, HBOT pressure, companion diseases during HBOT, combination medication (dexamethasone, butalbital, edaravone), and rehabilitation treatment. A total of 20 possible influencing factors were identified. Among them, age, underlying diseases, duration of CO exposure, duration of coma during acute CO poisoning, impairment of consciousness during DEACMP, incontinence, muscle strength damage, hypermyotonia, site of intracranial injury, HBOT pressure, and companion diseases during HBOT were factors affecting the prognosis of the DEACMP patients (Table 3).

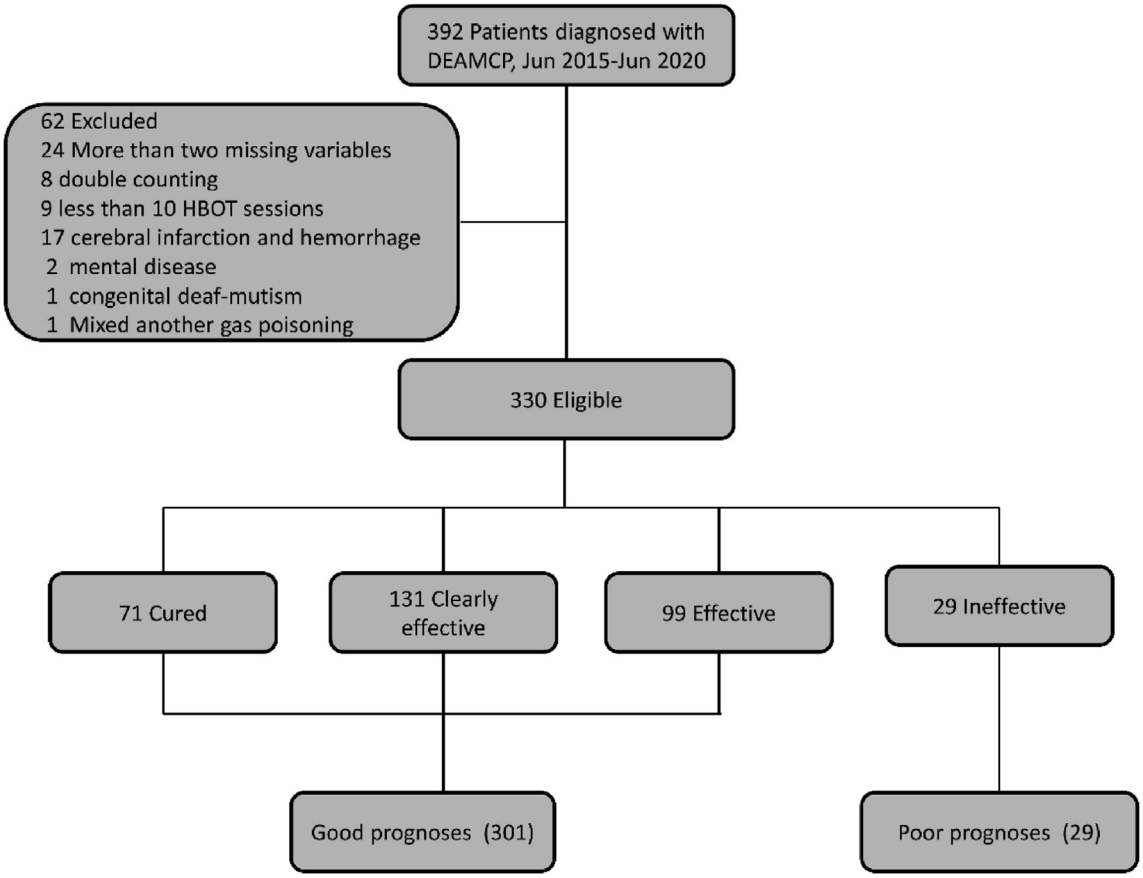

Figure 1. Data flow diagram.

Table 2. Characteristics of the enrolled patients.

Characteristics		Cases/ Median	Percentage (%)/Range
Gender	Male	156	47.27
	Female	174	52.73
Age (years)		55	8–85
Degree of CO poisoning	Mild	48	14.55
	Moderate	115	34.85
	Severe	167	50.60
Lucid interval (day)		13.5	0.5–180
HBOT sessions		40	10–450
Underlying diseases	None	219	66.36
	Hypertension disease	27	8.18
	Diabetes mellitus	10	3.03
	Coronary heart disease	10	3.03
	No (mild) symptomatic central nervous system/mental illness	7	2.12
	Osteoarticular diseases	7	2.12
	Bronchial disease	5	1.52
	Liver disease	5	1.52
	Other	15	4.55
	2 or more underlying diseases	25	7.58
Companion diseases	None	255	77.27
	Respiratory infection	24	7.27
	Myocardial damage	5	1.52
	Electrolyte disorder	4	1.21
	Upper gastrointestinal bleeding	4	1.21
	Others	27	8.18
	2 or more companion diseases	11	3.33

Categorical data were presented as the number of cases and percentage, and non-normally distributed count data were described by median and range.

3.3. Multivariate analysis of prognostic influences for DEACMP

In this study, 11 significant prognostic factors derived from univariate analysis as well as other 9 non-significant factors were further analyzed by binary logistic regression to obtain independent prognostic factors of poor prognosis for the DEACMP patients. Before multivariate analysis, the study transformed the continuous variables with outliers into stratified variables. By adjusting the stratified classification, we arrived at a stable and reliable way of continuously grouping variables (Supplementary Table S3). Multivariate analysis identified three independent adverse prognostic factors for HBOT of 330 DEACMP patients: underlying diseases (Odds radio (OR) = 2.886, P = 0.048), hypermyotonia (OR = 5.255, P = 0.008), and HBOT pressure exceeding 2.3 ATA (OR = 7.812, P = 0.004) (Table 4). DEACMP Patients with underlying diseases, clinical symptoms of hypermyotonia, and using HBOT with a pressure exceeding 2.3 ATA were more likely to get a poor prognosis.

4. Discussion

The pathological changes of DEACMP mainly include mitochondrial dysfunction, oxidative stress, lipid peroxidation, and apoptosis [2, 10]. By opening mitochondrial ATP-sensitive potassium channels [11], increasing antioxidant defense mechanisms [12], and inducing protective autophagy of nerve cells [13], HBOT reduces nerve cell death and promotes the functional recovery of nerve cells. Therefore, it is beneficial for DEACMP patients to improve their symptoms.

In previous reports on the prognosis of DEACMP, the number of patients included in a single report often fluctuated between 20 and 90 cases. There were many possible influencing factors, but the number of cases admitted to each hospital in a short time was relatively small. This contradiction was an important limitation for multivariate analysis of prognostic factors of DEACMP. This study collected more than 300 samples from 15 hospitals to ensure the reliability of the results of multivariate regression analysis. In this multicenter retrospective study, HBOT had high overall efficiency, manageable companion diseases, and no serious adverse events that were not tolerated by patients, making it a safe and effective treatment for DEACMP. Three adverse prognostic markers for HBOT of DEACMP derived from this study: underlying diseases, hypermyotonia, and HBOT pressure no less than 2.3ATA.

According to this study, HBOT pressure of no less than 2.3ATA could not benefit patients to obtain good clinical outcomes. One of the biggest concerns for HBOT in treating diseases of the central nervous system is whether high oxygen level causes excessive oxidative stress, resulting in reinjury of the cells. Current studies show that long-term HBOT could improve cell mitochondrial function, increase antioxidant defense mechanisms that combat free radicals brought by high oxygen, and ultimately protect cell survival and function [12]. However, some studies show that high-pressure HBOT (2.8ATA-3ATA) over 1–3 weeks inhibit free-radical scavenger functions [14] and cause apoptosis of mouse retinal nerve cells [15]. Therefore, the increased oxidative stress and the obstruction of nerve cell damage repair associated with the long-term HBOT above 2.3ATA may be possible reasons that affect the prognosis of DEACMP.

The relationship between underlying diseases and DEACMP is complicated. Underlying diseases (such as thyroid function [16]) are not only closely related to the occurrence of DEACMP but also affect the progress and therapeutic efficacy of DEACMP. For example, hypertension disease adversely affects the regression of white matter lesions and hinders the recovery of patients' cognitive function [17]. Patients with persistent hyperglycemia could produce excessive reactive oxygen species, causing DEACMP patients to suffer severe oxygen-free radical damage [14]. Hence, actively controlling underlying diseases effectively improves the therapeutic efficacy and prognosis of patients with DEACMP.

Hypermyotonia is a clinical symptom that predicts an adverse prognosis of DEACMP in this study. The pathogenesis of hypermyotonia is complex, and injuries in multiple parts, including the basal ganglia, could lead to hypermyotonia [18]. Meanwhile, hypermyotonia can take various forms, including spasticity, dystonia, and rigidity [19]. In the follow-up study, we will further verify the influence of different forms of hypermyotonia on the prognosis of DEACMP.

In the previous study reported by Huijun Hu in 2011 [6], factors associated with poor prognosis were age, companion diseases, and the lucid interval. Still, underlying diseases could not affect the prognosis significantly. Those were not consistent with the results of this experiment. The possible reasons are as follows: 1. The causes of CO poisoning are different. This study's leading cause of CO poisoning was charcoal heating at night. Patients in the previous study came from northern China, where central heating was available and charcoal were rarely needed. 2. The health status of the population changes. The disease spectrum of the Chinese population has changed in the past 11 years, which affects the composition of underlying diseases of DEACMP patients. 3. Experimental bias. There were 10 variables but a small sample size (46 cases) in Hu's report, which caused inherent bias in the study to a certain extent.

There are still some deficiencies in this study. Due to the limitations of previous clinical work in each center, more objective indicators, such as scale results and scoring standards, were not used to evaluate efficacy in this study. In subsequent studies, we will set up more rigorous prospective experiments to verify the experimental results of this study.

5. Conclusion

The results prove that underlying diseases, hypermyotonia, and HBOT pressure no less than 2.3ATA are independent adverse prognostic factors of DEACMP patients in this study with HBOT. According to the results, active control of underlying diseases, relaxation of hypermyotonia, and a low-pressure HBOT regimen may further improve the prognosis of DEACMP patients. It is worth affirmatory that, through a large sample and multicenter study, the adverse prognostic markers of

Table 3. Univariate analysis of adverse prognosis factors for DEACMP.

Factors		Poor prognosis group	Good prognosis group	OR/W value	P value
Underlying diseases*	Yes	18(5.45%)	93(28.18%)	3.660	0.001
	No	11(3.33%)	208(63.03%)		
Incontinence*	Yes	21(6.36%)	132(40.00%)	3.361	0.030
	No	8(2.42%)	169(51.21%)		
Muscle strength damage*	Yes	15(4.55%)	78(23.64%)	2.732	0.004
	No	14(4.24%)	223(67.58%)		
Hypermyotonia*	Yes	23(6.97%)	114(34.55%)	6.288	<0.001
	No	6(1.82%)	187(56.67%)		
Limb tremor*	Yes	8(2.42%)	60(18.18%)	1.530	0.226
	No	21(6.36%)	241(73.03%)		
Companion diseases*	Yes	13(3.94%)	62(18.79%)	3.132	0.005
	No	16(4.85%)	239(72.42%)		
Dexamethasone*	Yes	6(1.82%)	90(27.27%)	0.612	0.206
	No	23(6.97%)	211(63.94%)		
Butalbital*	Yes	7(2.12%)	75(22.73%)	0.959	0.565
	No	22(6.67%)	226(68.48%)		
Edaravone*	Yes	11(3.33%)	125(37.88%)	0.861	0.433
	No	18(5.45%)	176(53.33%)		
Rehabilitation therapy*	Yes	11(3.33%)	117(35.45%)	0.961	0.545
	No	18(5.45%)	184(55.76%)		
Degree of acute CO poisoning#	mild	1(0.30%)	47(14.24%)	49507.5	0.490
	moderate	13(3.94%)	102(30.91%)		
	severe	15(4.55%)	152(46.06%)		
Impairment of consciousness during DEACMP#	No	8(2.42%)	176(53.33%)	48293.5	0.001
	Clouding of consciousness	12(3.64%)	88(26.67%)		
	Coma	9(2.73%)	37(11.21%)		
Site of intracranial injury#	None	1(0.30%)	61(18.48%)	48765.5	0.026
	Basal ganglia region	9(2.73%)	112(33.94%)		
	Other site	11(3.33%)	56(16.97%)		
	Both	8(2.42%)	72(21.82%)		
HBOT pressure*	≥2.3ATA	9(2.72%))	18(5.45%)	7.075	<0.001
	<2.3ATA	20(6.06%)	283(85.76%)		
Age#		64(44–80)	55(8–85)	3095.5	0.010
Duration of CO Exposure#		10(2–24)	8(0.3–72)	48363.0	0.003
Duration of coma during acute CO poisoning#		10(1–96)	6(0–4320)	48603.5	0.013
Lucid interval#		16(3–34)	13(0.5–180)	49132.0	0.163
Interval between the first HBOT and CO poisoning#		8(2–648)	8(1–1440)	49627.0	0.700
HBOT sessions#		35(10–200)	42(10–450)	4120.5	0.166

* Chi-square test, OR = "Yes"/"No" or "≥2.3ATA"/"<2.3ATA".
# Wilcoxon rank-sum test; Categorical data were presented as N(%), and non-normally distributed count data were described as median(range).

Table 4. Multivariate analysis of adverse prognostic influences for DEACMP.

	Estimate	Std. Error	Wald	P	OR	95% Confidence Interval	
						Lower Bound	Upper Bound
Underlying diseases *(Yes vs. No)*	1.06	0.535	3.926	0.048	2.886	1.011	8.236
Hypermyotonia *(Yes vs. No)*	1.659	0.627	7.007	0.008	5.255	1.538	17.954
HBOT pressure ≥2.3ATA *vs.* <2.3ATA	2.056	0.715	8.257	0.004	7.812	1.922	31.746

HBOT for DEACMP will provide significant guidance for further clinical work and research.

Declarations

Author contribution statement

Fangling Huang: Conceived and designed the experiments; Performed the experiments; Analyzed and interpreted the data; Wrote the paper.

Lin Yang, Zhen Tan, Bangmin Yang, Pingqiong Liu, Zuomin Li, Wanli Shi, Kun Peng, Jing Yuan, Qing He, Li Yang, Xinyu Li, Can Li, Dezhong Chen: Performed the experiments; Contributed reagents, materials, analysis tools or data.

Zhengrong Peng: Conceived and designed the experiments; Analyzed and interpreted the data; Contributed reagents, materials, analysis tools or data.

Funding statement

Dr. Fangling Huang and Zhengrong Peng were supported by Natural Science Foundation of Hunan Province [2020JJ5902& 2021JJ31089].

Data availability statement

Data will be made available on request.

Declaration of interest's statement

The authors declare no competing interests.

Additional information

Supplementary content related to this article has been published online at https://doi.org/10.1016/j.heliyon.2022.e12351.

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