ORIGINAL ARTICLE

Acute Carbon Monoxide Poisoning in the Emergency Department: A Descriptive Study of 660 Cases in Tunisia

Intoxication aiguë au monoxyde de carbone aux urgences: Etude descriptive de 660 cas en Tunisie

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Abstract

Introduction: Carbon monoxide (CO) poisoning is a frequent and potentially fatal medical emergency, accounting for significant global morbidity and mortality.

Aim: This study aims to determine the epidemiological and clinical characteristics of patients presenting to the emergency department (ED) with acute CO poisoning in Tunisia.

Methods: A retrospective, descriptive, single-center study was conducted at the ED of Mahmoud Yacoub Center for Urgent Medical Assistance in Tunis over two years. Patients aged over 12 years with acute CO poisoning were included. Epidemiological data, clinical presentation, severity, laboratory findings, and management were analyzed.

Results: A total of 660 cases were included, all secondary to accidental exposure. The mean age was 35 ±15 years, with a female predominance (73%). CO poisoning was most frequent during winter (70.4%). The main source was gas water heaters (78.2%). Severe cases of CO poisoning were observed in 27.9% of patients, with neurological symptoms in 90.8% of them. The median carboxyhemoglobin level was 21% [13–29]. In univariate analysis, age ≥50, asthenia, chest pain, carboxyhemoglobin ≥25%, and hyperleukocytosis were associated with severe CO poisoning, with hyperleukocytosis remaining an independent predictor in multivariate analysis. Oxygen therapy was administered to all patients, and hyperbaric oxygen therapy was performed in 10.6%. Hospitalization was required in 21% cases. No deaths were recorded in our series.

Conclusion: CO poisoning remains a significant public health concern in Tunisia, predominantly affecting young adults, with water heaters as the leading source. Neurological and cardiac manifestations predominate in severe cases.

Key words: Emergency department, Carbon monoxide, poisoning, toxicology, hyperbaric oxygen therapy

Résumé

Introduction: L'intoxication au monoxyde de carbone (CO) est une urgence médicale fréquente et potentiellement mortelle, responsable d'une morbidité et d'une mortalité significatives dans le monde.

Objectif: Décrire les caractéristiques épidémiologiques et cliniques des patients consultant pour une intoxication aiguë au CO en Tunisie.

Méthode: Une étude rétrospective, descriptive et monocentrique a été menée aux urgences du Centre Mahmoud Yacoub sur deux ans. Les patients de plus de 12 ans présentant une intoxication aiguë au CO ont été inclus. Les données épidémiologiques, cliniques, biologiques et thérapeutiques ainsi que la gravité ont été analysées.

Résultats: Un total de 660 cas (438 femmes) a été inclus. L'âge moyen était de 35±15 ans. L'intoxication était plus fréquente en hiver (70,4%). La principale source de CO était les chauffe-eaux à gaz (78,2%). Des formes sévères ont été observées chez 27,9% des patients, avec des manifestations neurologiques dans 90,8% des cas. Le taux médian de carboxyhémoglobine était de 21%.En analyse univariée, un âge ≥50 ans, l'asthénie, les douleurs thoraciques, un taux de carboxyhémoglobine ≥25% et l'hyperleucocytose étaient associés à une intoxication sévère, cette dernière restant un facteur prédictif indépendant en analyse multivariée. L'oxygénothérapie a été administrée à tous les patients, et l'oxygénothérapie hyperbare réalisée chez 10,6%. Une hospitalisation a été nécessaire dans 21% des cas. Aucun décès n'a été rapporté.

Conclusion: L'intoxication au CO demeure un problème majeur de santé publique en Tunisie, touchant principalement les jeunes adultes, avec les chauffe-eaux comme principale source. Les atteintes neurologiques et cardiaques prédominent dans les cas graves.

Mots-clés: Service des urgences, monoxyde de carbone, intoxication, toxicologie, oxygénothérapie hyperbare

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Introduction

Carbon monoxide (CO) poisoning is a common and potentially fatal medical emergency, responsible for significant global morbidity and mortality [1,2]. Known as the "silent killer" due to its colorless, odorless, and non-irritating nature, CO is one of the leading causes of accidental poisoning worldwide. It primarily occurs in poorly ventilated domestic environments, often linked to heating or water-heating appliances, and certain industrial settings [3].

Although many cases remain underdiagnosed, the global incidence of CO poisoning is on the rise [1]. In Tunisia, CO poisoning is particularly concerning during the winter season, with a high incidence that has remained unchanged since 2004, despite public awareness campaigns [4]. Emergency departments (EDs) frequently face CO poisoning cases, with peaks of incidents leading to patient surges and increased pressure on medical resources [4].

The toxicity of CO is primarily due to its high affinity for hemoglobin, forming carboxyhemoglobin (COHb), which reduces oxygen delivery to tissues and causes hypoxia [5]. This hypoxia affects oxygen-sensitive organs, such as the brain and heart, resulting in neurological and cardiovascular symptoms frequently seen in severe cases [6]. Clinical manifestations range from mild symptoms, such as headaches and dizziness, to severe outcomes, including coma and cardiac injuries, reflecting its complex pathophysiology [6,7].

To the author's knowledge, relevant data regarding CO poisoning in a Tunisian population is scarce.

The aim of this study was to determine the epidemiological and clinical characteristics of patients consulting the ED for acute CO poisoning.

Methods

This was a retrospective, descriptive, single-center study conducted in the ED of Mahmoud Yacoub Center for Urgent Medical Assistance in Tunis over a two-year period, from June 1st 2021 to June 30th 2023.

Study population

Our study included patients aged over 12 years presenting with acute CO poisoning. The diagnosis was based on multiple criteria, including anamnesis revealing exposure to a CO source, suggestive clinical signs, and COHb levels exceeding 3% in non-smokers and 6% in smokers. We excluded chronic exposure to CO, CO poisoning associated with other toxic gases, as well as incomplete medical records.

Data collection

For data collection, we recorded epidemiological characteristics and comorbidities. Information about symptoms and the circumstances of intoxication was primarily obtained from patient self-reports. For unconscious patients, this information was provided by relatives. We assessed the initial clinical presentation, including respiratory rate, SpO2, heart rate, blood pressure, ECG findings, and neurological status using the Glasgow Coma Scale (GCS). Severity was defined as the presence of neurological and or cardio vascular symptoms. Laboratory findings, including COHb levels, were measured using the GEM OPL CO-Oximeter. We also noted medical interventions in the ED, such as oxygen therapy administered via a high-concentration mask. Hyperbaric oxygen therapy (HBO) was indicated in cases of neurological, cardiovascular manifestations and or COHb levels exceeding 25%. Symptomatic treatment was provided whenever required by the patient's condition. Outcomes such as length of stay, the need for hyperbaric oxygen therapy or mechanical ventilation, and ICU admission were also documented.

Statistics

Data entry and statistical analysis were performed using SPSS (Statistical Package for the Social Sciences), version 23. Descriptive analysis involved calculating means, medians, and standard deviations, as well as determining the minimum and maximum values for quantitative variables. Absolute and relative frequencies, expressed as percentages, were calculated for qualitative variables. We analyzed factors associated with severe CO poisoning using univariate and multivariate methods. Categorical variables were compared using the chi-square test or Fisher's exact test when appropriate, while continuous variables were analyzed with the student's t-test or Mann-Whitney U test, depending on data distribution. Univariate logistic regression was performed to estimate odds ratios (OR) and 95% confidence intervals (CI). Significant variables were then included in a multivariate logistic regression model using a stepwise backward selection method. For all statistical tests, the significance threshold was set at $p \leq 0.05$.

Results

A total of 660 cases of acute CO poisoning were included in the study (figure1), all of them were secondary to accidental exposure.

The clinical and laboratory findings of the study population are presented in Table 1. Our population had a sex ratio of 0.36, with a mean age of 35 ± 15 years. Notably, 75% of the individuals had no pre-existing medical conditions. Among those with comorbidities, thyroid dysfunction (6.5%), hypertension (5.9%), and diabetes (5.6%) were the most common. At the time of the study, 20% of patients were identified as active smokers.

The seasonal distribution of CO poisonings demonstrated a significant peak during the coldest months (December to February), accounting for 70.4% of cases (Figure 2). Furthermore, consultations were most frequent on Sundays (21% of cases, n = 140), with weekends collectively comprising 51% of all consultations.

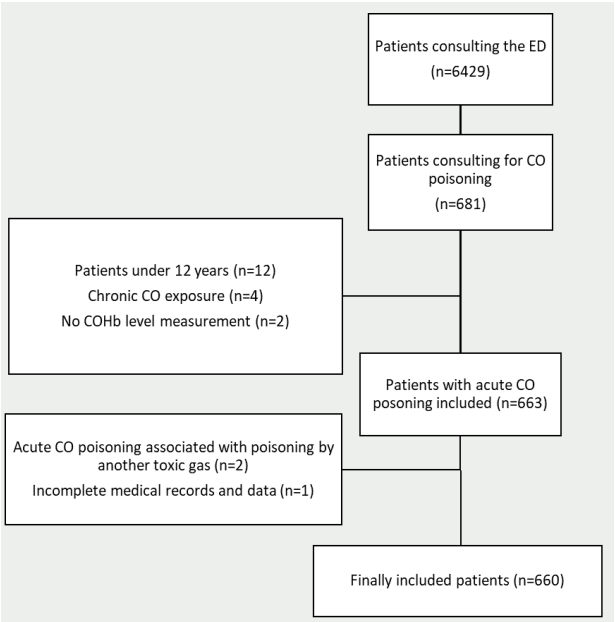

Figure 1. Flowchart of patients of our study

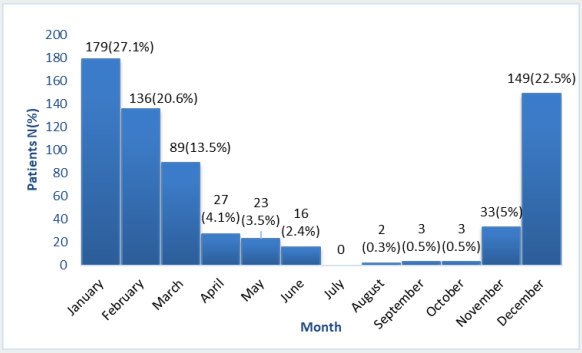

Figure 2. Carbon monoxide poisoning distributed by month

Collective intoxications were observed in 60% of cases,with a median of 2 [1-2] victims per incident and a maximum of seven. The primary sources of CO poisoning was water heaters (78.2%) with a median exposure duration of 2 [1- 4] hours. Exposure to multiple sources simultaneously was present in 2% of the cases.

Transportation to the ED was predominantly via non-medical means (70%, n = 460), while 30% were transported by medical services. Fourteen percent of patients had received oxygen therapy en route to the ED. The median consultation time after CO exposure was 1 hour [1–2].

The most prevalent symptom was headache (85%), followed by loss of consciousness (16%) and altered mental status (11%). Upon arrival, 12 patients presented with a GCS of 14, while seven were comatose (GCS ≤ 8). All patients exhibited intermediate and reactive pupils, with no focal neurological deficits or hemodynamic instability noted during the initial examination.

Electrocardiographic abnormalities were identified in 13% of patients, including conduction disorders (2.5%), rhythm disturbances (5.5%), and repolarization abnormalities (5%). Among patients tested for troponin levels, 14% demonstrated elevations, with 50% diagnosed with acute coronary syndrome and the other 50% classified as having myocardial injury.

Table 1. Clinical and laboratory findings in the study population

	All patients (n=660)	
Age (years)	35 ± 15	
Female sex n (%)	484 (73.3)	
Comorbidities n (%)		
Thyroid dysfunction	43(6.5)	
Hypertension	39(5.9)	
Diabetes	37(5.6)	
Asthma	13 (2)	
COPD	12 (1.8)	
Anemia	12 (1.8)	
Other	69 (10.4)	
Sources of CO n(%)		
Moorish bath	6 (0.9)	
Home	654 (99)	
Water heaters	516 (78.2)	
braziers	99 (15)	
gas heaters	27 (4.1)	
Means of arrival to the ED n(%)		
own mean of transportation	460 (70)	
civil protection services	129 (19.5)	
mobile emergency and resuscitation service	61 (9.2)	
hospital ambulance	8 (1.2)	
private ambulance	2 (0.3)	
Symptoms n (%)		
Headaches	561(85)	
Initial loss of consciousness	104(15.7)	
drowsiness	48(7.3)	
Confusion	17(2.6)	
Coma	7(1.1)	
Seizures	16(2.4)	
Digestive symptoms	181(27.4)	
Chest pain	44(6.7)	
Severity n (%)	184 (27.9)	
Clinical findings	**Median [IQR]**	**Extreme values**
GCS		
GCS < 15	26	
GCS ≤ 8	7	
SBP (mmHg)	122 [114, 131]	(90- 206)
DBP (mmHg)	71 [67, 80]	(45- 156)
Heart rate (beats/minute)	85 [78, 96]	(56- 143)
Respiratory rate (breaths/minute)	16 [16, 18]	(12- 35)
Oxygen saturation (%)	99 [98, 100]	(77- 100)
Laboratory findings	**N**	
Blood glucose (mmol/L)	192	6.4 [5.7, 8] (3-35)
Urea (mmol/L)	186	4 [3, 5] (1-11)
Creatinine (µmol/L)	192	65 [56, 78] (34-643)
Serum sodium (mmol/L)	156	137 [135,138] (129-145)
Serum potassium (mmol/L)	156	4 [3.9, 4] (2.77-6.28)
ASAT (UI/L)	15	19 [14, 23] (11-295)
ALAT (UI/L)	15	14 [9, 20] (6-105)
CPK (UI/L)	163	94 [71, 139] (29-2442)
LDH (UI/L)	141	198 [172,232] (62-489)
CRP (mg/L)	79	6 [6,6] (1-46)
Hemoglobin (g/dl)	175	13 [12,14] (6-17)
Leukocytes (cells/ mm³)	175	10150 [8100,12900] (3800-23900)
Troponins (ng/mL)	146	1 [1,5.9] (0-2306)
Lactates (mmol/L)	11	3 [2,6.3] (1.1-11.7)

ALAT: Alanine Aminotransferase, **ASAT**: Aspartate Aminotransferase, **cells** / mm³: cells per cubic millimeter, **COPD**: Chronic Obstructive Pulmonary Disease, **CPK**: Creatine Phosphokinase, **CRP**: C-Reactive Protein, **DBP**: Diastolic blood pressure, **GCS**: Glasgow Coma Scale, **g/dl**: grams per deciliter, **LDH**: Lactate Dehydrogenase, **mg/l** : milligrams per liter, mmHg: millimeters of mercury, **mmol/l**: millimoles per liter, **SBP**: Systolic blood pressure, **UI/l**: Units per liter, **µmol/L**: micromoles per liter.

Severe acute CO poisoning was documented in 27.9% of cases (n = 184). Neurological manifestations were the most frequent severe presentations (90.8%), followed by cardiac complications (10.9%). The median COHb level was 21% [13–29], ranging from 0.5% to 67% with levels exceeding 25% observed in 35.5% of patients (Figure 3). Additional biochemical and hematological parameters, along with extreme values, are outlined in Table 2. Factors significantly associated with severe acute CO poisoning, identified through both univariate and multivariate analyses, are detailed in Table 2. Hyperleukocytosis was identified as a predictive factor for severe acute CO poisoning in multivariate analysis, with an OR of 2.46 (95% CI: 1.32–4.58, p = 0.004).

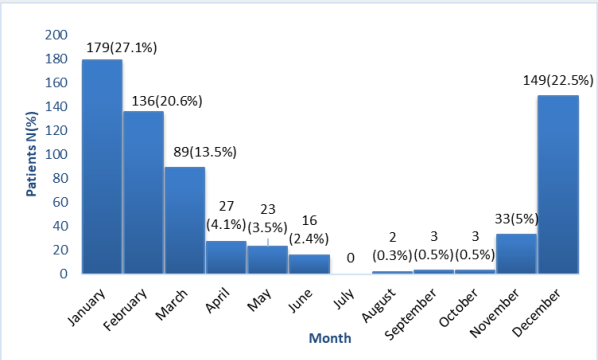

Figure 3. The distribution of COHb levels in our study population

Table 2. Univariate and multivariate analysis of predictive factors for severe acute CO poisoning

Factors	Univariate OR (95% CI)	p-value	Multivariate OR (95% CI)	p-value
Age >50 years	1.53 (1-2.33)	0.04	1.14 (0.44-2.93)	0.77
Medical history	1.47 (1-2.18)	0.05	0.88 (0.41-1.91)	0.76
Collective poisoning	0.71 (0.5-1)	0.05	0.83 (0.44-1.55)	0.83
Asthenia	2.51 (1.47-4.27)	0.001	1.58 (0.5-5)	0.43
Chest pain	2.29 (1.23-4.26)	0.009	1.21 (0.46-3.2)	0.68
COHb >25%	2.85 (2-4.04)	<0.001	0.83 (0.43-1.62)	0.59
Leukocytes >10000 cells/ mm^3	2.38 (1.29-4.38)	0.005	2.46 (1.32-4.58)	0.004

cells/ mm^3: cells per cubic millimeter, **CO**: Carbon monoxide, **HBCO**: carboxyhemoglobin, **CI**: Confidence interval, **OR**: Odds Ratio

Management of acute CO poisoning involved normobaric oxygen therapy with non-rebreather masks at 15 liters per minute for a median duration of 6 hours [2–6], ranging from 1 to 19 hours. Invasive mechanical ventilation was required for three patients due to neurological distress. HBO was indicated in 32.6% of patients (n = 215) and administered to 10.6% (n = 70). Neurological symptoms were the leading indication for HBO (74.9%), followed by myocardial injury (4.9%), acute coronary syndrome (4.9%), pregnancy (4.5%), and elevated COHb levels (10.8%).

Hospitalization was necessary for 21% of patients (n = 141), while 2% (n = 16) were transferred to other EDs. The majority (70%, n = 451) were discharged following treatment. Importantly, no deaths were recorded in our study population.

Discussion

Over the two-year study period, 660 cases of CO poisoning were identified. The global incidence of CO poisoning is estimated at 137 cases per million, with a mortality rate of 4.6 deaths per million [8]. In the United States, CO poisoning accounts for approximately 50,000 annual ED visits, with many cases underdiagnosed [9]. Similarly, in France, an estimated 6,000 hospitalizations are attributed to CO poisoning annually [10].

In our study, the mean age was 35±15 years, with more than half of the patients aged 20–40 years, and 73% were women. This demographic profile aligns with findings from other studies, such as a Taiwanese series reporting a mean age of 37±16 years and 53% female patients [11], and a Turkish study with a mean age of 36±16 years and 60% women [12]. However, a study in Algeria identified a predominance of male patients (72%), underscoring regional differences in demographic trends [13].

Characteristics of CO Exposure

CO poisoning in our study primarily occurred on weekends (51%) and involved gas water heaters as the leading source (78%), followed by braziers (15%) and gas heaters (4%). These patterns are consistent with prior Tunisian studies, where gas water heaters accounted for 69.5% of cases, and the median exposure duration was three hours [14]. A historical comparison revealed a shift in exposure sources, with water heaters replacing braziers as the predominant cause of CO poisoning over the past 45 years [15].

Internationally, regional variations in CO exposure sources are evident. In France and Spain, poisoning is often seasonal and linked to heating systems [2,16]. In the United States, CO exposure is frequently attributed to boilers (23%), motor vehicles (21%), and generators (16%) [17]. Meanwhile, in Taiwan, gas water heaters were implicated in 79% of cases [11].

While all of our patients presented with accidental CO exposure, approximately 15,000 cases of intentional CO poisoning occur annually in the United States, contributing to over two-thirds of reported fatalities [18] of particular concern is the increasing prevalence of intentional CO poisoning through charcoal burning, which has become a significant public health issue in East Asia over the past decade [11].

The median consultation delay in our study was one hour shorter than the two-hour delays reported in previous studies [14,17]. Variations in consultation delays may reflect differences in symptom severity, population characteristics, and healthcare system organization

Clinical Features Associated with Severity

The resulting systemic hypoxia disproportionately affects highly oxygen-dependent organs, particularly the brain and myocardium, which are particularly vulnerable to ischemic injury [6]

Neurological symptoms, although nonspecific to CO poisoning, are diverse [19]. They range from minor

disturbances of consciousness, quickly reversible with ambient air or oxygen therapy, to coma, reported in 3% to 13% of studies [2]. In our study, one-quarter of the population exhibited neurological symptoms, with 1% presenting in a comatose state. Other neurological symptoms included loss of consciousness (6%), confusion syndrome (3%), somnolence (7%), seizures (2%), and one case of unconscious agitation.

These findings align with Raphael et al. prospective study of 629 patients over 4 years, which reported coma in 4%, loss of consciousness in 34%, stupor in 6%, and seizures in 3% [20]. A Moroccan study of severe CO poisoning documented loss of consciousness in 42%, coma in 26%, confusion in 16%, vertigo in 11%, and neurological deficits in 5% of cases [19].

The severity of CO poisoning is further marked by the risk of delayed neurological sequelae, occurring 1–3 weeks after an apparent recovery, affecting up to 10% of survivors [21,22].

Cardiovascular effects were also reported to be associated with CO poisoning, from mild, transient lesions to necrosis and contractile dysfunction [7,23]. CO's pro-thrombotic potential has been linked to fibrinogen binding [24], increased platelet aggregation, polycythemia, and coronary vasospasm [18].

While our study reported no hemodynamic instability, it has been documented in the literature, particularly in severe cases of CO poisoning [23,25]. This instability is often linked to myocardial dysfunction, characterized by impaired contractility due to the direct toxic effects of CO on cardiac myocytes, oxidative stress, and tissue hypoxia [7,23].

ECG abnormalities were seen in 13% of cases (n = 88). Literature reports a higher frequency of ECG changes, between 40% and 60% [22,26]. In a U.S. study by Satran et al., 37% of CO poisoning patients had elevated cardiac biomarkers or ECG changes [27]. Similarly, Henry et al. observed myocardial injury in 37% of patients over an 8-year prospective study [28].

The lower detection rate in our study could be due to the absence of systematic ECG and cardiac biomarker assessments, potentially underestimating cardiac involvement compared to studies where these tests were routinely performed.

Additional complications of CO poisoning included acute pulmonary edema, rhabdomyolysis leading to renal failure, and visceral injuries such as pancreatitis [5].

Non-Severity-Related Clinical Features

Headaches, though not pathognomonic for CO poisoning [23], were the most common clinical symptom in our study, occurring in 85% of cases (n = 561).

This finding aligns with the study by Raphael et al., where headaches were present in 83% of cases [20].

Table 3 summarizes the main functional symptoms observed in our study and those reported in various studies in the literature

Tableau 3. Comparison of functional symptoms observed in CO poisoning across studies

Functional signs	Headaches (%)	Dizziness (%)	Hypotonia/ muscular weakness/ asthenia (%)	Chest pain (%)	Dyspnea (%)	Digestive disorders (Diarrhea/ Vomiting) (%)	Nausea (%)
Burney et al. [29] N= 184	90	82	53	-	40	46	
Raphael et al. [20] N= 629	83	75		-	-	51	
Duenas-Laita et al. [16] N= 149	94	56	61	6	-	7	45
Selini [30] N= 230	33	10	15	2	-	12	
Jerraya [14] N= 100	34	16	22	-	-	40	
Hampson et al. [17] N= 1323	56	34	25	8	8	38	
Hemery [31] N =91	58	40	9			40	
Our study N= 660	85	57	11	7	4	20	6

Additional symptoms documented in the literature include visual disturbances, challenges with concentration [29,32], auditory disturbances, and dry mouth [16].

Severity predicting factors in carbon monoxide poisoning

Individuals with pre-existing health conditions are at a higher risk of experiencing severe outcomes when exposed to CO poisoning, as these conditions can impair the body's ability to manage low oxygen levels effectively [33].

In our study, we observed that medical history was significantly associated with severe CO poisoning in univariate analysis (OR = 1.47, p = 0.05), though it was not an independent predictor in the multivariate model (OR = 0.88, p = 0.76).

The role of COHb in diagnosis is well-established, yet its utility as a prognostic marker remains debated [34]. While COHb levels exceeding 25% are frequently linked with severe symptoms and considered a threshold for recommending HBOT [10], this association is inconsistent. For example, Satran et al. found no correlation between COHb levels and myocardial injury [27].

Similarly, a study of over 1,000 CO poisoning cases in the United States revealed no significant relationship between COHb levels and approximately 50 reported symptom categories [17].
In our study, although COHb levels greater than 25% were significantly associated with poisoning severity in univariate analysis (OR = 2.85, p < 0.001), this factor was not an independent predictor in the multivariate model (OR = 0.83, p = 0.59). This further supports the hypothesis that COHb levels alone may not reliably predict the clinical severity of CO poisoning.
Although not specific to CO poisoning, elevated white blood cell counts have been frequently observed in patients with more severe forms associated with neurological or cardiac complications, reflecting an acute inflammatory response linked to the physiological stress of poisoning [35,36]. In a study conducted in Germany involving 173 patients admitted to the intensive care unit for CO poisoning, a significant association was found between the severity of poisoning and a high white blood cell count [37].
Our study confirms these findings, demonstrating hyperleukocytosis as a significant factor in univariate (OR = 2.38, p = 0.005) and multivariate analysis (OR = 2.46, p = 0.004).

Management

CO poisoning should be managed in a hospital, by taking a thorough history and physical exam while monitoring vital signs such as heart rate, blood pressure, and oxygen saturation, along with necessary biological and electrocardiographic tests [38]. Oxygen therapy is the cornerstone of treatment [6].
The elimination of CO depends on the dissociation rate of COHb complexes [21]. Once exposure ends, COHb dissociation follows an exponential curve, with a half-life of about 320 minutes in ambient air [22]. Oxygen therapy accelerates this process; under normobaric oxygen (1 atmospheres absolute, the half-life drops to 90 minutes, and under HBO (2-3 atmospheres absolute), it is reduced to 20 minutes [22].
In our study, invasive mechanical ventilation was required for three patients due to neurological distress. The other patients received normobaric oxygen therapy with a high-concentration mask at 15 liters per minute for a median duration of 6 [2, 6] hours (range 1–19 hours).
A 2015 Taiwanese study on 796 patients with CO poisoning reported that 23.4% required mechanical ventilation [39]. The discrepancy between our study and others may be attributed to the lower suicide rate from carbon monoxide poisoning in Tunisia, where the majority of cases are unintentional, in contrast to countries like Taiwan, where poisoning is often linked to suicide attempts associated with poor outcomes [11].
Currently, there is no clear consensus for HBO due to the variability in evidence from different studies [40]. Decisions are made based on the severity of the clinical presentation, aiming to prevent neurological sequelae and cardiac complications, thus reducing mortality. Indications for HBO include altered neurological status, coma, focal neurological deficits, pregnancy, and acute myocardial ischemia [40]. Relative indications include loss of consciousness, severe metabolic acidosis (pH < 7.2), COHb > 25-40%, a history of heart disease, hemodynamic instability, extreme age, and persistent symptoms despite normobaric oxygen therapy [40].
In our study, HBO was indicated for 215 patients (32.6 %) but performed in only 70 patients (10.6 %). Similarly, a French study conducted by Brianchon T. (2006–2010) reported that 29% of patients with CO poisoning received HBO [3].
The low utilization rate of hyperbaric oxygen therapy (HBO) observed in our study can be explained by two primary factors: the refusal of HBO treatment by some patients and the limited accessibility to hyperbaric facilities.

Prevention and resource optimization

To optimize resources and improve outcomes, we propose public awareness campaigns on placing carbon monoxide sources outside homes and using detection devices, enhancing access to hyperbaric oxygen (HBO) therapy through better emergency coordination, refining HBO criteria to prioritize high-risk neurological cases, mandating regular maintenance of CO-emitting sources through legislation, and providing healthcare professionals with ongoing training. A collaborative, multidisciplinary approach is vital to enhancing patient care efficiency.

Strength and limitations of our study

Strengths of our study include the inclusion of measurement of COHb levels and a relatively large sample size, enhancing the statistical power and generalizability of the findings.
Limitations of our study include its retrospective design, which may introduce biases such as incomplete data or reliance on existing medical records. Furthermore, as a monocentric study conducted at a single institution, the findings may not be fully representative of broader populations or healthcare settings.

Conclusion

CO poisoning in Tunisia predominantly affects young adults and women (73.3%), with gas water heaters as the leading source (78.2%). Neurological and cardiac manifestations predominate in severe cases with respectively 90.8% and 10.9%, emphasizing the need for public health interventions based on prevention and early management.

Abbreviations List
ALAT: Alanine Aminotransferase
ASAT: Aspartate Aminotransferase
cells / mm³: cells per cubic millimeter
CI: Confidence interval
CO: Carbon monoxide poisoning
COHb: Carbon monoxide hemoglobin
COPD: Chronic Obstructive Pulmonary Disease
CPK: Creatine Phosphokinase
CRP: C-Reactive Protein
DBP: Diastolic blood pressure
ED: Emergency department
GCS: Glasgow Coma Scale
g/dl: grams per deciliter
HBO: Hyperbaric oxygen therapy
LDH: Lactate Dehydrogenase
mg/l: milligrams per liter
mmHg: millimeters of mercury
mmol/l: millimoles per liter
SBP: Systolic blood pressure
UI/l: Units per liter
µmol/L: micromoles per liter

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