Clinical Outcomes of Carbon Monoxide Poisoning Managed with Hyperbaric Oxygen Therapy in Oman

Hassan ALqartoobi[1], Badriya Al Farsi[1], Yousif AlBalushi[1], Darpanarayan Hazra[2] and Suad Abdullah Al Abri[2]*

[1]Diving and Hyperbaric Medicine Unit, Royal Navy of Oman, Medical City for Military and Security Services, Muscat, Oman

[2]Department of Emergency Medicine, Sultan Qaboos University Hospital, University Medical City, Muscat, Oman

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*Corresponding author: ghammari@squ.edu.om

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Abstract

Background: Carbon monoxide (CO) poisoning is a major cause of hypoxic injury with neurological and cardiac complications. Hyperbaric oxygen therapy (HBOT) is the standard treatment for moderate-to-severe poisoning. This study evaluated the clinical characteristics and outcomes of patients treated with HBOT in Oman.

Methods: This retrospective cohort study included patients treated between January 2015 and January 2025. Demographic, clinical, COHb, HBOT timing, treatment-session, and outcome data were analyzed.

Results: Of 45 referred patients, 32 were included. Mean age was 36.1 ± 12.4 years and 71.9% were male. Neurological and cardiac manifestations occurred in 84.3% and 15.6%, respectively. Mean COHb decreased from 30.82% to 1.721% after HBOT ($p < 0.001$). Patients received 2–7 sessions (mean 3.3 ± 1.5). At 24 hours, 40.6% were asymptomatic; by one week, 81.3% had fully recovered, while 18.8% had residual neurological symptoms at one month. Recovery was 100% with HBOT ≤6 hours versus 66.7% with treatment ≥12 hours ($p = 0.002$). Treatment delay was associated with poorer outcomes ($r = 0.52$, $p < 0.01$), while COHb showed a weaker association ($r = 0.34$, $p = 0.07$). Neurological and cardiac manifestations were associated with partial recovery, although the odds ratio was imprecise because of the small sample and sparse events.

Conclusion: HBOT was associated with 81% full recovery within one week. Earlier treatment was linked to better outcomes, while delays were associated with poorer recovery. Clinical presentation may provide greater prognostic value than COHb concentration alone, but larger prospective studies are needed to confirm this finding.

Keywords: Carbon monoxide poisoning; Hyperbaric oxygen therapy; HBOT; Delayed neurological sequelae; HBOT timing.

Introduction

Carbon monoxide (CO) poisoning remains a major cause of toxic morbidity and mortality worldwide, leading to neurocognitive and cardiac complications through hypoxia-induced cellular injury.[1-3] The global incidence varies by region and socioeconomic context, with higher prevalence in Europe, Asia, and the Middle East due to the use of gas heaters and poorly ventilated systems.[4,5] In the Middle East and North Africa (MENA), true incidence is likely underestimated because of underreporting, especially during colder seasons.[6,7] Delayed neurological sequelae (DNS), such as cognitive decline, behavioural changes, and movement disorders, further contribute to long-term disability among survivors.[8]

Hyperbaric oxygen therapy (HBOT) remains the cornerstone of moderate to severe CO poisoning management.[9-11] While protocol optimization continues to be debated, high-concentration oxygen is universally accepted as essential therapy.[10,11] HBOT rapidly displaces CO from hemoglobin, restores cellular respiration, and mitigates reperfusion injury through reduced lipid peroxidation and improved microcirculation.[10,11] Randomized controlled trials and meta-analyses confirm that HBOT reduces neurological deficits and mortality by inhibiting CO binding to mitochondrial cytochrome oxidase, restoring oxidative phosphorylation, and limiting oxidative stress.[12,13] Expert consensus recommends HBOT for patients with loss of consciousness, neurological impairment, myocardial ischemia, metabolic acidosis (pH <7.1), or pregnancy with COHb >20%.[9,10,12,14] The therapeutic benefit is greatest when initiated within six hours but remains beneficial up to 24 hours.[15-17] Treatment at 2.4–3.0 ATA for 60–90 minutes per session, guided by clinical response, is considered optimal.[18] Multi-session HBOT has been associated with lower mortality (≈7%) and morbidity (<5%) compared to single treatments.[19] The therapy is safe overall, including during pregnancy, where it accelerates fetal CO clearance and prevents hypoxic injury.[10,12,16,18,19]

In Oman and the wider Gulf region, the establishment of hyperbaric facilities represents a major advancement in critical care. This study presents a ten-year experience in the clinical application of HBOT for CO poisoning, contributing novel regional data on outcomes, complications, and evolving epidemiological trends. Although COHb is useful for confirming exposure, it may not accurately reflect poisoning severity, particularly when oxygen therapy has been administered before blood sampling. Clinical features such as loss of consciousness, altered mental status, myocardial involvement, exposure duration, and delay to HBOT may therefore provide important additional prognostic information. By integrating local findings with international evidence, this work aims to optimize HBOT protocols, enhance regional capacity, and strengthen evidence-based management of CO poisoning in Oman. Specifically, the study aimed to characterize clinical presentations and outcomes, evaluate HBOT selection and treatment patterns, and explore whether treatment timing, COHb level, and neurological or cardiovascular manifestations were associated with recovery.

Methods

Study Design, Settings and Duration: This retrospective cohort study analyzed all patient records with confirmed CO poisoning treated at the Royal Navy of Oman Hyperbaric Medicine Unit (HMU) between January 2015 and January 2025.

Study Population: All patients with suspected or confirmed CO poisoning referred to the Royal Navy of Oman Hyperbaric Medicine Unit during the 10-year study period were screened. Inclusion criteria were[1]: documented or strongly suspected CO exposure[2]; treatment with HBOT[3]; available pre- and post-HBOT COHb measurements[4]; available HBOT timing and treatment-session data; and[5] documented immediate and follow-up clinical outcome. Exclusion criteria were[1]: missing pre- or post-HBOT COHb values[2]; missing HBOT timing or session data[3]; absent follow-up outcome documentation; or[4] incomplete records that prevented outcome classification. Of 45 referred patients, 32 met inclusion criteria and 13 were excluded because of incomplete data.

Variables: Data collection was conducted from both electronic and paper-based medical records using a structured data abstraction form. The data were categorized into four primary domains. The first domain included demographic characteristics such as age, sex, and referral source. The second domain focused on clinical parameters including presenting symptoms, pre- and post-treatment carboxyhemoglobin (COHb) levels, and the time interval between CO exposure and initiation of HBOT. The third domain captured treatment details such as the number of HBOT sessions administered, oxygen pressure settings, session duration, and any concurrent medical interventions provided. The final domain comprised clinical outcomes, including the resolution of symptoms and COHb clearance measured at 24 hours post-HBOT, as well as at one-week and one-month follow-up visits.

Hyperbaric Oxygen Therapy Protocol: The HBOT regimen followed standardized Royal Navy treatment tables, selected according to poisoning severity, COHb clearance rate, and clinical response. All patients received 100% oxygen at pressures of 2.4–2.8 atmospheres absolute (ATA) for 60–135 minutes per session. The number and frequency of sessions were individualized based on clinical progress, COHb clearance, and symptom severity. Supplementary Figures 1–3 depict the HBOT tables employed for mild, moderate, and severe CO poisoning cases. These structured regimens ensured effective CO elimination, optimized tissue oxygenation, minimized oxygen toxicity, and enhanced neurological recovery.

Outcome Definitions: Operational definitions of outcome: "Full recovery" was defined as complete resolution of presenting symptoms with no documented residual neurological, cognitive, cardiac, or systemic complaints at follow-up. "Improved recovery" was defined as substantial symptom improvement with no major functional limitation but with minor residual symptoms such as dizziness, fatigue, or lethargy. "Partial recovery" was defined as persistent neurological or functional symptoms, including unsteady gait, drowsiness, lethargy, or residual cognitive/neurological complaints at follow-up. No standardized neurocognitive test battery was applied; outcomes were based on treating-clinician documentation in the medical record. This limitation is acknowledged in the Discussion.

Statistical Analysis: Statistical analyses were performed using IBM SPSS Statistics version 29.0. Continuous variables were summarized as mean ± standard deviation, and categorical variables as frequencies and percentages. Pre- and post-HBOT COHb levels were compared using paired t-tests. Associations between categorical variables were assessed using chi-square or Fisher's exact tests, as appropriate, while exploratory relationships involving continuous variables and recovery outcome were evaluated using Pearson or point-biserial correlation. For exploratory binary analyses, full and improved recovery were grouped as favorable recovery and compared with partial recovery. Univariable logistic regression was used to report unadjusted odds ratios (ORs) with 95% confidence intervals and p-values. Multivariable logistic regression was considered but not performed because only six patients experienced partial recovery, which was insufficient to support a stable adjusted model. All analyses were exploratory, and a two-sided p-value <0.05 was considered statistically significant.

Ethical Approval: The study protocol was reviewed and approved by the Medical City for Military and Security Services Medical Research Ethics Committee (MCMSS-MREC-052/2025) on 27 March 2025. Given the retrospective observational chart review design, the requirement for informed consent was waived by the ethics committee.

Results

The study cohort included 32 patients diagnosed with CO poisoning, with a mean age of 36.1 ± 12.4 years (range 17–67). A male predominance was observed, representing 71.9% (n = 23) of the total cohort. Notably, 34.4% of the cases (11/32) were from a single academic hospital, suggesting a potential referral concentration pattern. (Table 1).

Table 1: Demographic Characteristics, Referral Centers, and Presenting Symptoms of Patients Undergoing Hyperbaric Oxygen Therapy (HBOT)

Variables	Frequency (%)
Age (Mean ± Standard Deviation)	36.1 ± 12.4 years
Age range	17–67 years
Males	23 (71.9)
Females	9 (28.1)
Referral Centres	
Sultan Qaboos University Hospital, University Medical City	11 (34.4)
Suhar Hospital, Al Batinah North Governorate	7 (21.8)
The Royal Hospital, Muscat	5 (15.7)
Armed Forces Hospital, Muscat	5 (15.7)
Rustaq Hospital, Al Batinah South Governorate	2 (6.2)
Private Hospital, Muscat	2 (6.2)
Presenting Symptoms	
Neurological Symptoms	27 (84.3)
Loss of consciousness (LOC), altered mental status	20 (62.5)
Dizziness	19 (59.4)
Headache	17 (53.1)
Cardiac symptoms (chest pain, arrhythmia)	5 (15.6)
Gastrointestinal or nonspecific complains	5 (15.6)

The majority of patients presented with central nervous system (CNS) involvement, including loss of consciousness (LOC), altered mental status (AMS), or cognitive impairment. Loss of consciousness/altered mental status (20/32; 62.5%), dizziness (19/32; 59.4%), and headache (17/32; 53.1%) were the most common presenting symptoms. CNS manifestations were present in 84.3% (27/32) of patients, while cardiac symptoms,

including chest pain or arrhythmias, were present in 15.6% (5/32) (Table 1). The remainder exhibited gastrointestinal or nonspecific symptoms. Baseline COHb levels of ≥20% were recorded in 26 (81.3%) of cases, with a mean pre-HBOT COHb concentration of 30.82 ± 9.84%. (Table 2) Intubated patients (12.5%, n = 4) had higher baseline COHb levels (34.9%) compared to non-intubated patients (31.1%), although this difference was not statistically significant.

Table 2: Distribution of Carboxyhemoglobin (COHb) Level (%) Categories Among Patients.

Variable	Frequency (n = 32)	Percentage (%)
COHb Categories (%)		
>50	2	6.2
40 - <50	7	21.9
30 - <40	7	21.9
20 - <30	10	31.2
10 - <20	6	18.8

All patients received multiple HBOT sessions following clinical indications and established hyperbaric protocols. Nearly thirty-five percent (11/32) of patients underwent two sessions, while 65.6% (21/32) received three or more. The mean number of sessions was 3.3 ± 1.5 (range 2–7). HBOT was associated with a marked reduction in COHb levels, from 30.82 ± 9.84% before treatment to 1.721 ± 0.99% after treatment ($p < 0.001$) (Figure 1). Early initiation of HBOT was significantly associated with full recovery compared to delayed therapy ($p = 0.002$) (Table 3, Figure 2). The average time to HBOT initiation was 12.6 (3–48) hours. Only 12.5% (n = 4) received therapy within the optimal six-hour window. Early treatment (≤6 hours) was associated with full recovery in 4/4 patients (100%), compared with 6/9 patients (66.7%) treated after ≥12 hours ($p = 0.002$). (Table 3, Figure 2). Early-treated patients also recovered faster (mean 2.1 days vs. 4.3 days, $p = 0.02$), highlighting treatment delay as a significant prognostic factor (Figure 2). At 24 hours, 13 patients (40.6%) were asymptomatic, while the remaining patients had one or more residual symptoms (Table 4). At one-month follow-up, 18.8% (6/32) exhibited partial recovery. Partial recovery was associated with a delay to HBOT of ≥12 hours ($p = 0.04$) and a baseline COHb level of ≥30% ($p = 0.03$). Patients presenting with both neurological and cardiac manifestations required significantly more HBOT sessions (mean 5.2 vs. 2.7, $p = 0.01$) and showed slower recovery trajectories.

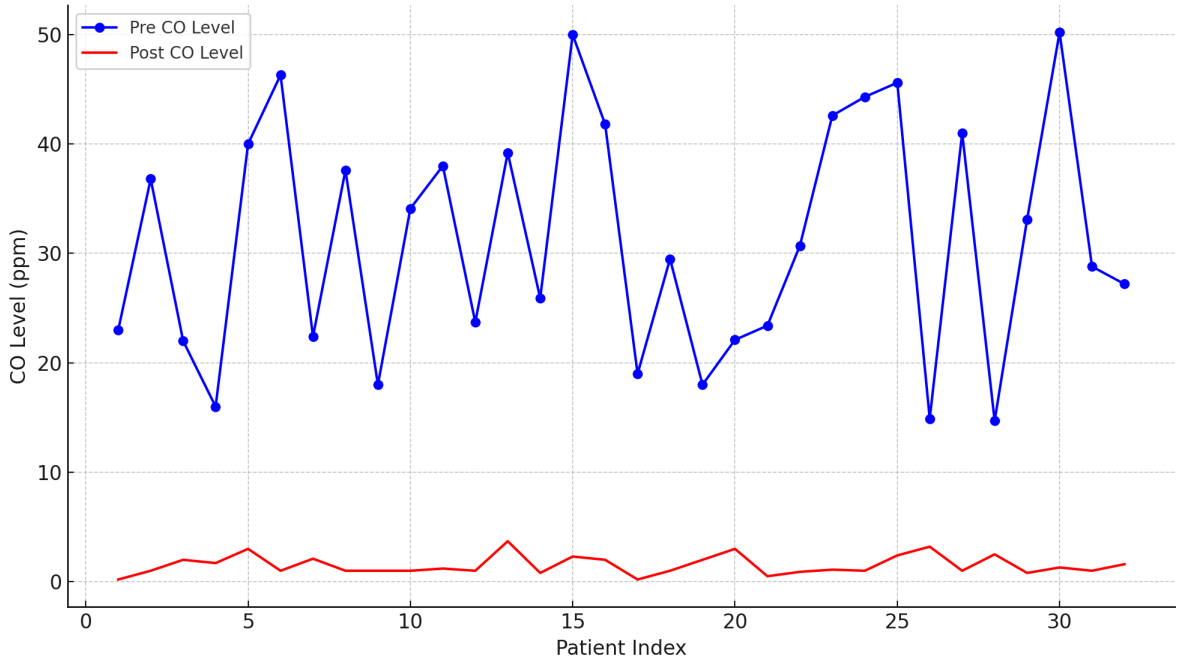

Figure 1: Reduction in Carboxyhemoglobin Levels Following Hyperbaric Oxygen Therapy. *Mean COHb decreased from 30.82% before HBOT to 1.721% after treatment ($p < 0.001$), demonstrating a significant biochemical reduction following HBOT.

Table 3: Hyperbaric Oxygen Therapy (HBOT) Parameters and Outcomes.

Parameter	Value

Pre -HBOT* Carboxyhemoglobin Level (%)	mean ± SD: 30.82 ± 9.84, Median (range) 27.00 (11.00–53.2)
Mean HBOT sessions ± SD	mean ± SD: 3.3 ± 1.5 (range 2–7)
Post -HBOT Carboxyhemoglobin Level (%)	mean ± SD: 1.721 ± 0.99, Median (range) 1.45 (0.2–3.7)
Statistical significance (pre-HBOT vs. post HBOT)	*p < 0.001*
Patients with 2 sessions	11 (34.4%)
Patients with ≥3 sessions	21 (65.6%)
Patients treated within 6 hours	4 (12.5%)
Mean time to HBOT initiation (hours)	12.6 (range 3–48)
Recovery rate (≤6 h treatment)	100% full recovery
Recovery rate (≥12 h treatment)	66.7% full recovery
Statistical significance (early vs. delayed)	$p = 0.002$

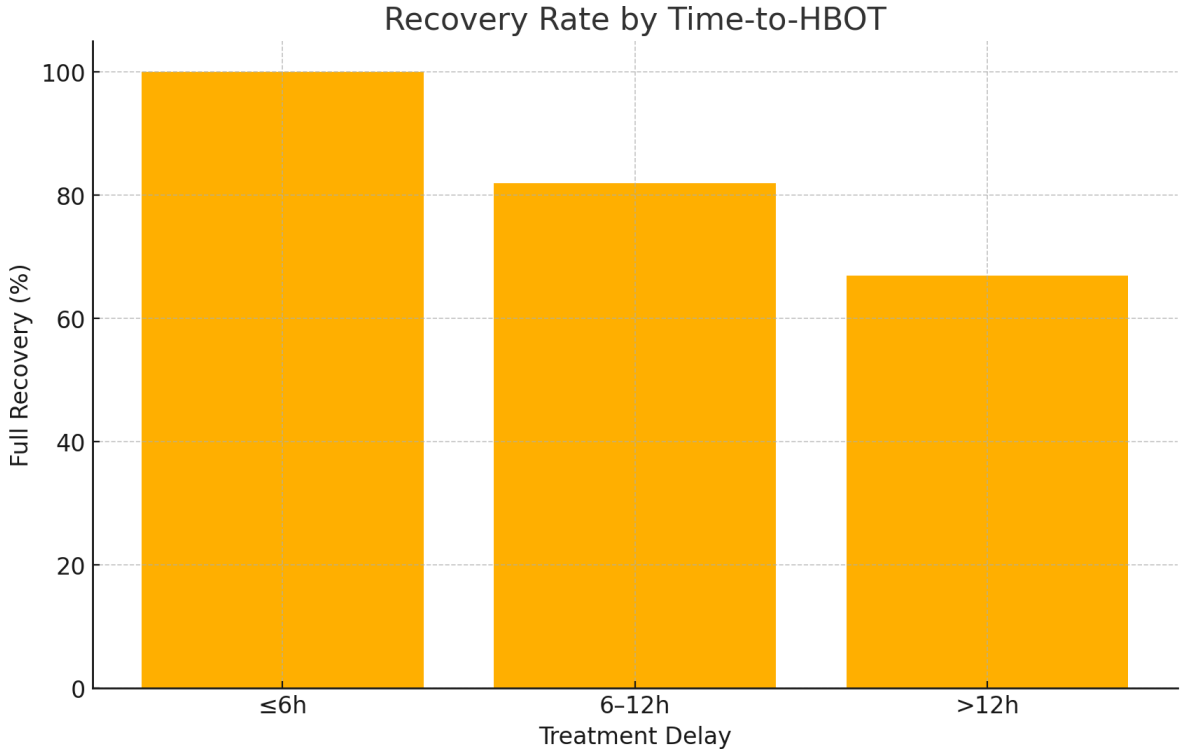

Figure 2: Influence of Time-to-Hyperbaric Oxygen Therapy on Clinical Recovery in CO Poisoning.

Table 4: Immediate Clinical Outcomes Following Hyperbaric Oxygen Therapy (HBOT).

Variables	Frequency (%)
Asymptomatic	13 (40.6)
Lethargy	10 (31.2)
Unsteady Gait	9 (28.1)
Drowsiness/ Dizziness	7 (21.9)
Headache	2 (6.2)
Chest Pain	1 (3.1)
Fatigued	2 (6.2)
Nausea	2 (6.2)

Correlation analysis identified neurological and/or cardiac involvement as the strongest predictor of incomplete recovery (r = 0.41, p = 0.03), with an unadjusted odds ratio (uOR) of approximately 33.1, 95% confidence interval (CI) 2.11-319.60. (Table 5). Although neurological ± cardiac symptoms demonstrated a high odds ratio, the corresponding confidence interval was wide, reflecting substantial statistical uncertainty related

to the small sample size and limited number of outcome events. While higher COHb levels (≥25%) showed a weaker association with partial recovery (r = 0.34, uOR 1.08 per 1% increase (95% CI 0.99–1.18; p = 0.07), neurological and cardiac manifestations demonstrated a stronger association with outcome. Patients with shorter pre-HBOT delays (≤6 hours) and fewer treatment sessions (<3) demonstrated higher rates of full recovery. Mean pre-HBOT delay for fully recovered patients was 9.6 ± 4.2 hours, compared to 14.2 ± 5.8 hours for those with partial improvement (r = 0.52, p < 0.01). Similarly, patients requiring more HBOT sessions (3.9 ± 1.5) had slower recovery than those needing fewer (2.8 ± 1.1; r = 0.41, p = 0.02).

Table 5: Exploratory univariable associations with incomplete recovery

Predictor	Correlation coefficient (r)	Unadjusted Odds ratio (OR)	95% CI	p-value
Neurological ± Cardiac symptoms	0.41	≈ 33.10	2.11–319.60	0.03
Carboxyhemoglobin level (%) ≥25	0.34	1.08 per 1% increase	0.99–1.18	0.07
Pre-HBOT delay (hours)	0.52	—	—	<0.01
HBOT sessions (number)	0.41	—	—	0.02

Footnote: ORs were calculated using univariable logistic regression. Confidence intervals are wide because of the limited sample size (n = 32) and the small number of patients with partial recovery (n = 6). Therefore, these estimates should be interpreted cautiously, as the magnitude of the odds ratios may be overestimated because of sparse data.

Discussion

This ten-year Omani cohort provides regional evidence on the use of HBOT for severe CO poisoning. Overall, 81.3% of patients achieved full recovery within one week of HBOT, while 18.8% had partial recovery at one month. These findings support the clinical relevance of HBOT, particularly when treatment is initiated promptly and guided by neurological and cardiovascular manifestations rather than COHb concentration alone. However, the observational design does not establish a causal treatment effect, and the findings should be interpreted within the context of the small, single-center cohort.

Treatment timing was strongly associated with recovery. All patients treated within six hours of exposure achieved complete recovery, compared with 66.7% of those treated after 12 hours (p = 0.002). The mean time to HBOT was 12.6 hours, and longer delay was associated with an increased likelihood of partial recovery. This finding is consistent with previous reports identifying time to treatment as an important prognostic factor.[15-17] Early HBOT may limit ischemia–reperfusion injury, oxidative stress, and the progression of hypoxic neurological damage, whereas its therapeutic benefit may diminish as treatment is delayed.[10,12,18,19] Evidence regarding the neurological benefits of HBOT remains mixed. Several randomized controlled trials and systematic reviews have reported a reduction in delayed neurological sequelae when HBOT is administered during the early post-exposure period.[10,16,20] In the double-blind randomized trial by Weaver et al. (2002), three HBOT sessions within 24 hours reduced cognitive deficits at 6 weeks and 12 months compared with normobaric oxygen.[21] Similarly, the meta-analysis by Wang et al. (2019) reported a lower risk of delayed neuropsychiatric manifestations, including memory and attention impairment, among HBOT-treated patients.[22] In the present study, 84.3% of patients presented with central nervous system involvement, including loss of consciousness, altered mental status, or cognitive impairment, and 92% of these patients achieved full recovery when HBOT was administered within 12 hours. These observations support early consideration of HBOT in patients with significant neurological manifestations.[8,15,16] Nevertheless, heterogeneity in patient selection, treatment timing, chamber pressure, number of sessions, outcome definitions, and follow-up has prevented universal agreement regarding optimal HBOT protocols. The Cochrane reviews by Buckley et al. (2011) and Juurlink et al. (2005) concluded that HBOT may offer neurological benefit, but the available evidence was insufficiently consistent to

support firm conclusions.[11,12] More recently, the network meta-analysis by Ho et al. (2022) found that multiple-session protocols did not consistently improve outcomes compared with a single session.[23] In the present cohort, patients receiving four or more sessions had slower recovery, despite a mean of 3.3 ± 1.5 sessions overall. This likely reflects greater initial disease severity and persistent neurological symptoms rather than an adverse effect or inefficacy of additional HBOT sessions. Accordingly, the number of sessions should be individualized according to clinical response rather than interpreted as an independent determinant of poor outcome.

The association between pre-HBOT delay and partial recovery was among the strongest observed in this study ($r = 0.52$, $p < 0.01$). Neurological and cardiac manifestations were also associated with poorer recovery ($r = 0.41$, $p = 0.03$; reported OR ≈33.1), whereas COHb concentration showed a weaker and statistically non-significant correlation with outcome ($r = 0.34$, $p = 0.07$). The apparently large odds ratio for neurological ± cardiac symptoms should be interpreted cautiously because the small number of partial-recovery events and sparse contingency-table cells may have substantially inflated the estimate and produced limited precision. Furthermore, because a statistically reliable multivariable model could not be constructed, this odds ratio represents an exploratory, unadjusted association and does not demonstrate that these symptoms independently predicted recovery. These results are broadly consistent with reports by Casillas et al. (2019) and Annane et al. (2011), which emphasized that neurological involvement and overall clinical severity may be more informative for prognosis and HBOT selection than COHb concentration alone.[18,24] COHb remains valuable for confirming exposure, but it may correlate poorly with tissue injury, symptom severity, and subsequent neurological outcome, particularly when oxygen has been administered before blood sampling. Other studies, however, have found that COHb may still contribute to severity assessment when considered alongside lactate, neurological findings, cardiovascular injury, and exposure history. Therefore, the present findings should not be interpreted as showing that COHb has no prognostic value; rather, COHb should not be used in isolation, and clinical presentation should remain central to risk stratification.

HBOT produced a marked biochemical response, reducing mean COHb from 30.82 ±9.84% before HBOT to 1.721 ±0.99% after treatment ($p < 0.001$), consistent with the established ability of hyperbaric oxygen to accelerate CO elimination and improve tissue oxygenation.[9,12] A baseline COHb level of ≥30% was associated with partial recovery, suggesting that substantial exposure may still identify patients at increased risk. However, the timing of the recorded COHb measurements in relation to pre-hospital or emergency oxygen therapy was not consistently documented. Because supplemental oxygen rapidly reduces COHb, the recorded values may have underestimated peak exposure and weakened the observed relationship between COHb and outcome. Mechanistically, the observed clinical and biochemical improvements may reflect restoration of mitochondrial oxidative phosphorylation, reduced leukocyte adhesion, and attenuation of lipid peroxidation, as described by Garrabou et al. (2011).[25]

Although multivariable regression was considered to identify independent predictors of partial recovery, only six patients experienced this outcome. This was insufficient to support a statistically reliable adjusted model without substantial risk of overfitting. Consequently, the reported associations and odds ratios are unadjusted and exploratory and may be influenced by confounding variables, including poisoning severity, treatment delay, referral pathway, pre-HBOT oxygen administration, and the clinical indication for repeated HBOT sessions. Only four patients (12.5%) received HBOT within 6 hours, indicating that most patients experienced delayed treatment. Potential barriers include delayed recognition of CO poisoning, initial stabilization at referring institutions, inter-hospital referral processes, transport logistics, and limited access to hyperbaric facilities. Patients treated later may have differed systematically in poisoning severity and referral characteristics, introducing potential selection bias. Because the early-treatment group included only four patients, meaningful adjusted comparison between early- and late-treated patients was not feasible. The apparent benefit of early treatment should therefore be interpreted cautiously.

Despite variation in international treatment regimens, protocols using 2.4–3.0 atmospheres absolute for 60–90 minutes per session are commonly applied. The Royal Navy Treatment Tables 61 and 14:90:30 used in this cohort produced outcomes comparable with those described using the Weaver and Lin protocols, suggesting that these regimens may be suitable for regional practice.[21,26] However, direct protocol comparisons were not performed, and their relative effectiveness cannot be established from this study. An individualized approach, in which additional sessions were administered according to persistent neurological symptoms, appeared clinically practical, particularly in a setting where treatment decisions must account for available resources, referral delays, and patient severity.

Finally, the findings should be interpreted in light of the small cohort of 32 patients and the limited number of partial-recovery events. These factors reduced statistical power, increased uncertainty around effect estimates, and restricted both subgroup comparisons and multivariable analysis. Therefore, statistically non-significant findings should not be regarded as definitive evidence of no association, while statistically significant findings with large effect estimates should be interpreted cautiously. Larger prospective multicenter studies incorporating standardized neurological and cognitive assessments, accurately timed COHb measurements, exposure duration and source, comorbidities, and detailed pre-hospital treatment data are needed to validate these findings and clarify independent predictors of recovery.

Limitation: Its retrospective, single-center design and small sample size limited statistical precision, restricted the feasibility of multivariable analysis, and reduced the generalizability of the findings. The timing of COHb measurement in relation to pre-hospital or emergency oxygen therapy was not consistently documented; therefore, recorded COHb levels may have underestimated the severity of the initial exposure. In addition, only a small proportion of patients received HBOT within 6 hours, limiting meaningful comparison between early- and late-treated groups and introducing potential selection bias. Incomplete documentation of some clinical variables may also have introduced information bias.

Recommendations: Based on our findings and corroborating international evidence, we recommend establishing a national CO poisoning management guideline emphasizing early initiation of HBOT (≤6 hours post-exposure), symptom-based patient selection, and standardized treatment protocols using 100% oxygen at 2.5–3.0 ATA for 60–90 minutes per session. Strengthening referral networks, expanding hyperbaric capacity, and training multidisciplinary teams will be essential to improve outcomes and reduce long-term neurological sequelae across Oman and the broader MENA region. These findings support efforts to improve timely, clinically guided referral for HBOT while awaiting confirmation from larger prospective multicenter studies.

Conclusion

This ten-year retrospective study provides preliminary regional evidence that HBOT was associated with substantial reductions in COHb levels and generally favorable short-term clinical outcomes among patients with carbon monoxide poisoning in Oman. Earlier treatment and greater clinical severity appeared to be associated with recovery, while clinical presentation may provide prognostic information beyond COHb concentration alone. However, these findings should be interpreted cautiously because of the small sample size, limited number of adverse outcome events, retrospective single-center design, and uncertainty regarding the timing of COHb measurement relative to oxygen administration. Larger prospective multicenter studies are needed to confirm these associations and clarify the independent predictors of recovery.

Disclosure

There are no conflicts of interest.

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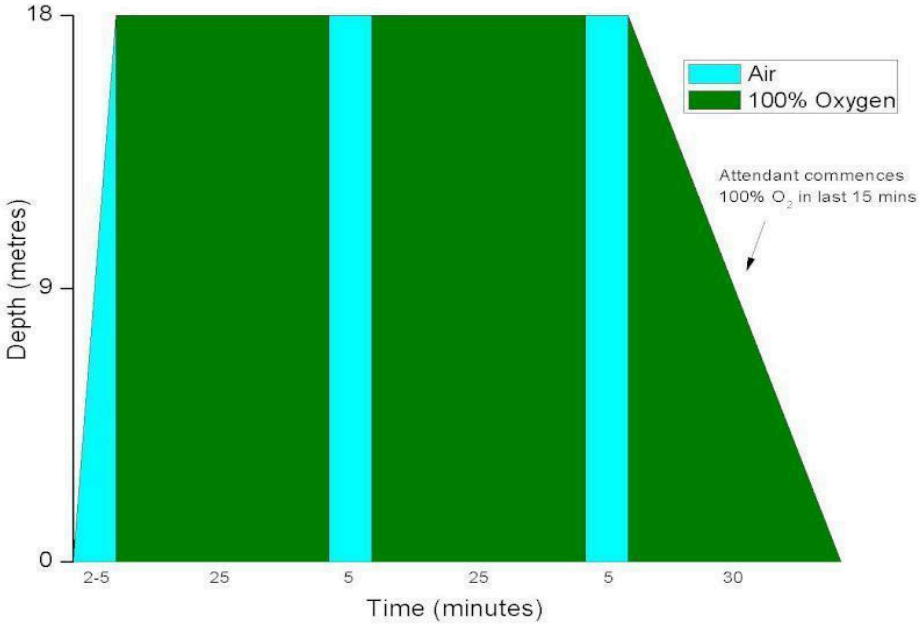
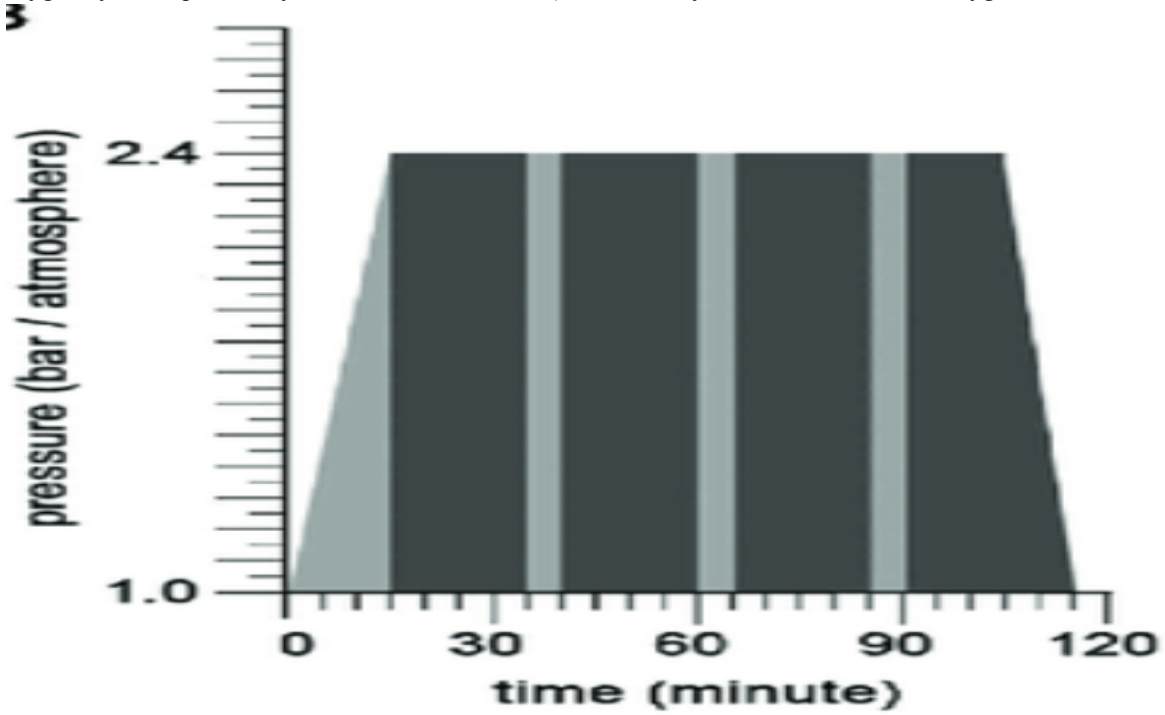
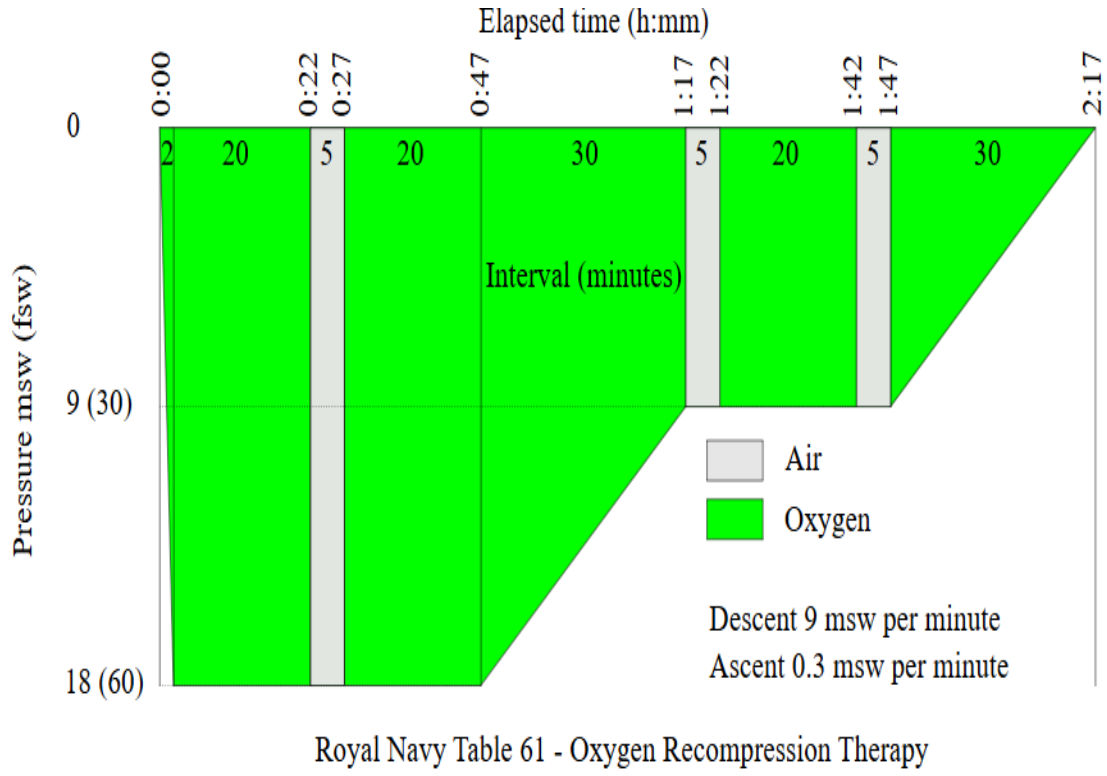


Supplementary Figure 1: Royal Navy Table 0 – Mild to moderate CO poisoning *or* follow-up treatment. RN Table 60: Used for mild to moderate or follow-up therapy; oxygen at 2.8 ATA for 60 minutes (two 25-minute oxygen cycles separated by two 5-minute air breaks), followed by 30 minutes ascent on oxygen.



Supplementary Figure 2: Royal Navy Table 14 – *Moderate CO poisoning* with extended oxygen exposure. RN Table 14: Used for moderate cases; oxygen at 2.4 ATA for 90 minutes (three 25-minute oxygen cycles separated by three 5-minute air breaks), followed by 30 minutes ascent on oxygen.



Supplementary Figure 3: Royal Navy Table 61 – Severe CO poisoning *(oxygen recompression therapy)*. RN Table 61: Used for severe CO poisoning; treatment at 2.8–1.9 ATA for 105 minutes, including three 5-minute air breaks, followed by 30 minutes ascent on oxygen (total 135 minutes). Repeatable after 6–12 hours if symptoms persist.