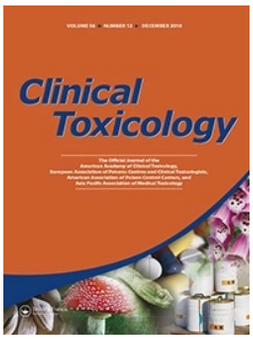

Clinical Toxicology

ISSN: (Print) (Online) Journal homepage: https://www.tandfonline.com/loi/ictx20

Dexamethasone therapy prevents delayed neuropsychiatric sequelae after carbon monoxide poisoning: a prospective registry-based study

Sechan Kim, Sungwoo Choi, Yujin Ko, Choung Ah Lee, Gi Woon Kim, Ji Eun Moon, Sangun Nah & Sangsoo Han

To cite this article: Sechan Kim, Sungwoo Choi, Yujin Ko, Choung Ah Lee, Gi Woon Kim, Ji Eun Moon, Sangun Nah & Sangsoo Han (2023): Dexamethasone therapy prevents delayed neuropsychiatric sequelae after carbon monoxide poisoning: a prospective registry-based study, Clinical Toxicology, DOI: 10.1080/15563650.2023.2169707

To link to this article: https://doi.org/10.1080/15563650.2023.2169707

CLINICAL TOXICOLOGY
https://doi.org/10.1080/15563650.2023.2169707

Taylor & Francis
Taylor & Francis Group

CLINICAL RESEARCH

Dexamethasone therapy prevents delayed neuropsychiatric sequelae after carbon monoxide poisoning: a prospective registry-based study

Sechan Kim[a], Sungwoo Choi[a], Yujin Ko[b,c], Choung Ah Lee[d], Gi Woon Kim[a], Ji Eun Moon[e], Sangun Nah[a]* and Sangsoo Han[a]*

[a]Department of Emergency Medicine, Soonchunhyang University Bucheon Hospital, Bucheon, Republic of Korea; [b]Department of Psychiatry, Soonchunhyang University Bucheon Hospital, Bucheon, Republic of Korea; [c]Department of Psychiatry, Gangnam Severance Hospital, Yonsei University College of Medicine, Seoul, Republic of Korea; [d]Department of Emergency Medicine, Hallym University Dongtan Sacred Heart Hospital, Hwaseong, Republic of Korea; [e]Department of Biostatistics, Clinical Trial Center, Soonchunhyang University Bucheon Hospital, Bucheon, Republic of Korea

ABSTRACT

Background: Delayed neuropsychiatric sequelae are major complications of carbon monoxide poisoning; carbon monoxide triggers brain oxidation and inflammation. Corticosteroids such as dexamethasone modulate neurological damage after carbon monoxide poisoning through anti-inflammatory actions and immune response inhibition. However, it is not known whether corticosteroids prevent delayed neuropsychiatric sequelae. We thus studied whether dexamethasone reduced the incidence of delayed neuropsychiatric sequelae.

Methods: This registry-based study enrolled patients with carbon monoxide poisoning treated in a Korean tertiary care hospital from March 1st, 2020 to November 30th, 2021. Data of patients were prospectively collected during the study period, and retrospectively analyzed. One group received intravenous dexamethasone. We performed multivariable logistic regression analysis to identify factors associated with delayed neuropsychiatric sequelae.

Results: A total of 128 patients were enrolled, of which 99 patients received dexamethasone therapy and 29 patients did not. The incidences of delayed neuropsychiatric sequelae in the dexamethasone and non-dexamethasone groups were 16.2% and 37.9%, respectively. Multivariable logistic regression analysis revealed that dexamethasone use (odds ratio = 0.122, 95% confidence interval 0.031–0.489) and a higher Glasgow Coma Scale (odds ratio = 0.818, 95% confidence interval 0.682–0.981) was associated with a lower incidence of delayed neuropsychiatric sequelae.

Conclusion: Early dexamethasone treatment was significantly associated with a decreased incidence of delayed neuropsychiatric sequelae. A higher *Glasgow Coma Scale* at presentation also was associated with a lower incidence of delayed neuropsychiatric sequelae.

ARTICLE HISTORY
Received 28 June 2022
Revised 5 January 2023
Accepted 12 January 2023

KEYWORDS
Carbon monoxide; dexamethasone; poisoning; neuropsychiatric sequelae

Introduction

Carbon monoxide (CO) poisoning is a global health problem. In the United States, about 50,000 such patients visit emergency departments (EDs) every year [1]. Carbon monoxide is colorless, tasteless, and odorless, and has a 250-fold greater affinity for hemoglobin than oxygen. Carbon monoxide (even at low concentrations) causes tissue hypoxia by compromising oxygen transportation and dissociation [2]. Carbon monoxide poisoning is one of the main causes of death and morbidity attributable to poisoning; the worldwide incidence and mortality rates are respectively 137 and 4.6 per million annually [3,4].

Carbon monoxide principally damages organs such as the heart and brain, which exhibit high metabolic activities and are thus highly dependent on oxygen supply [5]. Delayed neuropsychiatric sequelae are new neuropsychiatric symptoms that develop a few days to 6 weeks following symptom absence after acute CO poisoning [6]. The symptoms include confusion, psychotic behavior, speech disorders, emotional dysregulation, and cognitive dysfunction [7]. The pathophysiology of CO poisoning is mainly due to inflammatory response and hypoxia. The etiology of delayed neuropsychiatric sequelae is unclear, but it is presumed to be caused by cell apoptosis, necrosis of neuronal cells, interfered cellular respiration, and lipid peroxidation [8].

Hyperbaric oxygen therapy, corticosteroids, and acetylcysteine have been suggested to treat acute CO poisoning and; these treatments exert antioxidant activities or serve as anti-inflammatory and immunosuppressive therapies [9]. Several studies have shown that corticosteroids such as dexamethasone and prednisolone modulate the neurological damage after CO poisoning [6,10–13]. However, the aforementioned studies were animal-targeted studies or studies on the efficacy of corticosteroid treatment in patients already diagnosed with delayed neuropsychiatric sequelae.

CONTACT Sangsoo Han brayden0819@daum.net; Sangun Nah potter325@naver.com Department of Emergency Medicine, Soonchunhyang University Bucheon Hospital, 170 Jomaru-ro, Bucheon, 14584, Republic of Korea
*These authors contributed equally to this work as the co-corresponding author.

Supplemental data for this article can be accessed online at https://doi.org/10.1080/15563650.2023.2169707

We hypothesized that early dexamethasone therapy for patients with acute CO poisoning might significantly reduce delayed neuropsychiatric sequelae development.

Materials and methods

Study design

This prospective-registry-based study was conducted in an ED of an urban tertiary care hospital (in Bucheon, Korea) that receives over 70,000 patient visits per year. We prospectively collected a registry of information on patients with CO poisoning commencing in March 2020. The diagnostic criteria of CO poisoning include the appropriate symptoms and history suggesting for exposure and an elevated the carboxyhemoglobin concentration. Symptoms that may include nausea, vomiting, dizziness, confusion, chest pain, fatigue, shortness of breath, and loss of consciousness [14]. And carboxyhemoglobin concentrations exceeding 5% in non-smokers (10% in smokers) at the time of ED arrival were used as threshold values for diagnosing CO poisoning. This study was approved by the hospital Institutional Review Board (IRB file no. 2020-03-019). Our study was based on retrospective analysis of a data registry, so informed consent was waived.

Study population selection

We retrospectively studied CO poisoned patients in our registry, who visited the ED from March 1st, 2020 to November 30th, 2021. Corticosteroid treatment was initiated after a change in our treatment protocol that commenced in July 1^{st,} 2020. All patients who visited following that date received dexamethasone if it was not contraindicated. The exclusion criteria were age under 18 years, discharged against medical advice, lost to follow-up, and persistent neurological symptoms at discharge (patients who showed persistent neurologic deficits, which was occurred after acute CO poisoning or during hospitalization). If side effects (such as uncontrolled hyperglycemia) developed during dexamethasone use, therapy was stopped and the patient was excluded.

Clinical and laboratory measurement

The clinical features recorded in the registry included age; gender; vital signs; comorbidities; any underlying disease; smoking status; the Glasgow Coma Scale (GCS); symptoms (headache, loss of consciousness, dizziness, dyspnea, chest pain); laboratory findings (white blood cell count, hemoglobin, total protein, albumin, glucose, blood urea nitrogen, creatinine, troponin I, C-reactive protein, lactate, and carboxyhemoglobin concentrations and creatine kinase activity); the CO exposure duration; the CO exposure type; administration of hyperbaric oxygen (or not); and delayed neuropsychiatric sequelae status. The total exposure time of CO was determined by history estimated maximum possible exposure time was recorded for uncertain exposure times. If it was unclear or difficult to determine from the patient, the information was obtained and estimated from emergency medical service (EMS) providers or caregivers.

Since there are no established diagnostic criteria for delayed neuropsychiatric sequelae, we referred to previous research for definitions [9,12]. Therefore, patients admitted to our hospital for CO poisoning routinely had magnetic resonance imaging (MRI) performed and were examined by a neurologist, before admission and discharge. Delayed neuropsychiatric sequelae was defined as a change in clinical neuropsychiatric status within 3 months after discharge (thus when the acute CO poisoning had been completely treated) [15]. Therefore, our total research period was from March 1st, 2020 to February 28th, 2022. The features that we considered to be due to delayed neuropsychiatric sequelae were impaired mental activity (e.g., concentration, memory, or cognitive disorders; dementia; headache; dizziness; delayed mental activity; dysarthria; or mutism); emotional dysregulation (e.g., depression, anxiety, obsession, or a sleep disorder); motor impairment (e.g., a tremor, gait disorder, or ataxia); and defecation and urinary impairments (e.g., urinary and/or fecal incontinence) [16].

The neuropsychiatric symptoms after CO poisoning can be mistaken for psychiatric diseases. Therefore, if poisoning was intentional or there was a relevant psychiatric history, a psychiatrist interviewed the patients at the beginning and the end of hospitalization. If such patients experienced symptoms of delayed neuropsychiatric sequelae within 3 months, they were referred to the psychiatrists who had conducted the initial interviews. Psychiatrists decided whether psychological symptoms worsened after CO poisoning, or were newly developed. Depression was diagnosed if a patient showed five or more symptoms of Diagnostic and Statistical Manual of Mental Disorders-5. The neurologist diagnosed delayed neuropsychiatric sequelae based on a patient's history, and brain imaging, excluding other neuropsychiatric conditions.

Patients without neuropsychiatric symptoms were informed about delayed neuropsychiatric sequelae before discharge, and advised to visit our neurology and psychiatry departments or ED when suspected symptoms occurred later. Thus, at 2 and 6 weeks and 3 months after discharge, delayed neuropsychiatric sequelae symptoms were investigated *via* telephone interviews with patients and their legal guardians, and the information was cross-checked. The telephone interviews were performed by two emergency medicine physicians, who were trained in the toxicology of CO poisoning. Both interviewers were blinded to the patient's data including administration of dexamethasone. Figure S1 shows our checklist for delayed neuropsychiatric sequelae which was used to evaluate and exclude symptoms of delayed neuropychiatric sequelae on telephone inteview.

If any of the symptoms of delayed neuropsychiatric sequelae were suspected by telephone interview, we asked patients to revisit us for examination by a neurologist, psychiatrist, or emergency medicine physician. These physicians had over 3 years of experience in treating CO poisoning and related complications including delayed neuropyschiatric sequelae. Our physicians tried to confirm the subjective

symptoms and abnormalities. For all patients who revisited our medical center for suspected symptoms of DNS, a Mini-Mental State Examination was administered and non-contrast MRI was performed [17,18]. Cognitive disorder was confirmed when Mini-Mental State Examination score was less than 24.

Treatment protocol

Dexamethasone 10 mg per day was given intravenously for 3 days at 24-h intervals immediately after admission [12,19]. Before administration, we checked for contraindications (cardiovascular disease, diabetes, glaucoma, or an infectious disease). If a risk factor was evident, dexamethasone was administered only when the therapeutic benefit was adjudged to be greater than the risk [20]. Regular laboratory tests were performed and the vital signs checked; any side-effects (such as uncontrolled hyperglycemia) were recorded.

In South Korea, EMS providers at pre-hospital stage provide 15 L/min oxygen *via* non-breather face mask for CO poisoned patients before ED arrival. After ED arrival, all patients were immediately treated with 15 L/min oxygen *via* non-breather mask with reservoir bag before HBOT. Hyperbaric oxygen therapy was applied as soon as possible if the patient met the indications. The indications were carboxyhemoglobin concentration of 25% or more, neurological deficits (changes in consciousness, convulsions, or cognitive dysfunction), and suspected myocardial damage (persistent chest pain and/or cardiac marker elevations) [21].

Hyperbaric oxygen therapy was performed three times at 6–12 h intervals within 24 h. The first session was conducted at 3 atmospheres for 150 min, and the second and third sessions at 2 atmospheres for 120 min [22]. During hyperbaric oxygen therapy, pressurization was performed over the first 30 min of each session and decompression over the 30 min before the end of each session.

Statistical analysis

Continuous variables that were normally distributed are presented as means ± standard deviations and non-normally distributed data as medians with interquartile ranges (derived using the Shapiro–Wilk test). Categorical variables are presented as absolute values with percentages. We used a Student's *t*-test to compare normally distributed continuous data and the Mann–Whitney U-test to compare non-normally distributed data. Categorical variables were compared using the chi-squared and Fisher's exact tests when appropriate. We employed multivariable logistic regression analysis to identify factors associated with delayed neuropsychiatric sequelae; the results are presented as odds ratios (ORs) with 95% confidence intervals (CIs). A *P*-value less than 0.05 was considered significant; all *P*-values are two-sided. All statistical analyses were performed with SPSS for Windows software ver. 26.0 (IBM Corp., Armonk, NY, USA).

Results

A total of 163 CO-poisoned patients were identified during the study, of whom 33 were excluded: Age under 18 years (7 patients), discharge against medical advice (13), lost to follow-up (11), and persistent neurological symptoms at discharge (2). A total of 128 patients were included, after excluding 2 patients with uncontrolled hyperglycemia developing after dexamethasone use (those patients were not able to continue therapy for 3 days). Otherwise, there were no other patients who had contraindications. Patients were divided into a non-dexamethasone group (29 patients, 22.7%) and a dexamethasone group (99, 77.3%) (Figure 1).

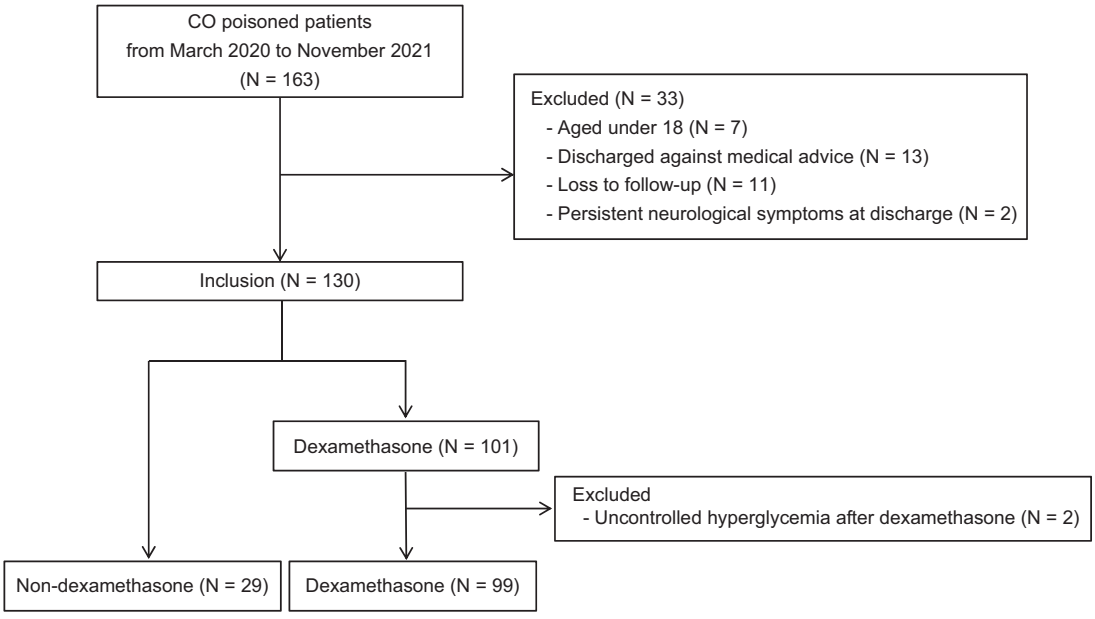

Figure 1. Flow chart of patent selection. CO: carbon monoxide.

From total of 163 patients, 42 patients were recommended for evaluation of suspected symptoms of delayed neuropsychiatric sequelae, and 38 patients revisited our medical center. Four patients who did not revisit to our center were excluded as 'lost to follow-up'. Table 1 shows the overall characteristics of the patients.

The comparison of variables between both groups is presented in supplementary Table S1.

Table 1. Patient demographics.

	Total ($N = 128$)
Age, years	43.5 ± 14.7
Sex, *n* (%)	
Female	42 (32.8)
Male	86 (67.2)
Comorbidities, *n* (%)	
Diabetes	12 (9.4)
Hypertension	8 (6.3)
Current smoker, *n* (%)	69 (58.5)
Duration of exposure to carbon monoxide, min	165 [60–360]
Exposure type, *n* (%)	
Unintentional	28 (21.9)
Intentional	100 (78.1)
Delayed neuropsychiatric sequelae, *n* (%)	27 (21.1)

Values are presented as means ± standard deviations, medians [interquartile ranges], or numbers (proportions).

Comparison of variables between the non-dexamethasone and dexamethasone groups

Table 2 shows the differences between the non-dexamethasone and dexamethasone groups. The median GCS of both groups were 15 ($P = 0.352$). The median CO exposure time was 150 min in the non-dexamethasone group and 180 min in the dexamethasone group ($P = 0.236$). Of patients with intentional CO poisoning, 22 were in the non-dexamethasone and 78 in the dexamethasone group.

The non-dexamethasone and dexamethasone group differed significantly in terms of comorbidities such as diabetes (13.8 vs. 8.1%; $P = 0.015$), loss of consciousness at the time of CO exposure (48.3 vs. 26.2%; $P = 0.043$), the troponin I concentration (0.1 vs. 0.09 ng/mL; $P = 0.008$), and delayed neuropsychiatric sequelae development (37.9 vs. 16.2%; $P = 0.023$).

Predictors of DNS: Multivariable logistic regression analysis

Multivariable logistic regression analysis revealed that the use of dexamethasone (OR = 0.122, 95% CI 0.031–0.489) and a greater GCS score at presentation (OR = 0.818, 95% CI

Table 2. Comparison of the non-dexamethasone and dexamethasone patient groups.

	Non-dexamethasone group (*n* = 29)	Dexamethasone group (*n* = 99)	*P* Value
Age, years	46.5 ± 17.8	42.6 ± 13.7	0.289
Sex, *n* (%)			>0.99[a]
Female	10 (34.5)	32 (32.3)	
Male	19 (65.5)	67 (67.7)	
Vital signs			
Systolic blood pressure, mmHg	130 [110–140]	130 [115–140]	0.890
Diastolic blood pressure, mmHg	80 [70–90]	80 [70–90]	0.594
Heart rate, beats/min	93.1 ± 19.4	91 ± 17.9	0.591
Respiratory rate, breaths/min	20 [20–20]	20 [18–20]	0.9
Comorbidities, *n* (%)			
Diabetes	5 (17.2)	3 (3.0)	0.015[b]
Hypertension	4 (13.8)	8 (8.1)	0.467[b]
Current smoker, *n* (%)	16 (55.2)	53 (53.5)	>0.99[a]
Glasgow Coma Scale at presentation	15 [12–15]	15 [12.5–15]	0.352[b]
Score < 15, *n* (%)	13 (44.8)	32 (32.3)	0.308[a]
Symptoms, *n* (%)			
Headache	3 (10.3)	9 (9.1)	>0.99[b]
Loss of consciousness	14 (48.3)	26 (26.2)	0.043[a]
Dizziness	2 (6.9)	13 (13.1)	0.518[b]
Dyspnea	2 (6.9)	6 (6.1)	>0.99[b]
Chest pain	1 (3.5)	1 (1)	0.403[b]
Laboratory findings			
White blood cell count, $10^3/\mu L$	10.9 [8.7–13.7]	11.8 [8.8–15.4]	0.734
Hemoglobin, g/dL	14.5 [13.1–15]	15.1 [13.5–16]	0.106
Protein, g/dL	7.2 ± 0.5	7.3 ± 0.6	0.581
Albumin, g/dL	4.3 ± 0.4	4.4 ± 0.4	0.327
Glucose, mg/dL	124 [95.8–171.5]	113 [101.5–131]	0.559
Blood urea nitrogen, mg/dL	12.5 [10.3–20.6]	13 [11.1–16.8]	0.988
Creatinine, mg/dL	1.05 [0.8–1.3]	1 [0.9–1.2]	0.476
Creatine kinase, U/L	134 [81.5–538]	121 [84–433]	0.569
Troponin I, ng/mL	0.1 [0.1–0.2]	0.09 [0.05–0.15]	0.008
C-reactive protein, mg/L	0.2 [0.1–1.3]	0.1 [0–0.4]	0.206
Lactate, mg/dL	1.4 [1.2–4.2]	2.1 [1.8–4.7]	0.553
Carboxyhemoglobin, %	9.0 [3.8–13.7]	10.2 [5.5–17.6]	0.330
Duration of carbon monoxide exposure, min	150 [30–240]	180 [85–420]	0.236
Type of exposure, *n* (%)			0.936[a]
Unintentional	7 (24.1)	21 (21.2)	
Intentional	22 (75.9)	78 (78.8)	
Hyperbaric oxygen therapy, *n* (%)	21 (72.4)	85 (85.9)	0.101[b]
Delayed neuropsyciatric sequelae, *n* (%)	11 (37.9)	16 (16.2)	0.023[a]

Values are presented as means ± standard deviations, medians [interquartile ranges], or numbers (proportions).
[a]Pearson's χ^2 test,[b]Fisher's exact test.

Table 3. Multivariable logistic regression analysis of factors predictive of delayed neuropsychiatric sequelae.

	OR	95% CI
Dexamethasone use	0.122	0.031–0.489
Glasgow Coma Scale score	0.818	0.682–0.981
Carboxyhemoglobin, %	0.944	0.888–1.003
C-reactive protein, mg/L	0.988	0.836–1.168
Duration of exposure to carbon monoxide, min	1.001	0.999–1.003
Hyperbaric oxygen therapy	5.662	0.517–62.011

OR: odds ratio; CI: confidence interval.

0.682–0.981) was associated with a lower incidence of DNS (Table 3).

Discussion

This prospective registry-based study investigated the association between initial dexamethasone administration and DNS development in patients with CO poisoning. We found that dexamethasone usage was significantly associated with a reduced incidence of delayed neuropsychiatric sequelae (OR = 0.122).

Although the pathophysiology of DNS in patients with CO poisoning has not been clearly identified, DNS is presumed to reflect destruction of hypoxia-sensitive cells, disturbance of the mitochondrial electron transport system, demyelination of cerebral white matter, and lipid peroxidation [9]. Thom et al. [23] found that brain tissue damaged by CO produced malondialdehyde, which triggered an immunological cascade. Malondialdehyde alters myelin basic protein; the modified myelin basic protein is attacked by macrophages and $CD4^+$ lymphocytes, perhaps explaining delayed neuropsychiatric sequelae. We suggest that the dexamethasone may have prevented delayed neuropsychiatric sequelae by exerting anti-inflammatory and anti-immune actions [24]. Li et al. [12,13] reported that dexamethasone administration after CO poisoning in an animal model prevented DNS, lipid peroxidation, and the myelin basic protein-associated immune reaction, and reduced tissue ischemia and edema [13]. We also found that dexamethasone usage was significantly associated with a reduced incidence of DNS, perhaps by preventing lipid peroxidation and the myelin basic protein-associated immune reaction.

Xiang et al. [12] reported that a combination of HBOT and steroids in patients with DNS developing after CO poisoning significantly reduced the the concentration of myelin basic protein in cerebrospinal fluid and improved the MMSE scores [12]. We prescribed HBOT for almost all patients (85.9%) in the dexamethasone group. Clinicians treating patients with CO poisoning may thus try to add dexamethasone therapy to HBOT unless otherwise contraindicated. However, a future study comparing the concentrations of myelin basic protein in cerebrospinal fluid between patients treated with a combination of HBOT and dexamethasone, and dexamethasone monotherapy, is required.

Regarding the correlation between the GCS score and delayed neuropsychiatric sequelae development, Ku et al. [18] reported that low GCS scores correlated significantly with development of delayed neuropsychiatric sequelae in 43 CO-poisoned patients. Zhang et al. [25] in a multi-center study of 326 CO-poisoned patients, came to a similar conclusion. We also found that a high initial GCS was significantly associated with a low incidence of delayed neuropsychiatric sequelae (OR = 0.823). Prockop et al. [26] reported that acute and delayed brain injury caused by CO poisoning reflected hypoxic damage to brain tissue; the nature and distribution of such damage depended on the duration and severity of oxygen deficiency. Thus, both previous works and our study suggested that patients with lower initial GCS scores were exposed to CO longer or at higher concentrations. Such poisoning is associated with brain hypoxia and inflammation, and development of delayed neuropsychiatric sequelae.

Our work had several limitations. First, this was a single-center retrospective study with a small number of patients. It is thus difficult to generalize the results; also, there may have been some biases present. Other factors that may affect delayed neuropsychiatric sequelae were not fully controlled, and there was also a difference in the number of patients between the dexamethasone and non-dexamethasone group. Second, many patients sought to commit suicide, and such patients were often uncooperative with the medical treatment [27]. Carbon monoxide poisoned patients with suicidality often consume psychiatric drugs and alcohol at the same time, but we did not consider the toxic effects of drug usage [28,29]. Also, reliability of self-reported data (e.g., exposure time) may have been influenced if patients were uncooperative and had psychiatric comorbidities. Third, dexamethasone was given at only fixed dose for a set period. The preferred corticosteroid classes, and the optimal dose and administration period, require further study. Fourth, we additionally performed further study including MRI and MMSE only to patients who were suspected delayed neuropsychiatric sequelae, and it might bring bias. Fifth, although hyperbaric oxygen therapy is widely used as treatment for CO poisoning, its efficacy may be questioned in our study, along with other previous research [30,31]. Since our study was performed retrospectively, hyperbaric oxygen was not administered to all patients, and matching for each group was not attempted. Therefore, a well-designed prospective study comparing hyperbaric oxygen therapy and corticosteroids will be needed to overcome these limitations. Finally, our study did not consider the relationship between CO poisoning and seasonal changes in Korea. Korea has four distinct seasons, and it is known that incidence of CO poisoning fluctuated with the weather due to usage of coal and fuel for domestic heating [32]. To overcome these limitations, further research will be needed.

Conclusion

We found that early dexamethasone treatment of patients with carbon monoxide poisoning was significantly associated with a reduced incidence of delayed neuropsychiatric sequelae. Also, higher GCS score at presentation predicted lower incidence of delayed neuropsychiatric sequelae.

Disclosure statement

No potential conflict of interest was reported by the author(s).

Funding

This research was supported by the Soonchunhyang University Research Fund [grant no. 10220009].

ORCID

Sechan Kim http://orcid.org/0000-0001-5516-844X
Sungwoo Choi http://orcid.org/0000-0001-6548-4921
Yujin Ko http://orcid.org/0000-0003-4201-9343
Gi Woon Kim http://orcid.org/0000-0003-2720-7442
Ji Eun Moon http://orcid.org/0000-0003-3541-058X
Sangun Nah http://orcid.org/0000-0003-2667-2211
Sangsoo Han http://orcid.org/0000-0002-9709-3332

References

[1] Hampson NB. Cost of accidental carbon monoxide poisoning: a preventable expense. Prev Med Rep. 2016;3:21–24.
[2] Ernst A, Zibrak JD. Carbon monoxide poisoning. N Engl J Med. 1998;339(22):1603–1608.
[3] Hampson NB. US mortality due to carbon monoxide poisoning, 1999–2014. Accidental and intentional deaths. Ann Am Thorac Soc. 2016;13(10):1768–1774.
[4] Mattiuzzi C, Lippi G. Worldwide epidemiology of carbon monoxide poisoning. Hum Exp Toxicol. 2020;39(4):387–392.
[5] Nah S, Choi S, Kim HB, et al. Cerebral white matter lesions on diffusion-weighted images and delayed neurological sequelae after carbon monoxide poisoning: a prospective observational study. Diagnostics (Basel). 2020;10(9):698.
[6] Sönmez BM, İşcanlı MD, Parlak S, et al. Delayed neurologic sequelae of carbon monoxide intoxication. Turk J Emerg Med. 2018;18(4):167–169.
[7] Choi S. Delayed neurologic sequelae in carbon monoxide intoxication. Arch Neurol. 1983;40(7):433–435.
[8] Weaver LK. Carbon monoxide poisoning. N Engl J Med. 2009;360(12):1217–1225.
[9] Oh S, Choi S-C. Acute carbon monoxide poisoning and delayed neurological sequelae: a potential neuroprotection bundle therapy. Neural Regen Res. 2015;10(1):36–38.
[10] Rose JJ, Wang L, Xu Q, et al. Carbon monoxide poisoning: pathogenesis, management, and future directions of therapy. Am J Respir Crit Care Med. 2017;195(5):596–606.
[11] Iwamoto K, Ikeda K, Mizumura S, et al. Combined treatment of methylprednisolone pulse and memantine hydrochloride prompts recovery from neurological dysfunction and cerebral hypoperfusion in carbon monoxide poisoning: a case report. J Stroke Cerebrovasc Dis. 2014;23(3):592–595.
[12] Xiang W, Xue H, Wang B, et al. Combined application of dexamethasone and hyperbaric oxygen therapy yields better efficacy for patients with delayed encephalopathy after acute carbon monoxide poisoning. Drug Des Devel Ther. 2017;11:513–519.
[13] Li Q, Song J, Zhang H, et al. Dexamethasone therapy for preventing delayed encephalopathy after carbon monoxide poisoning. Biotech Histochem. 2015;90(8):561–567.
[14] Kao LW, Nañagas KA. Carbon monoxide poisoning. Emerg Med Clin North Am. 2004;22(4):985–1018.
[15] Ide T, Kamijo Y, Ide A, et al. Elevated S100B level in cerebrospinal fluid could predict poor outcome of carbon monoxide poisoning. Am J Emerg Med. 2012;30(1):222–225.
[16] Wang P, Zeng T, Chi Z-F Recovery of cognitive dysfunction in a case of delayed encephalopathy of carbon monoxide poisoning after treatment with donepezil hydrochloride. Neurol India. 2009;57(4):481.
[17] Otubo S, Shirakawa Y, Aibiki M, et al. Magnetic resonance imaging could predict delayed encephalopathy after acute carbon monoxide intoxication. Jpn J Toxicol. 2007;20(3):253–261.
[18] Ku H-L, Yang K-C, Lee Y-C, et al. Predictors of carbon monoxide poisoning-induced delayed neuropsychological sequelae. Gen Hosp Psychiatry. 2010;32(3):310–314.
[19] Ma X. Clinical study of large dose of dexamethasone combined with hyperbaric oxygen in treatment of acute carbon monoxide poisoning. Med Innov China. 2013;10(23):45–47.
[20] Poetker DM, Reh DD. A comprehensive review of the adverse effects of systemic corticosteroids. Otolaryngol Clin North Am. 2010;43(4):753–768.
[21] Undersea, Gesell LB. Committee HMSHO Hyperbaric oxygen therapy indications: the hyperbaric oxygen therapy committee report: undersea and Hyperbaric Medical Society 2008.
[22] Weaver LK, Hopkins RO, Chan KJ, et al. Hyperbaric oxygen for acute carbon monoxide poisoning. N Engl J Med. 2002;347(14):1057–1067.
[23] Thom SR, Bhopale VM, Fisher D, et al. Delayed neuropathology after carbon monoxide poisoning is immune-mediated. Proc Natl Acad Sci U S A. 2004;101(37):13660–13665.
[24] Xiang W-P, Xue H, Wang B-J. Delayed encephalopathy of acute carbon monoxide intoxication in rats: potential mechanism and intervention of dexamethasone. Pak J Pharm Sci. 2014;27(6 Suppl):2025–2028.
[25] Zhang Y, Lu Q, Jia J, et al. Multicenter retrospective analysis of the risk factors for delayed neurological sequelae after acute carbon monoxide poisoning. Am J Emerg Med. 2021;46:165–169.
[26] Prockop LD, Chichkova RI. Carbon monoxide intoxication: an updated review. J Neurol Sci. 2007;262(1-2):122–130.
[27] Hampson NB, Bodwin D. Toxic CO-ingestions in intentional carbon monoxide poisoning. J Emerg Med. 2013;44(3):625–630.
[28] Choi Y-R, Cha ES, Chang S-S, et al. Suicide from carbon monoxide poisoning in South Korea: 2006–2012. J Affect Disord. 2014;167:322–325.
[29] Cherpitel CJ, Borges GL, Wilcox HC. Acute alcohol use and suicidal behavior: a review of the literature. Alcoholism: clinical and Experimental Research. 2004;28:18S–28S.
[30] Gilmer B, Kilkenny J, Tomaszewski C, et al. Hyperbaric oxygen does not prevent neurologic sequelae after carbon monoxide poisoning. Acad Emerg Med. 2002;9(1):1–8.
[31] Scheinkestel CD, Myles PS, Cooper DJ, et al. Hyperbaric or normobaric oxygen for acute carbon monoxide poisoning: a randomised controlled clinical trial. Med J Aust. 1999;170(5):203–210.
[32] Kim YS. Seasonal variation in carbon monoxide poisoning in urban korea. J Epidemiol Community Health. 1985;39(1):79–81.