Hyperbaric Oxygen Therapy for the Prevention and Treatment of Delayed Neurological Sequelae after Carbon Monoxide Poisoning

Sirada Chaichanachuchird,* Thanasawat Chaiyakul**
* Resident in Preventive Medicine (Maritime Medicine), Naval Medical Department, Royal Thai Navy, Bangkok
** Division of Preventive Medicine, Naval Medical Department, Royal Thai Navy, Bangkok
* Corresponding Author: sirada.chaichanachuchird@gmail.com

Abstract

Carbon monoxide (CO) poisoning remains a prevalent medical emergency with potentially fatal outcomes. Long-term neurological consequences, known as delayed neurological sequelae (DNS), may manifest several days to weeks following the acute exposure. Hyperbaric oxygen (HBO_2) has appeared as a valuable intervention in severe cases, enhancing the CO dissociation from hemoglobin and aiding tissue oxygenation. Recent case studies and clinical reports suggest that HBO_2 not only reduces the severity of acute CO poisoning but also plays a critical role in preventing and treating DNS, leading to improvements in neurocognitive function. The article aims to describe DNS and discuss the demonstrated benefits of HBO_2 in the treatment and prevention of neurological sequelae, emphasizing its potential to enhance recovery and reduce long-term neurological damage.

Keywords: carbon monoxide poisoning, Hyperbaric oxygen therapy, delayed neurological sequelae, prevention, DNS

Introduction

Carbon monoxide (CO) poisoning is one of the prevalent medical emergencies which could result in fatal outcomes. It is a poisonous gas that can be produced endogenously by the breakdown of heme-protein and exogenously by the incomplete combustion of carbon-fueled substances[1]. In Thailand, common sources of carbon monoxide are forest fires, biomass burning, motor vehicles, fuel combustion, and some industries with inadequate ventilation systems[2-4]. CO has been termed a "silent killer" due to its colorless, tasteless, odorless, and non-irritant properties[1].

Confusion, dizziness, nausea, abdominal pain, chest pain, and dyspnea are some of

Received: August 28, 2024; Revised: October 28, 2024; Accepted: November 9, 2024

the immediate symptoms of CO poisoning[5]. Carboxyhemoglobin (COHb) levels do not always correlate with the severity of symptoms, as various factors influence the clinical outcome[6]. However, COHb levels above 50% are considered lethal and can lead to severe symptoms such as coma, convulsions, respiratory distress, and even death[7-9]. In CO poisoning, patients often suffer from long term psychoneurological consequences that may appear immediately after exposure and continue over time, referred to as persistent neurological sequelae (PNS). In some cases, symptoms can arise days to weeks after the initial recovery phase, known as delayed neurological sequelae (DNS), which generally result in worse clinical outcomes compared to PNS[10]. The incidence of DNS varies from one study to another but is typically reported in about 6 - 40% of survivors of acute CO poisoning[11-15]. These symptoms can occur 2 days to 6 weeks after recovery from the injury[15-18]. Although some cases have been reported as late as 8 months, they typically appear within 20 days[5,8].

A non-rebreathing face mask with 100 percent normobaric oxygen (NBO) is the standard therapy for acute CO poisoning[19-20]. For patients with severe acute carbon monoxide poisoning, hyperbaric oxygen (HBO_2) is frequently suggested[21]. Although several studies have shown that HBO_2 improves clinical outcomes and may reduce DNS more than NBO in CO poisoning patients, there are currently debates about using HBO_2 for CO poisoning treatment and its potential benefit in preventing the development of DNS[22].

Mechanism of CO Poisoning and Development of DNS

CO is estimated to have a 240-fold higher affinity for hemoglobin than oxygen, creating a compound known as carboxyhemoglobin (COHb)[23]. As a result, there is less hemoglobin in the blood that can carry oxygen, resulting in tissue hypoxia. CO also binds to myoglobin and mitochondrial cytochrome oxidase, limiting mitochondrial respiration and interrupting oxygen transportation especially in the heart and brain[6]. This process decreases ATP production and disrupts the electron transport chain, leading to an influx of calcium ions into cells that worsens brain damage. Furthermore, CO induces reactive oxygen species (ROS) and nitric oxide production, causing oxidative stress. CO also stimulates platelet and neutrophil-endothelial adhesion, which results in the release of inflammatory mediators[17]. These inflammatory mediators and ROS lead to lipid peroxidation, particularly in the brain. This mechanism, which causes neuronal damage, is expected to have a considerable impact on the development of DNS by converting myelin basic protein (MBP) into adduct formation[6,24]. This alteration increases MBP's vulnerability to proteases, disrupts antibody recognition, triggers adaptive immune responses, and contributes to CO-induced delayed demyelination. This process begins after exposure to CO and continues during the lucid interval, a phase where patients show no noticeable neuropsychiatric symptoms or white matter changes on MRI scans. Once demyelination surpasses a critical threshold,

white matter alterations occur, MBP levels elevate, and neuropsychiatric symptoms of DNS emerge[25]. Furthermore, some authors have suggested that DNS development may be influenced by factors aside from demyelination, such as excessive activation of glial cells, inflammatory responses triggered by astrocytes, and dysfunction of oligodendrocytes[25-26].

Diagnosis of DNS

Although previous studies have revealed various clinical symptoms of DNS, there are currently no standard diagnostic criteria. DNS was frequently diagnosed based on the history of CO exposure, a thorough neurological examination, clinical symptoms, and consultation with a neurologist or psychiatrist[27-28].

DNS is difficult to diagnose because of the lucid intervals that range from days to weeks; also, various illnesses might resemble DNS presentations[12]. DNS symptoms include altered awareness, cognitive decline, difficulty concentrating, urine incontinence, parkinsonism, seizures, peripheral neuropathies, psychosis, changes in personality, depression, insomnia, loss of memory, and anxiety[21,29-30]. The Mini-Mental State Examination (MMSE) or other psycho-cognitive test is commonly used to assess disorientation and cognitive impairment [12,14-15].

Imaging studies and Prognostic Factors

During acute CO poisoning, no clinical signs or reliable predictors can help physicians identify which patients will develop DNS. However, the findings of a computerized tomography scan (CT-scan) and magnetic resonance imaging (MRI) might assist in the diagnosis of DNS[12,18]. One of the prognostic indicators of DNS that can be seen by CT-scan in the acute phase is cerebral edema. When a CT scan reveals diffuse cerebral edema at an early stage, the prognosis is poor[17]. Diffusion-weighted magnetic resonance imaging (DW-MRI) is one of the most sensitive and highly effective at early detection of acute ischemic brain lesions and CO-induced hypoxia. They can identify CO-related cytotoxic edema if performed within 72 hours of exposure, aiding early diagnosis of delayed DNS[31]. A meta-analysis of eight trials found that DNS could be diagnosed using MRI with 71% sensitivity and 85% specificity[18]. Typically, gray matter is more susceptible to cerebral hypoxia than white matter. However, during acute CO poisoning, pathological changes were observed in the white matter, while the adjacent gray matter showed few alterations[17]. Globus pallidus is one of the locations that is sensitive to hypoxia after carbon monoxide poisoning[18]. Symmetrical hyperintensities in the bilateral globus pallidus, cerebral white matter demyelination, cerebral atrophy, and hippocampus, thalamic, and basal ganglia abnormalities are typical MRI findings associated with DNS[30-32]. However, no official agreement on the results of an MRI associated with DNS was made.

Currently, there are no reliable predictors of DNS. Many studies have proposed predictive factors for DNS such as Glasgow coma score[12,33],

CO exposure duration[34], time to HBOT after CO exposure[34], carboxyhemoglobin level[15], abnormal findings on MRI[33], QTc prolongation on electrocardiogram[35], and creatinine kinase level[12,14,36] but none are conclusive. Therefore, individuals suffering from CO poisoning should be regularly observed, warned about the danger of developing DNS, and screened for neurocognitive sequelae 4 - 6 weeks following CO injury[19].

The Role of HBO_2 in Prevention of DNS

Hyperbaric oxygen (HBO_2) is a medical procedure in which the patient's entire body is placed inside a hyperbaric chamber while breathing medical-grade oxygen (> 99% purity) at a pressure not less than 2.0 ATA., with most clinical treatments using a pressure between 2.0 and 3.0 ATA[6]. HBO_2 assists in the dissociation of CO from hemoglobin and the transport of oxygen to tissues. When a patient breathes air at sea level, the half-life of COHb is approximately 5 hours. With administration of HBO_2 at 3 ATA, the half-life of COHb decreases to around 30 minutes. HBO_2 also provides several actions that relieve central nervous system (CNS) damage and prevent the development of DNS. It provides a protective oxidative stress response through the upregulation of heme oxygenase and antioxidant enzymes[17]. Furthermore, HBO_2 decreases leukocyte adhesion to endothelial cells, cytotoxic neurotransmitters, alteration in myelin protein structure, immune-mediated damage, lipid peroxidation, and neuronal apoptosis, all of which are postulated pathways for DNS following CO exposure[6,24].

Over the last few decades, there is an ongoing discussion over how HBO_2 prevents the development of neurocognitive sequelae. While some research contends that HBO_2 does not improve the outcome, many studies have demonstrated that HBO_2 can decrease the chance of developing DNS more than normobaric oxygen[37-38].

According to a 2018 meta-analysis that evaluated the data from 6 RCTs, patients who receive HBO_2 treatment after CO poisoning have less neurocognitive symptoms such as memory loss, headache, concentration problems, sleep disturbances, and lower incidence of DNS than those who receive NBO treatment[37]. A large-scale observational study (n = 2,034) by Nakajima et al. also found that patients who received HBO_2 within one day of admission had lower rates of depressed mental status at discharge than those who did not receive HBO_2 (n = 4,701)[38].

In contrast, research by Han et al. that followed 224 patients for six months, DNS incidence was not significantly different between individuals treated for acute CO poisoning with HBO_2 and those treated with normobaric oxygen (NBO)[39]. A similar conclusion was drawn from another large-scale study in Japan, which noted no notable difference in DNS incidence between the HBO_2 and NBO groups (3.6% vs. 7.6%). However, this study had inconsistent HBO_2 protocols and

did not involve patients with minor neurological symptoms which could possibly affect the outcome[13]. Furthermore, there's evidence indicating that more HBO_2 sessions might increase the chances of developing DNS[28].

Moreover, the optimum session of HBO_2 for CO poisoning patients is not universally agreed upon. Numerous research studies have examined the most effective HBO_2 protocols for the best neurological outcomes.

In a study by Weaver et al., it was demonstrated that receiving 3 HBO_2 sessions within 24 hours appeared to lower the likelihood of neurocognitive sequelae at six and twelve months following acute carbon monoxide poisoning[40]. However, there were no significant improvements in cognitive sequelae at one month following CO poisoning between patients who received single session and multiple-session (two or three sessions) of HBO_2, based on a recent study in 2023[41]. Wang et al.'s meta-analysis also reported that 2 HBO_2 sessions might not be more advantageous for memory loss than 1 HBO_2 session[42].

The optimal therapeutic pressure used also differs among the studies, ranging from 2.0 - 3.0 ATA[13,42]. According to several publications, HBO_2 treatment does not have any positive benefits at therapeutic pressures lower than 2.5 ATA. Based on a study by Thom et al., HBO_2 at 2.8 - 3.0 ATA can inhibit neutrophil adhesion to brain endothelial cells leading to prevention of CO-mediated brain lipid peroxidation, which is an important mechanism that leads to development of DNS. At all events, HBO_2 therapy should be administered without delay, ideally within 24 hours after being exposed CO[40,43-44].

The Role of HBO_2 in Treatment of DNS

For the treatment of DNS, the role of HBO_2 in DNS remains inconclusive as well. The proposed mechanism of HBO_2 for treating DNS includes reducing oligodendrocyte injury, promoting the remyelination of axons in white matter, stimulating differentiation of precursor cell[45]. It could be related to transferring mitochondria to the injury site, producing anti-inflammatory cytokines, protecting blood-brain barrier integrity, and promoting neurogenesis, as seen in animal models[46]. Several case reports have shown that HBO_2 is beneficial for treating patients who have been diagnosed with DNS[16,45,47].

A case report by Tapeantong et al. indicated that a patient who developed DNS four weeks after recovering from the acute phase of CO poisoning experienced improvements in memory, cognitive functions, and significant reduction of brain white matter lesions on MRI after HBO_2[48]. Furthermore, a recent study by Wong et al. reported a case of a patient who developed severe neurological symptoms resembling Parkinsonism eight weeks after CO poisoning, with MRI findings consistent with hypoxic-ischaemic encephalopathy. After 20 sessions of HBO_2 at 2.0 ATA and intravenous methylprednisolone,

the patient remarkably fully recovered and was able to return to work without recurrence[47]. Similarly, Martini et al. reported successful outcomes in two DNS patients who underwent 21 - 24 sessions of HBO_2 at 2.5 ATA alongside anti-inflammatory drugs and rehabilitation, though the effectiveness may have been influenced by the use of adjunctive treatments such as corticosteroids and acetylcysteine[16]. Liao et al. study also found that early HBO_2 significantly decreased the symptoms of DNS in patients after being exposed to CO, and the benefits of the treatment persisted for a year following DNS diagnosis[45].

To determine whether HBO_2 is an effective treatment for DNS, larger studies are needed. These studies should ideally include controlled trials with larger sample sizes to provide more reliable data.

Conclusion

Hyperbaric oxygen therapy (HBO_2) holds potential as a therapeutic intervention for the prevention and treatment of DNS, a condition that commonly manifests weeks after CO exposure. In severe cases, such as those involving loss of consciousness or COHb levels exceeding 25%, early HBO_2 administration is recommended, followed by ongoing evaluation of psychocognitive symptoms. Although recent studies have highlighted the benefits of HBO2, its use remains debatable due to inconsistencies in research results, which may arise from variations in HBO_2 protocols, exclusion of certain patient groups, and differences in treatment duration or pressure. To reach definitive conclusions and improve patient outcomes, especially in severe cases, standardized research methodologies are urgently needed.

References

1. Mattiuzzi C, Lippi G. Worldwide epidemiology of carbon monoxide poisoning. Hum Exp Toxicol 2020;39(4):387-92.
2. Hampson NB, Hauschildt KL, Deru K, Weaver LK. Carbon monoxide poisonings in hotels and motels: the problem silently continues. Prev Med Rep 2019;16:100975.
3. Khodmanee S, Amnuaylojaroen T. Impact of biomass burning on ozone, carbon monoxide, and nitrogen dioxide in Northern Thailand. Front Environ Sci 2021;9:641877.
4. Velasco E, Ho KJ, Ziegler AD. Commuter exposure to black carbon, carbon monoxide, and noise in the mass transport khlong boats of Bangkok, Thailand. Transportation Research Part D: Transport and Environment 2013;21:62-5.
5. Manaker S, Perry H. Carbon monoxide poisoning. [Internet]. [cited 2024 Aug 12]. Available from: https://www.uptodate.com/contents/carbon-monoxide-poisoning.
6. Huang E. UHMS hyperbaric medicine indications manual. 15th ed. North Palm Beach, FL: Best Publishing; 2024.

7. Creswell PD, Meiman JG, Nehls-Lowe H, Vogt C, Wozniak RJ, Werner MA, et al. Exposure to elevated carbon monoxide levels at an indoor ice arena-Wisconsin, 2014. MMWR Morb Mortal Wkly Rep 2015;64(45):1267-70.
8. Savioli G, Gri N, Ceresa IF, Piccioni A, Zanza C, Longhitano Y, et al. Carbon monoxide poisoning: from occupational health to emergency medicine. J Clin Med 2024;13(9):2466.
9. Palmeri R, Gupta V. Carboxyhemoglobin toxicity. In: StatPearls [Internet]. Treasure Island (FL): StatPearls Publishing; 2025 Jan-PMID: 32491811.
10. Ning K, Zhou YY, Zhang N, Sun XJ, Liu WW, Han CH. Neurocognitive sequelae after carbon monoxide poisoning and hyperbaric oxygen therapy. Med Gas Res 2020;10(1):30-6.
11. Huang CC, Ho CH, Chen YC, , Hsu CC, Wang YF, Lin HJ, et al. Impact of hyperbaric oxygen therapy on subsequent neurological sequelae following carbon monoxide poisoning. J Clin Med 2018;7(10):349.
12. Han S, Choi S, Nah S, Lee SU, Cho YS, Kim GW, et al. Cox regression model of prognostic factors for delayed neuropsychiatric sequelae in patients with acute carbon monoxide poisoning: a prospective observational study. Neurotoxicology 2021;82:63-8.
13. Fujita M, Todani M, Kaneda K, Suzuki S, Wakai S, Kikuta S, et al. Use of hyperbaric oxygen therapy for preventing delayed neurological sequelae in patients with carbon monoxide poisoning: a multicenter, prospective, observational study in Japan. PLoS One 2021;16(6):e0253602.
14. Lee H, Kang H, Ko BS, Oh J, Lim TH, Cho Y. Initial creatine kinase level as a predictor for delayed neuropsychiatric sequelae associated with acute carbon monoxide poisoning. Am J Emerg Med 2021;43:195-9.
15. Gaballah SZ, elkhishen IAR, Hashim NA, Hamid OIA. Predictors of delayed neurological sequelae after acute carbon monoxide poisoning at Zagazig University hospitals. Zagazig Journal of Forensic Medicine 2020;18(2):105-21.
16. Martani L, Giovanniello A, Bosco G, Cantadori L, Calissi F, Furfaro D, et al. Delayed neurological sequelae successfully treated with adjuvant, prolonged hyperbaric oxygen therapy: review and case report. Int J Environ Res Public Health 2022;19(9):5300.
17. Zhang JH. Hyperbaric oxygen for neurological disorders. Flagstaff, Az: Best Publishing; 2008.
18. Ahn C, Oh J, Kim CW, Lee H, Lim TH, Kang H. Early neuroimaging and delayed neurological sequelae in carbon monoxide poisoning: a systematic review and meta-analysis. Sci Rep 2022;12(1):3529.
19. Jüttner B, Busch HJ, Callies A, Dormann H, Janisch T, Kaiser G, et al. S2k guideline diagnosis and treatment of carbon monoxide poisoning. Ger Med Sci 2021;19:Doc13.
20. Eichhorn L, Thudium M, Jüttner B. The diagnosis and treatment of carbon monoxide poisoning. Dtsch Arztebl Int 2018;115(51-52):863-70.

21. Chambers CA, Hopkins RO, Weaver LK, Key C. Cognitive and affective outcomes of more severe compared to less severe carbon monoxide poisoning. Brain Inj 2008;22(5):387-95.
22. Huang CC, Ho CH, Chen YC, Lin HJ, Hsu CC, Wang JJ, et al. Hyperbaric oxygen therapy is associated with lower short- and long-term mortality in patients with carbon monoxide poisoning. Chest 2017;152(5):943-53.
23. Otto CN. Hemoglobin metabolism. In: Keohane EM, Smith LJ, Walenga JM. Rodak's hematology: clinical, principle and applications. 6th ed. St.Louis: Elsevier; 2020. p. 91-103.
24. Rose JJ, Wang L, Xu Q, McTiernan CF, Shiva S, Tejero J, et al. Carbon monoxide poisoning: pathogenesis, management, and future directions of therapy. Am J Respir Crit Care Med 2017;195(5):596-606.
25. Guo D, Hu H, Pan S. Oligodendrocyte dysfunction and regeneration failure: a novel hypothesis of delayed encephalopathy after carbon monoxide poisoning. Medical Hypotheses 2020;136:109522.
26. Tsai PH, Chou MC, Chiang SW, Chung HW, Liu HS, Kao HW, et al. Early white matter injuries in patients with acute carbon monoxide intoxication: a tract-specific diffusion kurtosis imaging study and STROBE compliant article. Medicine 2017;96(5):e5982.
27. Nah S, Choi S, Lee SU, Kim GW, Lee YH, Han S. Effects of smoking on delayed neuropsychiatric sequelae in acute carbon monoxide poisoning: a prospective observational study. Medicine (Baltimore) 2021;100(20):e26032.
28. Han S, Nah S, Choi S, Kim GW, Lee YH. Optimal sessions of hyperbaric oxygen therapy in patients with carbon monoxide poisoning: a prospective observational study. Am J Emerg Med 2021;44:132-36.
29. Ouahmane Y, Hattimy FE, Soulaymani A, Mounach J, Satte A, Bourazza A, et al. Hyperbaric oxygen therapy in carbon monoxide poisoning in Moroccan patients. Acta Neurologica Belgica 2023;123(1):139-43.
30. Pepe G, Castelli M, Nazerian P, Vanni S. Delayed neuropsychological sequelae after carbon monoxide poisoning: predictive risk factors in the Emergency Department. A retrospective study. Scand J Trauma Resusc Emerg Med 2011;19:16.
31. Kim YS, Cha YS, Kim MS, Kim HJ, Lee YS, Youk H, et al. The usefulness of diffusion-weighted magnetic resonance imaging performed in the acute phase as an early predictor of delayed neuropsychiatric sequelae in acute carbon monoxide poisoning. Hum Exp Toxicol 2018;37(6):587-95.
32. Beppu T. The role of MR imaging in assessment of brain damage from carbon monoxide poisoning: a review of the literature. AJNR Am J Neuroradiol 2014;35(4):625-31.
33. Sert ET, Kokulu K, Mutlu H. Clinical predictors of delayed neurological sequelae in charcoal-burning carbon monoxide poisoning. Am J Emerg Med 2021;48:12-17.

34. Choi S, Nah S, Han S. Correlation between time to hyperbaric oxygen therapy and delayed neurological sequelae in acute carbon monoxide poisoning patients. Diagnostics (Basel) 2024;14(2):186.
35. Liao SC, Mao YC, Hung YM, Lee CH, Yang CC. Predictive role of QTc prolongation in carbon monoxide poisoning-related delayed neuropsychiatric sequelae. Biomed Res Int 2018;2018: 2543018.
36. Lee H, Kang H, Ko BS, Oh J, Lim TH, Cho Y. Initial creatine kinase level as a predictor for delayed neuropsychiatric sequelae associated with acute carbon monoxide poisoning. Am J Emerg Med 2021;43:195-9.
37. Lin CH, Su WH, Chen YC, Feng PH, Shen WC, Ong JR, et al. Treatment with normobaric or hyperbaric oxygen and its effect on neuropsychometric dysfunction after carbon monoxide poisoning: a systematic review and meta-analysis of randomized controlled trials. Medicine (Baltimore) 2018;97(39):e12456.38.
38. Nakajima M, Aso S, Matsui H, Fushimi K, Yasunaga H. Hyperbaric oxygen therapy and mortality from carbon monoxide poisoning: a nationwide observational study. Am J Emerg Med 2020;38 (2):225-30.
39. Han S, Nah S, Choi S, Lee YH, Kim GW, Cho YS. Hyperbaric oxygen therapy did not prevent delayed neuropsychiatric sequelae in a prospective observational study with propensity score matching in 224 patients with acute carbon monoxide toxicity. J Emerg Med 2021;60(4):498-505.
40. Weaver LK, Hopkins RO, Chan KJ, Churchill S, Elliott CG, Clemmer TP, et al. Hyperbaric oxygen for acute carbon monoxide poisoning. N Engl J Med 2002;347(14):1057-67.
41. Lee JS, Cha YS, Lim J. Association between number of hyperbaric oxygen therapy sessions and neurocognitive outcomes of acute carbon monoxide poisoning. Front Med (Lausanne) 2023;10:1127978.
42. Wang W, Cheng J, Zhang J, Wang K. Effect of hyperbaric oxygen on neurologic sequelae and all-cause mortality in patients with carbon monoxide poisoning: a meta-analysis of randomized controlled trials. Med Sci Monit 2019;25:7684-93.
43. Liao SC, Mao YC, Yang KJ, Wang KC, Wu LY, Yang CC. Targeting optimal time for hyperbaric oxygen therapy following carbon monoxide poisoning for prevention of delayed neuropsychiatric sequelae: A retrospective study. J Neurol Sci 2019;396:187-92.
44. Mathieu D, Marroni A, Kot J. Correction to Mathieu D, Marroni A, Kot J: Tenth European Consensus Conference on Hyperbaric Medicine: recommendations for accepted and non-accepted clinical indications and practice of hyperbaric oxygen treatment. Diving Hyperb Med 2017;47(1):24-32.

45. Liao SC, Shao SC, Yang KJ, Yang CC. Real-world effectiveness of hyperbaric oxygen therapy for delayed neuropsychiatric sequelae after carbon monoxide poisoning. Sci Rep 2021;11(1):19212.
46. Hu Q, Manaenko A, Xu T, Guo Z, Tang J, Zhang JH. Hyperbaric oxygen therapy for traumatic brain injury: bench-to-bedside. Med Gas Res 2016;6(2):102-10.
47. Wong ZK, Teo CK, Kwek JW, Kim SJ, See HG. Hyperbaric oxygen for the treatment of carbon monoxide-induced delayed neurological sequelae: a case report and review of the literature. Diving Hyperb Med 2024;54(1):65-8.
48. Tapeantong T, Poungvarin N. Delayed encephalopathy and cognitive sequelae after acute carbon monoxide poisoning: report of a case and review of the literature. J Med Assoc Thai 2009;92(10):1374-9.