HEMATOLOGICAL PARAMETERS AS OUTCOME PREDICTORS IN ACUTE CARBON MONOXIDE POISONING

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ABSTRACT

Background: Carbon monoxide (CO) a silent killer is recognized as a major health threat in clinical toxicology that can result in neurological sequelae or even death. Among survivors of CO poisoning, 15–40% may experience long-term neurological and neuropsychiatric effects leading to brain damage. The neutrophil-to-lymphocyte ratio (NLR) is an inflammatory indicator generated from peripheral blood, indicating the equilibrium between systemic inflammation as well as immunological response.

Objectives: This work aims to study the hematological parameters as outcome predictors in patients who were acutely poisoned by CO and admitted to Tanta University Poison Control Center (TUPCC), Egypt.

Methodology: The data of the patients of this study were collected from the medical files including demographic Information, symptoms at the time of admission, Electrocardiogram (ECG) and laboratory tests complete blood count (CBC) with differential, renal and liver function tests, random blood glucose and arterial blood gases). Carboxyhemoglobin levels were measured using oximetry. Utilizing hyperbaric oxygen (HBO_2) therapy, ICU admission, total length of hospitalization and follow-up findings were studied.

Results: Poor outcomes of acutely CO-intoxicated patients were statistically significantly associated with sinus tachycardia, ICU admission, longer hospital stay, low GCS, lower O2 saturation and higher COHb level (P value< 0.005). Regarding CBC, a significantly increased white blood cell (WBC) count, mean neutrophil count and neutrophil-lymphocyte ratio (NLR) were linked to poor outcomes. Conversely, significantly lower lymphocyte count and platelet volumes were detected. Based on the ROC curves, NLR showed the most significant studied indicator of poor outcome (100.0% sensitivity, 100.0% specificity, and 1.00 area under the curve), while lymphocyte count displayed the weakest diagnostic capability (80.0% sensitivity, 68.9% specificity and 0.790 area under the curve).

Conclusion: CBC Differential is a simple and cost-effective laboratory test that is routinely performed in most laboratories. Combining CBC data with clinical findings enhances the ability to predict outcomes in acute CO poisoning and identify patients requiring ICU admission and HBO_2 therapy. NLR was a significant predictor of poor clinical outcomes of CO Poisoning.

Keywords: *Prediction; Poor outcome; Acute carbon monoxide poisoning*

INTRODUCTION

Carbon monoxide CO is an asphyxiant gas which represents one of the most common causes of death from poisoning worldwide. It can lead to neurological sequelae which have an intense effect on the fineness of life of patients and their families. Around 30% of

burn fatalities were subjected to CO (**Wankhade et al., 2024**) **and (Rose et al., 2017)**. The main sources of CO are: partial combustion of organic compounds and fuels, employing heating and cooking appliances with inadequate airflow or inadequate care, fire smoke, exhaust fume from means of transportation using internal combustion engines, primary smoke from cigarettes (3–4 %) and industrial accidents **(Kinoshita et al., 2020).** In heavy smokers, the saturation of blood carboxyhemoglobin (CO-Hb) is elevated to about 10–15 % **(Owens, 2010)**.

The manner of poisoning may be accidentally and intentionally used as a means of suicide **(Yoshioka et al., 2014)**. CO possesses attraction to hemoglobin that is 230 to 270-times greater than that of oxygen **(Powers and Dean, 2016).** The formation and the clearance half-life of CO-Hb in the blood is influenced by the amount of inspired CO, the length of CO exposure, the ventilatory function, physical activity and the patient's fitness (**Shimazu, 2006, Wankhade et al., 2024**). Tissue hypoxia occurs because of the formation of CO-Hb. Moreover, it leads to leftward shift in the hemoglobin-oxygen dissociation curve, complexes to various cytoplasmic proteins, and may disrupt adenosine triphosphate (ATP) synthesis at the cytochrome level **(Ghosh et al., 2016, Umahi-Ottah et al., 2022).**

CO triggers the launch of proteases, myeloperoxidase and reactive oxygen species, leading to the aggregation of platelets and neutrophils. Oxidative stress induces fatty acid peroxidation and apoptosis, with these results being particularly prominent in the central nervous system. This explains the medical presentation associated with nitric oxide-triggered vasodilation and oxidative injury (**Guzman, 2012)**.

When carboxyhemoglobin levels are below 10% of the total hemoglobin in the blood, symptoms typically do not manifest. At a concentration of 10–30%, neurological manifestations (dizziness, headache, weakness, confusion and visual troubles) will appear **(WHO, 1999).** Ischemic alterations in ECG and increased cardiac biomarkers have also been documented. However the level of carboxyhemoglobin 50–60% is fatal, levels approaching 20% may be lethal in individuals with advanced coronary artery disease **(Ozturk et al., 2015).**

The initial step of management is direct removal from the polluted situation. Next, managing the airway, securing intravenous access and monitoring the heart rate are essential. Delivering 100 % oxygen via an oxygen mask or endotracheal tube is necessary to remove CO from the bloodstream. HBO_2 therapy can be viewed to accelerate the clearance of CO, the inflammation resolution and mitochondrial restoration **(Taki, 2009 and Rose et al., 2017)**.

Hematological parameters are distinct components of blood, each serving crucial functions such as transporting nutrients throughout the body, removing waste products (including gases), defending the body against pathogens, and ensuring clotting efficiency. When the balance between the body and blood is disrupted, it can lead to abnormalities that may result in morbidity or mortality **(Umahi-Ottah et al., 2022)**.

Neutrophil lymphocyte ratio is a reliable inflammatory biomarker that effectively predicts adverse outcomes in patients with acute medical illnesses, including sepsis, acute pancreatitis, and infectious diseases **(Liu et al., 2022**). Furthermore, NLR performs a definitive role in assessing the prognosis of patients with neurological injuries and predicting neurological outcomes in individuals with acute ischemic stroke **(Chen et al., 2022)**.

At present, various laboratory, clinical and epidemiological markers can be used to proficiently expect the outcomes of CO-intoxicated patients (**Huang et al., 2019, Gao et al., 2021** and **Sert et al., 2021)**. Although, some of these parameters are not convenient to get, so it is challenging to access them easily during clinical settings.

AIM OF THE STUDY

This work aimed to investigate the hematological parameters as outcome predictors in acute CO-poisoning.

PATIENTS AND METHODS

Study design:

This prospective study was directed to analyze outcomes of CO acutely poisoned patients who were admitted to TUPCC, from the first of February 2024 to the end of March 2024.

Ethical consideration:

The present study began after the approval taken from Research Ethical Committee of Tanta Faculty of Medicine (Approval code 36264PR510/1/24). Data were retrieved from the patients' files with concealing their identity to protect confidentiality.

Inclusion and exclusion criteria:

All patients admitted with acute CO poisoning during the studied period with complete hospital medical records were included. Diagnosis was based on history given by patients or family members, characteristic clinical findings and laboratory investigations. Patients with chronic poisoning, those who had co-ingested substances, individuals who were transferred from other hospitals without comprehensive medical records and patients with an accompanying condition as concomitant head trauma were excluded.

Data collection tools

The data of the poisoned patients in this study were collected from the medical archives into an extraction excel sheet including:

1- Demographic Information: age, sex, and residence of the patients

2- Symptoms at presentation, involving altered mental status (Glasgow Coma Scale; GCS), respiratory distress, gastrointestinal symptoms, and cardiovascular instability.

3- ECG and laboratory tests were conducted at University's laboratory of the emergency hospital, Tanta, Egypt. The laboratory tests involved complete blood count (CBC) with differential, electrolytes, renal function tests (serum creatinine, blood urea), liver function tests (SGPT, SGOT), random blood glucose and arterial blood gases.

4- Carboxyhemoglobin levels using co-oximetry.

5- Use HBO_2 therapy.

6- Intensive Care Unit (ICU) Admission and total length of hospital stay from admission to discharge.

Outcome measures:

The acutely CO-poisoned patients were categorized into poor and non-poor. This categorization centered on the need for HBO_2, admission to ICU, hospital stay duration and development of delayed neurologic sequelae within 6 months of follow-up.

Statistical analysis:

The whole data were organized and analyzed by the IBM SPSS Statistics for Windows software program, version 27 using. Categorical data were presented as frequencies and percentages. Numerical data underwent normality testing using the Shapiro-Wilk test. Data followed a normal distribution were displayed as mean ± SD, while abnormally distributed or skewed data were displayed as median and interquartile range (25^{th} -75^{th} percentiles). Regarding categorical data, comparing between the studied groups was performed by Pearson's Chi-Square test. When more than 20% of cells expected count less than 5 Fisher's Exact and Fisher-Freeman-Halton Exact tests was adopted instead of Pearson's Chi-Square test. Normal distributed numerical data were compared by the Independent Samples T-test. Alternatively, Mann-Whitney U test stayed executed to contrast skewed data statistically significant is achieved if P less <0.05. Furthermore, receiver functioning characteristics (ROC) curves were created to judge the discriminating power (area

under the curve), sensitivity, specificity of the significant parameters in the basic bivariate comparisons.

RESULTS

Table (1) presented the demographic data and clinical presentations of acute CO-poisoned patients (non-poor; 74.4 % vs. poor outcomes (25.6%). Poor outcome patients were ten distributed as following, 2 died, 4 with neurological sequelae and 4 with neuropsychiatric sequelae. The median age of both groups is similar, with no statistically significant difference ($p = 0.341$). Gender distribution does not differ significantly among the examined groups ($p = 0.465$). The percentage of patients from rural and urban areas is comparable between the studied groups, without statistical significance ($p = 0.999$). Regarding medical history, the presence of asthma or other medical history does not significantly correlate with outcomes ($p = 0.452$). Moreover, symptoms like vomiting, headache and drowsiness were not significantly associated with poor outcomes. Convulsions had a borderline p-value ($p = 0.057$).

The source of CO exposure (e.g., gas heater, defective gas stove, charcoal burning, car motor, fire) does not appear to be significantly associated with outcomes ($p = 0.461$). The median delay in seeking care was parallel in both groups ($p = 0.862$). The median duration of exposure showed significant difference ($p = <0.001$) between poor and nonpoor outcomes groups.

Sinus tachycardia was significantly associated with poor outcomes ($p = 0.032$).

Ninety percent of patients with poor outcome received HBO_2 therapy ($p < 0.001$). ICU admission was significantly linked to unfavorable results ($p < 0.001$). Patients with poor outcomes had a longer hospital stay, with significant statistical support ($p < 0.001$).

Table (2) provided the statistical findings comparing both clinical and laboratory assessments among the two studied groups, where the median GCS is significantly reduced in the poor outcome group (12 vs. 15, $p < 0.01$), which highlighted the strong correlation between poor neurological status and worse outcomes in acute CO poisoning. The mean systolic and diastolic blood pressure expressed no statistically significant difference between groups ($p = 0.377$) ($p = 0.540$) respectively. No significant difference was found in respiratory rate (RR) and temperature between the groups ($p = 0.568$, 0.345, respectively). A significant difference is observed, with higher O_2 saturation in the non-poor outcome group (97.5% vs. 85.3%, $p < 0.001$). This emphasized the importance of oxygenation in determining outcomes. A significantly higher COHb level is observed in the poor outcome group (median 28.0% vs. 9.0%, $p < 0.001$).

Regarding laboratory findings, no significant difference was observed in the median random blood glucose (RBG) levels ($p = 0.334$), both sodium (Na) and potassium (K) Levels ($p = 0.167$ and $p = 0.952$, respectively), renal function tests (Urea $p = 0.176$ and Creatinine $p = 0.504$), and liver function tests (SGPT $p = 0.077$ and SGOT $p= 0.072$). Regarding arterial blood gases, the mean pH is slightly lower in the poor outcome group but not statistically significant ($p = 0.157$). It could be observed that, there was not a significant distinction in mean HCO_3 levels ($p = 0.247$) and PCO_2 ($p = 0.463$).

Table (3) Compared CBC parameters between CO poisoned patients with and without poor outcome. The mean hemoglobin was slightly worse in the group with poor outcomes (11.2 ± 1.4 g/dL) in comparison to the group with non-poor outcome (12.5 ± 1.9 g/dL), nonetheless this variability was not statistically significant ($p = 0.064$). The mean RBCs count was also lower in the poor outcome group (3.99 ± 0.73) compared to the non-poor group (4.38 ± 0.66). Nonetheless, this variation was not significant statistically ($p = 0.124$). The poor outcome group had a mean hematocrit of 34.1 ± 6.0%, while the non-poor group had 36.6 ± 5.7%. This difference was not statistically significant ($p = 0.245$).

A significant increase in WBCs count was observed in the group with poor outcome ($13.1 \pm 4.0 \times 10^3$) in comparison to the group with non-poor outcomes ($8.6 \pm 2.3 \times 10^3$) ($p = 0.006$). The poor outcome group showed a significantly higher mean neutrophil count ($9.4 \pm 3.0 \times 10^3$) compared to the non-poor group ($4.5 \pm 1.4 \times 10^3$) ($p < 0.001$). The count of lymphocyte was considerably lower in the poor outcome group ($2.4 \pm 0.8 \times 10^3$) in contrast to the non-poor group ($3.5 \pm 1.1 \times 10^3$) ($p = 0.006$). Neutrophil-lymphocyte ratio (NLR) was substantially higher in the group of poor outcome (4.0 ± 6) compared to the non-poor one (1.4 ± 5), showing a highly significant difference ($p < 0.001$). Regarding monocyte counts, there was no statistically significant difference between examined groups ($p = 0.074$).

Platelet counts were similar between the poor ($302.7 \pm 82.6 \times 10^9$) and non-poor ($294.2 \pm 70.4 \times 10^9$) outcome groups, without any statistically significance ($p = 0.754$).

The mean platelet volume was significantly reduced in the group with unfavorable results (6.6 ± 1.4 fL) relative to the non-poor group (9.8 ± 1.1 fL) ($p < 0.001$)

Table (4) Summarized key predictors of the poor outcomes in acute CO poisoning cases using pairwise comparisons of ROC curve analysis. Prolonged duration of CO exposure (>2 hours) correlated with poor outcomes (AUC = 0.86). Elevated carboxyhemoglobin (COHb) levels (>15%) and Glasgow Coma Scale (GCS ≤13) on admission showed perfect sensitivity and specificity (AUC = 1.0). An oxygen saturation ≤92% significantly predicted adverse outcomes (AUC = 0.974).

As regards to laboratory parameters; WBCs count $>11.2 \times 10^3$, Neutrophil count $>6.2 \times 10^3$, lymphocyte count $\leq 2.8 \times 10^3$, and neutrophil-to-lymphocyte ratio >2.3 all demonstrated strong predictive power (AUC > 0.9 for most). Platelet volume (<8.5) served as an additional marker (AUC = 0.966). All parameters were significant statistically with P-values <0.001, supporting the robustness of these findings in toxicological risk stratification.

Table (1): Demographic, toxicity, and clinical presentation of the acute carbon monoxide poisoned patients with and without poor outcome (N=39)

		Total		**Outcome**				**Tests of significance**	
		N = 39		**Non-poor**		**Poor**		**Test**	**P-**
				N = 29 (74.4 %)		**N = 10 (25.6%)**		**statistic**	**Value**
Age, years	Median (IQR)	22.0 (16.0-25.0)		22.0 (16.0-25.0)		21.0 (18.0-39.0)		Z_{mw}= 0.952	0.341
Sex	Female	18	46.2%	12	41.4%	6	60.0%	FE	0.465
	Male	21	53.8%	17	58.6%	4	40.0%		
Residence	Rural	18	46.2%	13	44.8%	5	50.0%	FE	0.999
	Urban	21	53.8%	16	55.2%	5	50.0%		
Medical history	No	37	94.9%	28	96.6%	9	90.0%	FE	0.452
	Asthma	2	5.1%	1	3.4%	1	10.0%		
Symptoms	Vomiting	20	51.3%	13	44.8%	7	70.0%	FE	0.273
	Headache	17	43.6%	12	41.4%	5	50.0%	FE	0.721
	Drowsiness	27	69.2%	19	65.5%	8	80.0%	FE	0.693
	Convulsion	7	17.9%	3	10.3%	4	40.0%	FE	0.057
Source of exposure	Gas heater	19	48.7%	14	48.3%	5	50.0%	FFH	0.461
	Defective gas stove	8	20.5%	7	24.1%	1	10.0%	Exact= 3.553	
	Charcoal burning	7	17.9%	5	17.2%	2	20.0%		
	Car motor	3	7.7%	1	3.4%	2	20.0%		
	Fire	2	5.1%	2	6.9%	0	0.0%		
Delay, hours	Median (IQR)	2.0 (1.0-4.0)		2.0 (1.0-4.0)		1.8 (1.0-4.0)		Z_{mw}= -0.179	0.862
Duration of exposure, hours	Median (IQR)	2.0 (1.0-3.5)		1.5 (0.5-2.0)		4.3 (2.5-10.0)		Z_{mw}= 3.394	<0.001 *
ECG	Sinus tachycardia	20	51.28%	12	41.38%	8	80.0%	FE	0.032*
HBO2	No	29	74.4%	28	96.6%	1	10.0%	FE	<0.001
	Yes	10	25.6%	1	3.4%	9	90.0%		*
ICU admission	No	33	84.6%	29	100.0%	4	40.0%	FE	<0.001
	Yes	6	15.4%	0	0.0%	6	60.0%		*
Hospital stays, days	< 1	26	66.7%	26	89.7%	0	0.0%	FFH	<0.001
	1-3	6	15.4%	2	6.9%	4	40.0%	Exact= 25.439	*
	>3	7	17.9%	1	3.4%	6	60.0%		

HBO2: Hyperbaric Oxygen, **ICU:** Intensive Care Unit, **IQR:** Interquartile Range, **ECG:** Electrocardiography, *Significant at p<0.05, **FE:** Fisher's Exact test, **FFH:** Fisher- Freeman –Halton Exact test, $\mathbf{Z_{mw}}$: Mann-Whitney U test

Table (2): Comparison of initial clinical and laboratory assessments of the CO- poisoned patients with and without poor outcomes

			Outcome		Tests of significance	
		Total N = 39	**Non-poor N = 29 (74.4 %)**	**Poor N = 10 (25.6%)**	**Test statistic**	**P-Value**
GCS	Median (IQR)	15.0 (13.0-15.0)	15.0 (15.0-15.0)	12.0 (8.0-13.0)	Z_{mw}= -5.711	<0.001*
SBP	Mean ± SD	117.6±16.6	119.0±17.0	113.5±15.6	t= -0.895	0.377
DBP	Mean ± SD	75.9±20.0	77.1±22.3	72.5±10.9	t= -0.618	0.540
RR	Mean ± SD	20.6±4.3	20.4±3.6	21.3± 6.1	t= 0.576	0.568
O_2 saturation%	Mean ± SD	94.3±7.0	97.5±2.9	85.2±7.7	t= -4.940	<0.001*
Temperature	Mean ± SD	37.0±0.2	37.1±0.2	37.0±0.2	t= -0.938	0.345
COHb level %	Median (IQR)	10.0 (8.0-18.0)	9.0 (8.0-10.0)	28.0 (18.0-39.0)	Z_{mw}= 4.686	<0.001*
RBG	Median (IQR)	119.0 (103.0-131.0)	115.0 (104.0-122.0)	125.5 (95.0-169.0)	Z_{mw}= 0.966	0.334
pH	Mean ± SD	7.45±0.10	7.46±0.09	7.41±0.12	t= -1.445	0.157
HCO_3	Mean ± SD	21.2±4.2	20.7±4.2	22.5±4.1	t= 1.176	0.247
PCO_2	Mean ± SD	29.3±6.1	28.8±6.6	30.5±4.4	t= 0.742	0.463
Na	Mean ± SD	137.7±3.2	137.2±3.4	138.9±2.0	t= 1.408	0.167
K	Mean ± SD	3.9±0.5	3.9±0.5	3.8±0.4	t= -0.061	0.952
Urea	Mean ± SD	32.3±6.1	33.1±5.7	30.0±7.0	t= -1.378	0.176
Creatinine	Mean ± SD	0.92±0.27	0.94±0.30	0.87±0.14	t= -0.675	0.504
SGPT	Mean ± SD	23.4±10.5	25.5±9.3	17.4±3.6	t= -1.817	0.077
SGOT	Mean ± SD	21.0± 8.2	22.4±8.7	17.0±4.7	t= -1.849	0.072

GCS: Glasgow Coma Scale, **SBP:** Systolic Blood Pressure, **DBP:** Diastolic Blood Pressure, **RR:** Respiratory Rate, **O_2**: Oxygen, **COHb:** Carboxyhemoglobin, **RBG:** Random Blood Glucose, **HCO_3:** Bicarbonate, **pH:** Potential of Hydrogen, **PCO_2**: Partial Pressure of carbon Dioxide, **Na:** Sodium, **K:** Potassium, **SGPT:** Serum Glutamate Pyruvate Transaminase, **SGOP:** Serum Glutamate Oxaloacetate Transaminase, **SD:** Standard Deviation, *****Significant at p<0.05, **t:** independent samples T test , **Z_{mw}**: Mann-Whitney U test, **IQR:** Interquartile Range.

Table (3): Comparison of complete blood count parameters between the CO- poisoned patients with and without poor outcome (N=39)

			Outcome		Independent T test	
		Total N = 39	**Non-poor N = 29 (74.4 %)**	**Poor N = 10 (25.6%)**	**T**	**P-Value**
Hemoglobin g/dl	Mean ± SD	12.2±1.9	12.5±1.9	11.2±1.4	-1.911	0.064
RBCs count	Mean ± SD	4.28±.69	4.38±.66	3.99±.73	-1.573	0.124
Hematocrit%	Mean ± SD	36.0±5.8	36.6±5.7	34.1±6.0	-1.182	0.245
WBCS count *10^3	Mean ± SD	9.8±3.4	8.6±2.3	13.1±4.0	3.391	0.006*
Neutrophil count *10^3	Mean ± SD	5.8±2.9	4.5±1.4	9.4±3.0	0.4899	<0.001*
lymphocyte count *10^3	Mean ± SD	3.2±1.1	3.5±1.1	2.4±.8	-2.905	0.006*
Neutrophil lymphocyte ratio	Mean ± SD	2.1±1.3	1.4±.5	4.0±.6	14.778	<0.001*
Monocyte count *10^3	Mean ± SD	0.4±0.1	0.6±0.1	0.4±0.2	1.838	0.074
Platelets count*10^3	Mean ± SD	296.4±72.7	294.2±70.4	302.7±82.6	0.316	0.754
Platelet volume fL	Mean ± SD	8.9±1.8	9.8±1.1	6.6±1.4	-7.335	<0.001*

*Significant at p<0.05, **t:** independent samples T test, **fL:** Femtoliter, **g/dl:** Gram per Deciliter, **SD:** Standard Deviation.

Table (4): Receiver operating characteristics (ROC curve) analyses to assess diagnostic performance of the significant clinical and laboratory parameters as predictors of poor outcome.

Predictors	Cut off	Sensitivity %	Specificity %	AUC	95% CI of AUC	P-Value
Duration of CO exposure (hours)	>2	80.0	79.3	0.860	0.712 to 0.950	<0.001*
COHb level (%)	>15	100.0	100.0	1.000	0.910 to 1.000	<0.001*
GCS on admission	≤13	100.0	100.0	1.000	0.910 to 1.000	<0.001*
Oxygen saturation (%)	≤92	100.0	89.6	0.974	0.865 to 0.999	<0.001*
WBCS count *10^3	>11.2	80.0	89.6	0.829	0.675 to 0.930	0.001*
Neutrophil count *10^3	>6.2	80.0	93.1	0.921	0.788 to 0.983	<0.001*
lymphocyte count *10^3	≤2.8	80.0	68.9	0.790	0.629 to 0.903	<0.001*
Neutrophil lymphocyte ratio	>2.3	100.0	100.0	1.000	0.910 to 1.000	<0.001*
Platelet volume fL	≤8.5	90.0	89.6	0.966	0.852 to 0.998	<0.001*

Pairwise comparisons of ROC curves of WBCs differential revealed significant differences only between lymphocytes count versus neutrophils-lymphocytes ratio (p=0.004*), and lymphocytes count versus platelet volume (p=0.031*)

Pairwise comparisons of ROC curves of clinical parameters revealed significant differences between GCS versus duration of exposure (p=0.024*), and COHB level versus duration of exposure (p=0.024*)

Otherwise, no significant differences between any of the studied parameters by ROC curve analysis

AUC: area under the curve, **CI:** confidence interval, **GCS:** Glasgow Coma Scale, **COHb:** carboxy hemoglobin, *significant at p<0.05, **WBCS:** White Blood Cells, **CO:** Carbon Monoxide, fL: Femtoliter

DISCUSSION

CO is an asphyxiant gas. It is released throughout the natural substances burning such as wood, coal, gasoline, natural gas, and tobacco. Humans can unintentionally face harmful CO levels primarily through breathing in smoke or exhaust in an enclosed space **(Rose et al., 2017)**.

This prospective cohort study was directed on thirty-nine acutely CO intoxicated patients who were admitted to TUPCC from the first of February 2024 till the end of March 2024. They grouped into non-poor outcome group (29 patients, 74.4 %) and poor outcome group (10 patients, 25.6%).

In the current study, there was statistically significant difference between the two groups of non-poor and poor outcome regarding length of exposure to CO, duration of hospital stay in days, sittings of HBO_2 therapy and admission to ICU. These findings were agreed with **Ku et al., 2010, Liao et al., 2019, Shahin et al., 2020 and Lee et al., 2021** who reported longer duration of exposure to CO, admission to ICU and a greater number of sessions of HBO_2 therapy in non-survived group and in patients with delayed neuropsychological complication. Oxygen administration speeds CO elimination from the human body. Administering 100% oxygen through a close-fitting face mask at standard atmospheric pressure reduces the elimination half-life of CO from 4 hours to 1 hour. In contrast, using HBO_2 chamber at 2.5 atmospheric pressure further decreases the elimination half-life to approximately 20 minutes. Thus, HBO_2 therapy effectively restores tissue oxygenation, enhances mitochondrial function, and modulates the inflammatory response caused by CO exposure **(Wolf et al., 2008)**.

Regarding the Glasgow coma scale and O_2 saturation, it was observed that there was a statistically significant reduction in both and increase in COHb level in the poor outcome group. The same result was concluded by **Ashry et al., 2023**, as they found that more than half of the acutely CO-poisoned patients had disturbed levels of consciousness and low GCS, indicating

poor neurological function with increased mortality. **Namgung et al., 2022** confirmed that an initial low GCS could be considered as a significant predictor for delayed neurologic complication.

Initially in CO poisoning, the oxyhemoglobin decreases leading to tissue hypoxia which prompts consciousness-related changes in the central nervous system. This effect is enhanced with increasing the time of exposure. The GCS serves as a vital tool in assessing the severity of brain injury. In CO-poisoning, a lower GCS may reflect more severe hypoxia and longer exposure to CO, which are critical determinants of neurological outcomes **(Hampson et al., 2008)**. So, the current findings suggest that timely evaluation of consciousness levels can guide clinical decision-making and potentially stratify patients based on their risk for poor outcomes.

Interestingly, while neurological status emerged as a significant predictor of outcomes, other clinical parameters such as mean SBP, DBP, RR, and temperature did not show significant differences between the groups. The lack of significant variation in hemodynamic parameters (SBP and DBP) ($p = 0.377$ and $p = 0.540$, respectively) indicates that traditional vital signs may not serve as reliable indicators of severity in cases of CO-poisoning. This observation aligns with findings from prior studies, which concluded that vital signs alone couldn't be considered as prognostic markers for acute CO-intoxication. Hence, continuous and close monitoring of patients is essential to ensure timely intervention and management. **(Aksu et al., 2012)**. The absence of significant differences in these parameters suggests that respiratory compensation may not be sufficient to counteract the effects of CO-induced hypoxia, particularly in patients experiencing poor outcomes. The same results reported by **Shahin et al., 2020**.

Sinus tachycardia was significantly associated with poor outcomes ($p = 0.032$), emphasizing its potential as a clinical indicator of severity. A research by **Eroglu et al. (2014)** found that the mean heart rate in CO-intoxicated patients was slightly higher than that of well individuals, although this difference was not statistically significant ($p > 0.05$). CO can cause both reversible and persistent myocardial damage due to myocardial hypoxemia and its direct effects on the heart. By binding to myoglobin, CO may reduce oxygen availability in cardiac tissue, leading to arrhythmias and impaired cardiac function **(Henz and Maeder, 2005).**

Furthermore, our study noted a lower mean pH in the group of poor outcome, but it wasn't statistically significant ($p = 0.157$). The lack of significant differences in mean bicarbonate (HCO_3) levels ($p = 0.247$) and PCO_2 ($p = 0.463$) further supports the idea that acid-base status may not be a primary determinant of outcomes in CO-poisoning. This is particularly relevant in the context of CO exposure, where the primary concern is the direct impact of hypoxia on the central nervous system rather than metabolic acidosis, which may be more pronounced in other toxicological emergencies. The same result was described by **Pan et al., 2019.**

The significantly higher carboxyhemoglobin (COHb) levels in the poor outcome group (median 28.0% vs. 9.0%, $p < 0.001$) further corroborate the association between acute CO exposure severity and adverse outcomes. This finding is associated with the established understanding that higher COHb concentrations correlate with more severe neurological impairment and increased mortality risk. Conversely, **Cervellin et al., 2014 & Hampson 2018** discussed the increased COHb concentrations were not related to the severity of acute CO-poisoning or complicated outcomes.

In contrast, laboratory findings regarding metabolic parameters, such as RBG, Na, K, and renal function tests (urea and creatinine), did not yield significant differences between examined groups. The absence of statistical significance in RBG levels ($p = 0.334$), Na ($p = 0.167$), K ($p = 0.952$), urea ($p = 0.176$), and creatinine ($p = 0.504$) suggested that these metabolic variables may not play a crucial role in determining outcomes in CO-poisoned patients. This observation may reflect the pathophysiology of CO poisoning, where the primary concern lies in hypoxia and its

downstream neurological effects, rather than in metabolic derangements typically seen in other forms of poisoning or critical illness **(Pan et al., 2019).**

A critical finding from our study is the contrast in oxygen saturation levels between the two studied groups, with non-poor outcome group exhibiting significantly higher O_2 saturation (97.5% vs. 85.3%, $p < 0.001$). **Rose et al., 2017** discussed that CO poisoning reduced the oxygen delivery to tissue besides mitochondrial oxidative phosphorylation, both leading to ischemic and anoxic brain injury. The relationship between oxygen saturation and clinical outcomes is well-established, as hypoxemia can worsen the neurological sequelae.

Understanding the hematological changes associated with acute CO poisoning identifies patients at risk for poor outcomes. In this study, we analyzed CBC parameters in acute CO-poisoned patients and compared those with poor outcomes to those with better prognosis. This practical approach allows targeted interventions, more aggressive treatment strategies, or referral to specialized care, thus optimizing patient management and improving overall prognosis.

Our findings revealed several differences significantly in CBC parameters between the two examined groups, particularly in WBCs counts, neutrophil counts, lymphocyte counts, NLR, and MPV. The WBC count and neutrophil counts in the group with poor outcome were significantly elevated ($p = 0.006$ and $p < 0.001$) respectively, indicating a strong inflammatory response. The increase in WBC count may reflect the human body's attempt to fight hypoxic insults and the following tissue damage. It was noticed that elevated WBC counts have been associated with poor outcomes in various medical conditions **(Yalçın et al., 2023)**.

Neutrophilia may reflect systemic inflammation and oxidative stress, which are known to exacerbate tissue injury and contribute to worse clinical outcomes. Neutrophils play a critical role in initiating and regulating essential immune responses **(Yalçın et al., 2023)**. This occurs because of intravascular neutrophil degranulation following acute CO intoxication, which promotes the production of oxidative agents and accelerates fatty acids oxidation in humans **(Thom et al., 2006)**. The significant increase in neutrophil counts in the poor outcome group may also be indicative of the presence of secondary infections or complications that can arise from the initial poisoning. Furthermore, the activation of neutrophils promotes synthesis of inflammatory cytokines; they serve as indicators of inflammation **(Huang et al., 2018)**.

The significantly higher NLR ($p < 0.001$) in the group of poor outcome align with findings in other critical illnesses. **Xu et al., 2023** said that NLR is a widely accessible biomarker for the immune-inflammatory response and neurological stress. Throughout the inflammatory course, leukocytes and platelets work together with endothelial lining cells. Every leukocyte subtype has a distinct function in both inflammatory and immune processes and each play in different way **(Moon et al., 2019)**.

On the other hand, the lymphocyte count was significantly reduced in the poor outcome group ($p = 0.006$). Lymphopenia in the situation of acute illness is a well-known phenomenon and is often associated with a worse prognosis. The decrease in lymphocyte counts may reflect an adaptive response to stress or may be indicative of a more severe systemic inflammatory response. **Xu et al., 2023** discussed that CO-induced cerebral injury triggers the activation of the hypothalamic-pituitary-adrenal axis, resulting in impaired lymphocyte activation and a subsequent decrease in lymphocyte levels. **Yalçın et al., 2023** revealed that lymphocytopenia reflects the elevated release of catecholamines and corticosteroids owing to acute brain lesions.

The mean platelet volume was significantly worse in the group with poor outcome ($p < 0.001$). MPV is a measure of platelet size and is associated with platelet activity and function. On the contrary, **Karabacak et al, 2014** found that platelet counts and MPV were significantly higher in patients with CO poisoning ($P = 0.01$). Increased MPV values may suggest that

patients with CO poisoning are at in danger for thromboembolic and cardiovascular complications, as a result of platelet activation. Activated platelet response, which could be a consequence of the inflammatory process prompted by CO intoxication. Additionally, platelets contribute by secreting various inflammatory agents which alter leukocyte and endothelial reaction to different inflammatory triggers **(Thomas and Storey, 2015)**.

In contrast, hemoglobin, RBCs count, hematocrit, monocyte count, and platelet count did not differ significantly between groups. The same result was reported by **Umahi-Ottah et al., 2022** who found no significant difference in monocyte counts in control and CO exposed groups.

The mean hemoglobin levels and hematocrit in the poor outcome group were slightly less than in the group of non-poor outcome, yet this difference did not extent to be a statistically significant ($p = 0.064$). A decrease in hematocrit is caused by endogenous dilution which is a transfer of interstitial fluids toward the blood vessels (**Ozturka et al., 2015).**

The current results differed from those of other researchers who have indicated an increase in both hemoglobin levels and hematocrit values in acutely CO-poisoned patients. The variations in results could be linked to either dosage or time **(Lee et al., 1994)**.

Hemoglobin levels may not be the most reliable indicator of severity in CO poisoning, as the primary pathology involves hypoxia rather than anemia. In clinical practice, although hemoglobin levels can offer some insight into the overall oxygen-carrying capacity, they may not accurately indicate the extent of tissue hypoxia resulting from CO binding to hemoglobin. **Lee et al., 1994** reviewed the results of sequential CBC's. They found that levels of hemoglobin and hematocrit rose only initially, possibly due to dehydration and hemoconcentration rather than hypoxia.

CONCLUSION

Finally, it could be concluded that the key predictors of poor outcomes in acute CO poisoning cases were prolonged duration of CO exposure (>2 hours), elevated COHb levels (>15%), GCS ≤13, an oxygen saturation ≤92%. Regarding laboratory parameters, neutrophil count $>6.2 \times 10^3$, lymphocyte count $\leq 2.8 \times 10^3$, neutrophil-to-lymphocyte ratio >2.3 and platelet volume (<8.5) all demonstrate strong predictive power. High sensitivity and specificity across these parameters validate their use in clinical settings for early detection of critical cases. These findings emphasize the necessity for an integrated approach, combining clinical observations and laboratory testing, for optimal management and prognosis of CO poisoning.

These hematological markers emphasize the importance of targeted monitoring and management strategies for high-risk patients. Conversely, traditional vital signs and metabolic parameters may not serve as reliable prognostic indicators.

LIMITATIONS

The current study is limited by the small sample size and observational nature.

RECOMMENDATIONS

1- Future research should focus on longitudinal studies to assess the temporal changes in these CBC parameters following CO exposure and their relationship with clinical outcomes over time.

2- Additionally, larger multicenter studies are necessary to confirm these findings and discover the underlying mechanisms that drive the observed hematological changes in CO poisoning.

3- The relationship between MPV and clinical outcomes in CO poisoning is an area suitable for further exploration, as it may provide additional prognostic information.

CONFLICTS OF INTEREST

There are no conflicts of interest.

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الملخص العربي

المؤشرات الدموية كعوامل تنبؤيه للنتائج في حالات التسمم الحاد بأول أكسيد الكربون

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الخلفيه: يعتبر أول أكسيد الكربون (CO) القاتل الصامت تهديدًا صحيًا كبيرًا في علم السموم السريري الذي يمكن أن يؤدي إلى عقابيل عصبية أو حتى الموت. من بين الناجين من التسمم بأول أكسيد الكربون، قد يتعرض ١٥-٤٠% منهم لآثار عصبية ونفسية عصبية طويلة المدى تؤدي إلى تلف الدماغ. نسبة العدلات إلى الخلايا الليمفاوية (NLR) هي علامة التهابية مشتقة من الدم الطرفي، مما يشير إلى التوازن بين الالتهاب الجهازي والاستجابة المناعية.

الاهداف: الهدف من هذا العمل هو دراسة المؤشرات الدموية كمتنبئات بالنتائج لدى مرضى التسمم الحاد بأول أكسيد الكربون الذين تم قبولهم في مركز مكافحة السموم بجامعة طنطا، مصر.

طرق الدراسة: تم جمع البيانات من السجلات الطبية بما في ذلك المعلومات الديموغرافية، والأعراض في وقت الدخول، وتخطيط رسم القلب الكهربائي (ECG) والاختبارات المعملية (تعداد الدم الكامل مع اختبارات وظائف الكلى والكبد التفاضلية، وجلوكوز الدم العشوائي وغازات الدم الشرياني). تم قياس مستويات الكربوكسي هيموجلوبين باستخدام قياس التأكسج. تمت دراسة استخدام العلاج بالأكسجين عالي الضغط (HBO_2) والقبول في وحدة العناية المركزة وإجمالي مدة الإقامة في المستشفى ونتائج المتابعة.

النتائج: أرتبطت النتائج السيئة للمرضى الذين يعانون من التسمم الحاد بأول أكسيد الكربون بشكل ملحوظ إحصائيًا مع سرعه ضربات القلب، دخول وحدة العناية المركزة، البقاء في المستشفى لفترة أطول، انخفاض GCS، انخفاض تشبع O2، وارتفاع مستوى COHb (قيمة P <٠,٠٠٥). و فيما يتعلق بتعداد الدم الكامل (CBC)، تم ربط الزيادة الكبيرة في عدد كرات الدم البيضاء (WBC) بشكل ملحوظ، متوسط عدد العدلات، نسبة العدلات إلى الخلايا الليمفاوية (NLR) بالنتائج السيئة. على العكس من ذلك، تم الكشف عن انخفاض ملحوظ في عدد الخلايا الليمفاوية وحجم الصفائح الدموية. استنادًا إلى منحنيات ROC، أظهر NLR أهم مؤشر مدروس للنتائج الضعيفة (حساسية ١٠٠,٠%، ونوعية ١٠٠,٠%، ومنطقة تحت المنحنى ١,٠٠)، في حين أظهر عدد الخلايا الليمفاوية أضعف قدرة تشخيصية (حساسية ٨٠,٠%، ونوعية ٦٨,٩%، و٠,٧٩٠). المساحة تحت المنحنى).

الاستنتاج: صوره الدم الكامله هو اختبار معملي بسيط وفعال من حيث التكلفة حيث يتم إجراؤه بشكل روتيني في معظم المختبرات. يؤدي الجمع بين بيانات صوره الدم الكامله والنتائج السريرية إلى تعزيز القدرة على التنبؤ بالنتائج في التسمم الحاد بأول أكسيد الكربون وتحديد المرضى الذين يحتاجون إلى دخول وحدة العناية المركزة والعلاج بـ HBO_2. كان NLR مؤشرا هاما للنتائج السريرية السيئة للتسمم بأول أكسيد الكربون.