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The Validity of Poisoning Severity Score in Acute Carbon Monoxide Intoxicated Patients

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Authors' contributions

This work was carried out in collaboration between all authors. All authors read and approved the final manuscript.

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ABSTRACT

Background and Objective: The poisoning severity score is a standardized and generally applicable scheme for grading the severity of poisoning. It allows a qualitative evaluation of morbidity and facilitates comparability of data. The aim of this study was to evaluate the validity of the poisoning severity score in patients with acute carbon monoxide poisoning as a result of their descriptive, laboratory, clinical data and outcome.
Subjects and Methods: An observational prospective study design was used in the data collection process for eighty Co poisoned patients who presented to Poison Control Center (PCC), Ain Shams University Hospital, Egypt over six months. Patients with coronary artery disease or other known heart disease, patients with renal failure as well as smoker subjects were excluded. The patients were divided into 3 grades according to the poisoning severity score (PSS) which was applied to all patients in the present study at the emergency department. Also a group consisted of twenty apparently healthy nonsmoker volunteers (of matched age and sex) were served as a

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control group. Arterial blood gases (ABG), carboxyhemoglobin level (COHb), random blood sugar, serum of sodium (Na), potassium (K), alanine aminotransferase (ALT), serum creatine phosphokinase (CPK), serum urea and creatinine, hematological parameters (red blood cells, white blood cells and hemoglobin), serial cardiac markers (serum of aspartate aminotransferase (AST), creatine kinase-MB, lactate dehydrogenase (LDH), and cardiac troponin-I (cTnI) quantitative determination and ECG however brain CT scan was done only for cases who admitted to ICU.

Results: Higher mean delay time, tachycardia, bradypnea, respiratory distress, pulmonary edema, lowest mean GCS, hypernatremia, leukocytosis, acidosis, hyperglycemia, high levels of CPK- MB, AST and cTnI, ECG changes, abnormal CT (infarction and brain edema) at presentation and the need of mechanical ventilation with hyperbaric oxygen showed significant relation with higher PSS and adverse outcome.

Conclusion: Age, delay time, PSS grade, Glucose, AST, CPK-MB and Cardiac Troponin I are significant predictors of outcome. These results indicated that those variables are the most important factors for determining the outcome within the limitations of our study. So the results of the present study revealed that PSS is reliable severity score for diagnosis, management and prognosis of COP cases especially with the added modifications in the form of laboratory, ECG and brain CT scan.

Keywords: CO poisoning; poisoning severity score; descriptive; laboratory; clinical data; outcome.

1. INTRODUCTION

Carbon monoxide (CO) is the leading cause of poisoning mortality in the United States. It may be responsible for more than half of all fatal poisonings worldwide, an estimated 5000 to 6000 people die in the United States each year as a result of CO exposure [1].

Disease severity among most patients in the intensive care unit (ICU) lies between two extremes (severely ill and excellent prognosis). Scoring systems represent classification systems or point systems which have been designed for making quantitative statements regarding the severity of a disease, its prognosis, and its course. Furthermore, scores may serve the purposes of assessing therapies, quality control and quality assurance, and of an economic evaluation of intensive care. Generally, one would recommend only using scores which have been strictly tested for their reliability, validity and practicability [2].

Severity scores have been used to assess the outcome of critically ill patients transferred from other institutions, compared to those admitted directly from the emergency department or ward. As an example, one study found that transferred patients were independently at increased risk of mortality [3].

The poisoning severity score is a standardized system for scoring clinical signs and symptoms due to poisoning. A standardized and generally applicable scheme for grading the severity of poisoning allows a qualitative evaluation of morbidity and facilitates comparability of data. Working from a simple grading scale proposed by the European Association of Poisons Centers and Clinical Toxicologists, a poisoning severity score has been developed jointly with the International Program on Chemical Safety and the European Commission.

The poisoning severity score has been elaborated tested and gradually revised during a project running 1991-1994. Fourteen Poison centers from various countries have participated, and 80% of concordance between centers to grading the poisonings was reported in this study. The poisoning severity score grades severity as (0) none, (1) minor, (2) moderate, (3) severe, and (4) fatal poisoning [4]. It is intended to be an overall evaluation of the case, taking into account the most severe clinical features. Use of the Poisoning Severity Score normally requires a follow-up of all cases, but may be used on admission or other times during the course of poisoning if this is clearly stated when data are presented [5].

Then Patch, performed severity grading in all cases using both the poisoning severity score (PSS) and special grading scales developed by the poison control centers in Krakow. The poisoning severity score is assessing severity on the basis of observed clinical signs and symptoms (at their maximum) but does not take into account potential risks or plasma serum concentrations.

The Krakow scales include both clinical symptoms on admission and results of toxicological analysis. Overall concordance was announced 72.7%, and lack of concordance was most evident for ethanol and carbon monoxide toxicities. The less satisfying concordance for carbon monoxide cases was mainly caused by discrepancies in the evaluation of neurological symptoms also the blood lactate concentration, COHb level, duration of exposure and patient's age are considered in the Krakow scale but not in PSS. In conclusion, authors recommended some modifications and additional criteria may be necessary, especially for some specific poisonings such as carbon monoxide [6]. Another study was conducted by Jung, to see if the poisoning severity score on arrival at the emergency department might be used as a prognostic factor and, to see if there are any other factors that might be used to deciding on treatment plans and whether to hospitalize or discharge a patient. In cases of high initial poisoning, the final poisoning severity score was with statistical significance, much higher than it was in cases of low initial poisoning. Also in cases of high final poisoning severity scores, the mechanical ventilation rate and the death rate were higher than they were in cases of low final poisoning severity scores, and this difference was statistically significant. In cases of high final poisoning severity scores, mean age was older, and the intentional poisoning rate, the hemodialysis / hemoperfusion rate, the base excess, and the initial poisoning severity score were higher than in cases of low final poisoning severity score. The author concluded that the initial poisoning severity can be a useful factor for giving a prognosis and for deciding on hospitalization and on a therapeutic plan. Also, concluded that multiple variables, such as the patient's age, the type of toxic material, the cause of poisoning, and the base excess are significant factors that can complement the initial poisoning severity score in deciding on a hospitalization and therapeutic plan. Consequently, early evaluation of the type of toxic material and early measurement of the initial poisoning severity score and the variables mentioned above are of utmost importance in formulating a prognosis and deciding on the need for hospitalization [5].

The aim of this study was to evaluate the validity of the poisoning severity score in patients with acute carbon monoxide poisoning as a result of their descriptive, laboratory, clinical data and outcome.

2. METHODOLOGY

An observational prospective study design was used in the data collection process. The cases of the present study were collected during the period extending from 1-11- 2007 to 30-4- 2008. This study included eighty CO poisoned patients, 42 males and 38 females and their ages ranged between 3 to76 years.

The diagnosis was confirmed by serum level of carboxyhemoglobin on admission. The cases under study were chosen to have no past history of neurological, cardiovascular, gastrointestinal, respiratory or renal disorders. Those with risk factors such as hypertension, diabetes and smoking were also excluded from this study to exclude their effects. Also a group consisted of twenty apparently healthy nonsmoker volunteers (of matched age and sex) was served as a control group. The patients were divided into 3 grades according to the poisoning severity score (PSS) which was applied to all patients in the present study at the emergency department. The clinical grading of CO poisoned patients into mild, moderate, severe was performed according to Tomaszewski [7].

-Grade 1 (minor) included 29 patients with mild, transient and spontaneously resolving symptoms (presented with headache, nausea, vomiting, dizziness, blurred vision).
-Grade 2 (moderate) included 21 patients with pronounced or prolonged symptoms (presented with confusion, syncope, weakness, chest pain, dyspnea, tachycardia, tachypnea and rhabdomyolysis).
-Grade 3 (severe) included 30 patients with severe or life threatening symptom (presented with seizures, coma, arrhythmias, hypotension, myocardial ischemia, cardiac arrest, non-cardiogenic pulmonary edema) [7].

To strengthen the poisoning severity score adopted in the present study and to make it more reliable in achieving accurate diagnosis cases of acute CO poisoned cases, the following investigations were added: Arterial blood gases ABG), carboxyhemoglobin level (COHb), random blood sugar, serum of sodium (Na), potassium (K), alanine aminotransferase (ALT), serum creatine phosphokinase (CPK), serum urea and creatinine, hematological parameters (red blood cells, white blood cells and hemoglobin), serial cardiac markers (serum of aspartate aminotransferase (AST), creatine kinase-MB, lactate dehydrogenase (LDH), and cardiac

troponin-I (cTnI) quantitative determination and ECG however brain CT scan was done only for cases who admitted to ICU.

As regards ethical consideration, written informed consent was obtained from patients or from their next kin. Outcome of the acutely CO intoxicated patients involved in the present study were described as full recovery, in-hospital mortality and development of in- hospital major complications (IHMC). IHMC were described as cardiac (myocardial infarction and arrhythmias causing hemodynamic instability), neurological (stroke, encephalopathy and seizure), respiratory (non-cardiogenic pulmonary edema), rhabdomyolysis. Treatment modalities were also assessed.

2.1 Statistical Analysis

Qualitative data were presented as frequencies and percentages. Chi-square (x 2) test was used for studying the comparisons between different qualitative variables. Quantitative data were presented as minimum, maximum, means and standard deviation (SD) values. Student's t-test was used for comparisons between means of two groups. One way ANOVA (Analysis of Variance) was used to compare between means of more than two groups. Duncan's test for pair-wise comparisons was used to determine significant differences between means when ANOVA test is significant. Kruskal-Wallis test was used to compare between non-parametric data of more than two groups. Mann-Whitney U test was used in the procedure of pair-wise comparison when Kruskal-Wallis test renders significant differences between the groups. The significance level was set at $P \leq 0.05$. Statistical analysis was performed with SPSS 16.0® (Statistical Package for Scientific Studies) for Windows.

3. RESULTS

Table 1 showed that, there was a statistically significant difference between temperatures in the three PSS grades and control group. Grade 1 patients showed the highest percentage of cases with normal temperature (93.1%) while grade 3 groups showed the highest percentages of cases with hyperthermia followed by grade 2 which had the highest percentage of hypothermia.

Table 2 showed that, there was a statistically significant difference between pulse in the three grades of PSS and control group. Grade 1 patients showed the highest percentage of cases (75.9%) with normal pulse followed by grade 2 (52.4%). Grade 3 group showed the highest percentage of cases with tachycardia and bradycardia (70% and 20% respectively).

Table 3 showed that, there was a statistically significant difference of blood pressure in the three grades and control group. Grade 1 patients showed higher percentage of cases (89.7%) with normal blood pressure while grade 3 patients showed higher percentages of cases with hypotension followed by grade 2.

Table 1. Temperature in the studied acute CO poisoned patients with the three PSS grades and control group

Groups	Grade 1 (No.= 29)		Grade 2 (No. = 21)		Grade 3 (No. = 30)		Control (No.= 20)		*P*-value
Temperature	**Frequency**	**%**	**Frequency**	**%**	**Frequency**	**%**	**Frequency**	**%**	
Normal (No.= 77)	27	93.1	12	57.1	18	60	20	100	
Hypothermia (No.= 14)	1	3.4	6	28.6	7	23.3	0	0	**0.003***
Hyperthermia (No.= 9)	1	3.4	3	14.3	5	16.7	0	0	

**: Significant at P ≤ 0.05 No.: number of cases PSS: poisoning severity score*

Table 2. Pulse in the three PSS grades of acute carbon monoxide poisoned cases and control group

Groups	Grade 1 (No. = 29)		Grade 2 (No. = 21)		Grade 3 (No. = 30)		Control (No. = 20)		*P*-value
Pulse	**Frequency**	**%**	**Frequency**	**%**	**Frequency**	**%**	**Frequency**	**%**	
Bradycardia (No.= 7)	0	0	1	4.8	6	20	0	0	
Tachycardia (No.= 37)	7	24.1	9	42.9	21	70	0	0	**<0.001***
Normal (No.= 56)	22	75.9	11	52.4	3	10	20	100	

**: Significant at P ≤ 0.05, No.: number of cases*

Table 4 showed that, there was a statistically significant difference between respiratory rates in the three PSS grades and control group. Grade 2 showed higher percentage of cases (38.1%) with normal respiration followed by grade 1(20.7%), while grade 3 showed the highest percentage of cases (80%) with tachypnea followed by grade 1 (75.9%). Grade 3 showed the highest percentage of cases with bradypnea (13.3%).

Table 5 showed the relation between PSS grades and other respiratory manifestations in the studied acute CO poisoned cases. The highest significant percentages of cases with respiratory distress (86.7%), pulmonary edema (75%) and that need mechanical ventilation (83.3%) were recorded in grade 3.

Table 6 showed the relation between PSS grades and neurological manifestations in the studied acute CO poisoned cases. Patients of grade 1 showed the highest percentage of headache (50%), Flu-like symptoms (66.7%) and dizziness (51.7%) compared to grade 2 and 3. Grade 3 showed the highest percentage of convulsions (53.8%) and agitation (64.3%) compared to grade 2 and 1. Grade 2 showed the highest percentage (50%) of blurring of vision compared to grade 1 and 3.

Table 7 showed that, grade 1 patients had significant highest mean GCS (13.6 ± 1.8); this was followed by grade 2 patients (9.1 ±3.5), while grade 3 patients showed the significant lowest mean GCS (6.7± 2.7).

Table 8 above showed that, grade 1 patients had highest significant percentage of nausea (55.6%). There is no statistical significant difference between all grades in vomiting, diarrhea and abdominal pain.

Table 3. Blood pressure in the three PSS grades in the studied acute CO poisoned cases and control group

Groups	Grade 1 (No. = 29)		Grade 2 (No. = 21)		Grade 3 (No. =30)		Control (No. = 20)		*P*-value
Blood pressure	**Frequency**	**%**	**Frequency**	**%**	**Frequency**	**%**	**Frequency**	**%**	
Hypotension (No.= 16)	2	6.9	4	19	10	33.3	0	0	
Hypertension (No.= 3)	1	3.4	1	4.8	1	3.3	0	0	**0.033***
Normal (No.= 81)	26	89.7	16	76.2	19	63.3	20	100	

*No.: number of cases, *: Significant at P ≤ 0.05*

Table 4. Respiratory rates in the studied acute CO poisoned cases with the three PSS grades and control group

Groups	Grade 1 (No. = 29)		Grade 2 (No. = 21)		Grade 3 (No. = 30)		Control (No. = 20)		*P*-value
Respiratory rates	**Frequency**	**%**	**Frequency**	**%**	**Frequency**	**%**	**Frequency**	**%**	
Tachypnea (No.= 59)	22	75.9	13	61.9	24	80	0	0	
Bradypnea (No.= 5)	1	3.4	0	0	4	13.3	0	0	**<0.001***
Normal (No.= 36)	6	20.7	8	38.1	2	6.7	20	100	

*No.: number of cases, *: Significant at P ≤ 0.05*

Table 5. The relation between PSS grades and other respiratory manifestations in the studied acute CO poisoned cases

PSS grades	Grade 1 (No. = 29)		Grade 2 (No. = 21)		Grade 3 (No. = 30)		*P*-value
Respiratory manifestations	**Frequency**	**%**	**Frequency**	**%**	**Frequency**	**%**	
Need of mechanical ventilation (No. = 31)	0	0	6	17.6	25	83.3	**<0.001***
Respiratory distress (No. = 15)	0	0	2	13.3	13	86.7	**<0.001***
Pulmonary edema (No. = 16)	0	0	4	25	12	75	**0.001***

*P>0.05 = non-significant, No.: number of cases, *: Significant at P ≤ 0.05*

Table 6. Neurological manifestations in the studied acute CO poisoned cases with the three PSS grades

Neurological manifestations	PSS grades						
	Grade 1 (No.= 29)		Grade 2 (No.= 21)		Grade 3 (No.= 30)		*P*-value
	No.	%	No.	%	No.	%	
Headache (No.= 54)	27	50	13	24.1	14	25.9	0.001*
Flu-like (No.= 21)	14	66.7	5	23.8	2	9.5	0.001*
Dizziness (No.= 29)	15	51.7	8	27.6	6	20.7	0.039*
Confusion (No.= 32)	9	28.1	9	28.1	14	43.8	0.450
Convulsions (No.= 13)	0	0	6	46.2	7	53.8	0.011*
Agitation (No.= 14)	1	7.1	4	28.6	9	64.3	0.027*
Blurred vision (No.= 10)	2	20	5	50	3	30	0.177

*P>0.05 = non-significant, *: Significant at P ≤ 0.05, No.: number of cases*
PSS: poisoning severity

Table 7. Glasgow coma scale in the studied acute CO poisoned cases with the three PSS grades

Glasgow coma scale (GCS)	PSS grades						
	Grade 1 (No.= 29)		Grade 2 (No.= 21)		Grade 3 (No.= 30)		*P*-value
	Mean	SD	Mean	SD	Mean	SD	
	13.6 a	1.8	9.1 b	3.5	6.7 c	2.7	**<0.001***

**: Significant at P ≤ 0.05, Means with different letters are statistically significantly different according to Duncan's test*
No.: number of cases GCS: Glasgow Coma scale; PSS: poisoning severity score

Table 8. Gastrointestinal manifestations in the studied acute CO poisoned cases with the three PSS grades

GIT manifestations	PSS grades						
	Grade 1 (No.= 29)		Grade 2 (No.= 21)		Grade 3 (No.= 30)		*P*-value
	No.	%	No.	%	No.	%	
Nausea (No. = 36)	20	55.6	8	22.2	8	22.2	0.004*
Vomiting (No. =60)	23	38.3	17	28.3	20	33.3	0.408
Diarrhea (No. = 15)	7	46.7	5	33.3	3	20	0.299
Abdominal colic (No. = 21)	10	47.6	7	33.3	4	19	0.126

*P>0.05 = non-significant, *: Significant at P ≤ 0.05 No.: number of cases, PSS: poisoning severity score*

Table 9 showed the laboratory investigations in the studied acute CO poisoned patients with the three PSS grades and control group. With regards to pH, there was no statistical significant difference between patients within grade 1, grade 2 when compared with control groups. Grade 3 showed significant lowest mean pH (7.1±0.1) (acidosis) when compared with grade 1 (7.4±0.06), grade 2 (7.4±0.05) and control group (7.4±0.02). With respect to PaO2, grade 1 showed significant highest mean PaO2, (57.9±60.1) when compared with grade 2(42.4±39.1) and grade 3 (46.3±37.4). There was no statistical significant difference between grade 2 and grade 3 that showed significant lowest means PaO2. Concerning HCO3, grade 1 showed significant highest mean HCO3 (23.9±9.6) compared with grade 2 (20.6±2.1) and grade 3(20.7±5) that showed significant lowest mean HCO3. Regarding COHb%, there was no statistically significant difference between grades 1, 2, 3 but they had statistically significant highest mean COHb (21.2±8.2 & 19.4±9.5 and 22.4±7.4 respectively) when compared with control group (0.38±0.2).As regards glucose, grade 3 patients showed significant highest mean glucose level (131.8±48.3) (hyperglycemia) when compared with grade 1 (120±31.3), grade 2 (114.3±34.2) and control group (85.1±7). There was no significant difference between patients with grade 1 and grade 2 whom showed lower values. Grade 3 patients showed significant highest means Na level (144± 6.3) (hypernatremia) followed by grade 2 (138±4). There was no statistical significant difference between patients with grade 1(135.4±5.8) and control group (136±1.2). For CPK, there was no statistical significant difference between grade 1 and grade 3 patients, whom showed significant highest means of CPK

(797.7±1018.4&831.3±1083.5 respectively), followed by grade 2 (413.4±628.7). As regards WBCs, grade 3 patients showed significant highest mean WBCs (11.8±5.1) (leukocytosis) when compared with grade 1 (9.6±3.2), grade 2 (9.8±4.3) and control group (6.8±1.5). There was no significant difference between patients with grade 1 and grade 2 whom showed lower values. Concerning cardiac enzymes involved in this study, there was no statistically significant difference between patients in PSS grades 1, 2 and 3 which showed the statistically significant highest values with AST, LDH and CPK- MB when compared with control group. Regarding cardiac troponin I, grade 3 showed the highest values (3.7±0.5) when compared with grade 2 (1.5±0.9), grade 1 (1.2±0.8) and control group (0.7±0.3). There was no statistically significant difference between patients with PSS grade 1, 2, while there is statistically significant difference when compared with control group.

Table 10 showed ECG findings in the studied acute CO poisoned patients in the three PSS grades. Grade 1 showed the highest statistical significant percentage of cases with normal ECG recordings (82.8%) as compared to grade 2 and 3. Grade 3 showed the lowest percentage of cases with normal ECG (23.3%) and the highest statistical significant percentage of cases with ECG changes (67.7%).Grade 1 showed the lowest percentage of cases with ECG changes (17.2%).

Histogram 1 showed the relation between PSS grades and CT findings in 35 acute CO poisoned cases admitted to ICU. The CT findings of them were in the form of acute hemorrhage, infarction and brain edema. It was observed that grade 3 showed the highest percentage of CT abnormalities which were acute hemorrhage in 5 cases, infarction in 5 cases and brain edema in 19 cases.

Table 9. Laboratory investigations in the studied acute CO poisoned patients with the three PSS grades and control group

Groups	Grade 1 (No. = 29)		Grade 2 (No. = 21)		Grade 3 (No. = 30)		Control (No. = 20)		*P*-value
Parameters	**Mean**	**SD**	**Mean**	**SD**	**Mean**	**SD**	**Mean**	**SD**	
pH	7.4 [a]	0.06	7.4 [a]	0.05	7.1 [b]	0.1	7.4 [a]	0.02	0.051*
$PaCO_2$ mmHg	40	8.8	39.3	5.8	39.2	9.7	39	2.9	0.969
PaO_2 mmHg	57.8 [b]	60.1	42.4 [c]	39.1	46.3 [c]	37.4	89.2 [a]	6.7	<0.001*
HCO_3(mmol/L)	23.9 [a]	9.6	20.6 [b]	2.1	20.7	5	25.4 [a]	2	0.017*
COHb%	21.2 [b]	8.2	19.4 [b]	9.5	22.4 [b]	7.4	0.38 [a]	0.2	<0.051*
Glucose(mg/dl)	120 [b]	31.2	114.3 [b]	34.2	131.8	48.3	85.1 [c]	7	<0.001*
Na(mEq/L)	135.4 [b]	5.8	138 [a]	4	144 [c]	6.3	136. [b]	1.2	0.017*
K(mEq/L)	4	1	4	0.6	3.9	0.6	3.7	0.1	0.578
ALT(U/L)	48.8	36.6	42.3	30.2	52.9	51	26.9	4.8	0.149
CPK(U/L)	797.7 [a]	1018.4	413.4 [b]	628.7	831.3 [a]	1083.5	19.7 [c]	2.6	<0.001*
Urea(mg/dl)	37.7	16.2	33.1	12.7	36	17.8	26.6	8.3	0.066
Creatinine(mg/dl)	0.92	0.4	0.94	0.4	0.99	0.4	0.80	0.2	0.365
WBC_S	9.6 [b]	3.2	9.8 [b]	4.3	11.8 [a]	5.1	6.9	1.5	0.001*
RBC_S	4.5	0.9	4.5	0.7	4.7	0.6	4.6	0.2	0.661
Hb%	13.3	2.1	13.9	1.8	12.5	2.5	13.1	1.3	0.123
AST(U/L)	55.8 [a]	46.3	47.7[a]	40	65[a]	47.7	28.2 [b]	3.3	<0.001*
CPK- MB(U/L)	42.4[a]	28.5	51.5[a]	35.8	44.8[a]	26.3	19.7[b]	2.6	<0.001*
LDH(U/L)	330.9[a]	76.2	359.6[a]	59.5	314.9[a]	72.3	254.2 [b]	28.6	<0.001*
Cardiac troponin I (cTnI)(ng / ml)	1.2[a]	0.8	1.5[a]	0.9	3.7 [c]	0.5	0.7[b]	0.3	0.036*

*: *Significant at P ≤ 0.05, Means with different letters are statistically significantly different according to Duncan's test and Mann-Whitney U test*

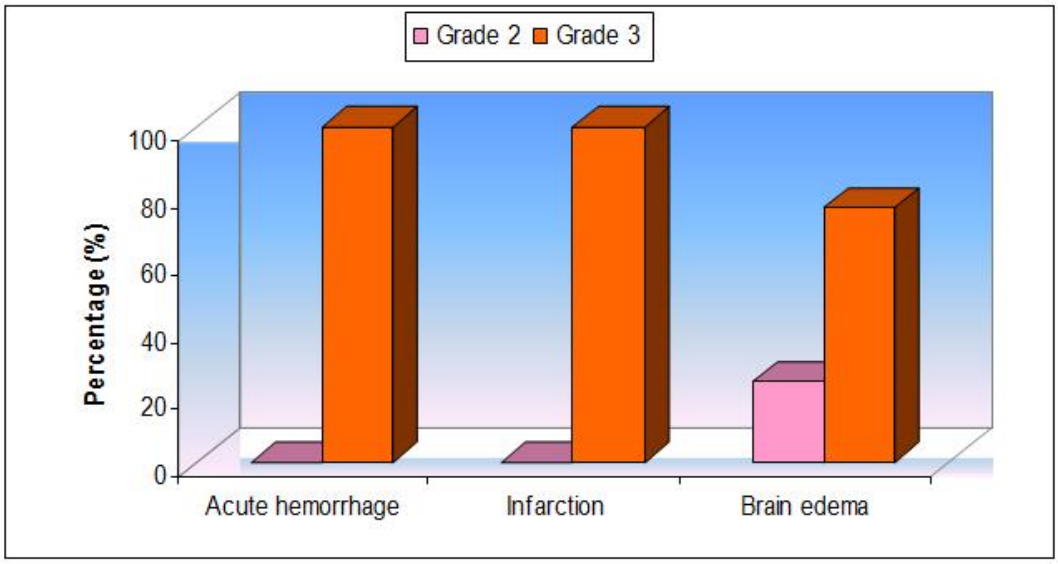
P>0.05 = non-significant, No.: number of cases

SD: standard deviation, PSS: poisoning severity score

Table 10. ECG findings in the studied acute CO poisoned patients in the three PSS grades

PSS grades	Grade 1 (No.= 29)		Grade 2 (No. = 21)		Grade 3 (No. = 30)		*P*-value
ECG	**Number**	**%**	**Number**	**%**	**Number**	**%**	
Normal (No. = 41)	24	82.8	10	47.6	7	23.3	
Changed (No. = 39)	5	17.2	11	52.4	23	67.7	**<0.001***

*No.: number of cases, PSS: poisoning severity score, *: Significant at P ≤ 0.05*

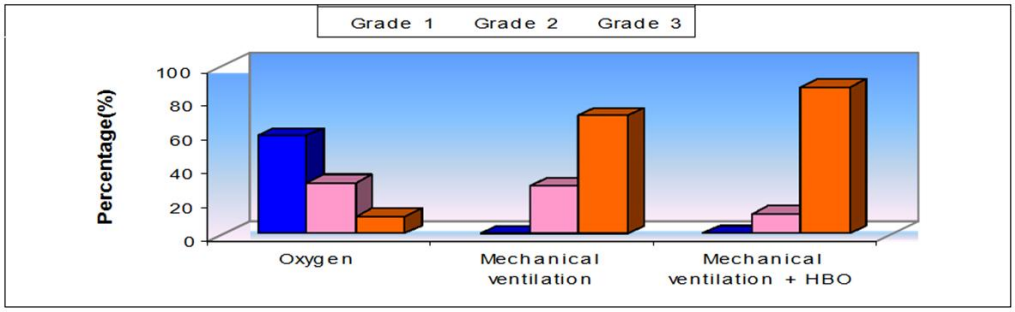


Histogram 1. CT findings and PSS grades in 35 acute CO poisoned cases admitted to ICU

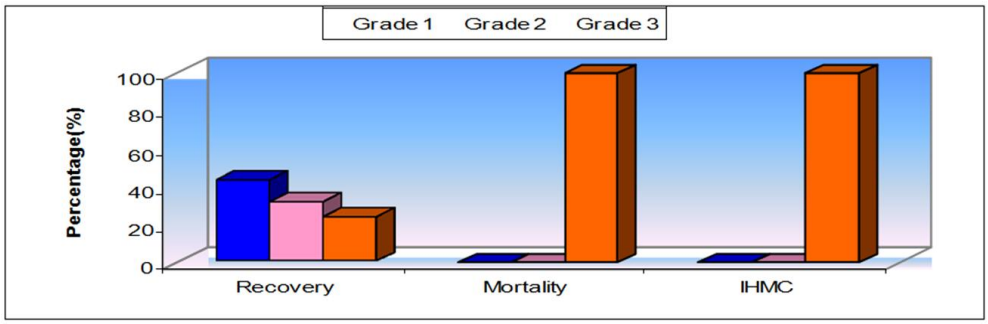
Histogram 2 showed that, 49 patients (53.75%) received oxygen by mask. Twenty-nine patients (59.2%) were in grade 1 PSS and 15 patients (34.1%) were in grade 2, grade 1 patients showed significant higher percentage of cases than grade 2. Nineteen patients (23.75%) required mechanical ventilation. Four patients (21.1%) were in grade 2 PSS and 15 patients (78.9%) were in grade 3, grade 3 patients showed significant higher percentage of cases than grade 2. Seventeen patients (21.25%) required mechanical ventilation and hyperbaric oxygen (HBO). Two patients (11.8%) were in grade 2 PSS and 15 patients (88.2%) were in grade 3, grade 3 patients showed significant higher percentage of cases than grade 2.

Histogram 3 showed that, sixty-six patients were recovered, twenty-nine of them (43.9%) were in grade 1 PSS, 21 patients (31.8%) were in grade 2 and 16 patients (24.2%) were in grade 3. This difference was not statistically significant. Six cases were died while in-hospital major complications (IHMC) occurred in 8 patients. All patients that died and with IHMC had Grade 3 PSS.

Tables 11 and 12 shows, the outcome in acute CO poisoned cases in the present study a special concern was put for studying the mortality and IHMC cases, through the emerged results, the indications of poor prognosis were described as higher mean of ages, higher mean delay time, tachycardia, bradypnea, cyanosis, respiratory distress, pulmonary edema, lowest mean GCS, musculoskeletal edema, tenderness, hypernatremia, leukocytosis, acidosis, hyperglycemia, high levels of CPK-MB, AST and cTnI, ECG changes, abnormal CT (infarction and brain edema) at presentation and the need of mechanical ventilation with hyperbaric oxygen.



Histogram 2. The treatment modalities of the studied acute CO poisoned patients in the three PSS grades



Histogram 3. The relation between PSS grades and outcome in the studied acute CO poisoned cases

3.1 Summary of Mortality Cases (6 cases) Data

Table 11 showed that, the mean age of mortality was 43.7 ± 19.9. 3 of them presented with cyanosis, two with bradycardia and three with tachycardia. Three of them had cardiac arrest. The six cases required mechanical ventilation, 5 cases had respiratory distress and 5 cases had pulmonary edema.

3.2 Summary of In-Hospital Major Complication Cases (8 cases)

Table 12 showed that, the mean age of IHMC was 22.8 ± 8. One of 8 patients presented with bradycardia and seven with tachycardia. The eight cases required mechanical ventilation, 3 cases had respiratory distress and 2 cases had pulmonary edema. The mean GCS was 6.8 ± 2.4. Three of them had rhabdomyolysis, one had weakness and one had asthenia. All cases were in grade III PSS.

Table 13 showed that, to find which variables are significant predictors of outcome, parameter estimates and Wald statistics are computed. The Wald statistic is the square of the ratio of the parameter estimate to its standard deviation. If the significance of Wald statistic is small (i.e. less than 0.05) then the parameter is useful to the model. So, the results of regression analysis showed that age, delay time, PSS grade, Glucose, AST, CPK- MB and Cardiac Troponin I are significant predictors of outcome. These results indicate that those variables are the most important factors for determining the outcome within the limitations of our study.

Table 11. Descriptive table showing variables detected for mortality cases in the studied acute CO poisoned patients

Variables	
Age (mean ± SD)	43.7 ± 19.9 years
Gender (Frequency, %)	
Male	2 (33.3%)
Female	4 (66.7%)
Delay time (mean ± SD)	19.1 ± 29.7 h.
Delay time less or more than 5 hours (Frequency, %)	
Less	3 (50%)
More	3 (50%)
Month 'Season' (Frequency, %)	
January	2 (33.3%)
December	4 (66.7%)
Temperature (Frequency, %)	
Normal	3 (50%)
Hypothermia	3 (50%)
Skin (Frequency, %)	
Normal	3 (50%)
Cyanosis	3 (50%)
Pulse (Frequency, %)	
Normal	1 (16.7%)

Variables	
Bradycardia	2 (33.3%)
Tachycardia	3 (50%)
Blood pressure (Frequency, %)	
Normal	3 (50%)
Hypotension	3 (50%)
CVS manifestations (Frequency, %)	
Cardiac arrest	3 (50%)
Dyspnea	1 (16.7%)
Respiratory rate (Frequency, %)	
Bradypnea	3 (50%)
Tachypnea	3 (50%)
Respiratory manifestations (Frequency, %)	
Mechanical ventilation	6 (100%)
Respiratory distress	5 (83.3%)
Pulmonary edema	5 (83.3%)
Neurological manifestations (Frequency, %)	
Headache	3 (50%)
Dizziness	2 (33.3%)
Confusion	3 (50%)
Convulsions	1 (16.7%)
GCS (mean ± SD)	9.8 ± 4
Gastrointestinal manifestations (Frequency, %)	
Nausea	2 (33.3%)
Vomiting	5 (83.3%)
Diarrhea	1 (16.7%)
Musculo-skeletal manifestations (Frequency, %)	
Musculo-skeletal edema	1 (16.7%)
Weakness	2 (33.3%)
Asthenia	1 (16.7%)
Malaise	3 (50%)
Tenderness	1 (16.7%)
Site of admission (Frequency, %)	
Inpatient	1 (16.7%)
ICU	5 (83.3%)
Hospital stay (mean ± SD)	5 ± 6.9 Days
Treatment (Frequency, %)	
Mechanical ventilation Mechanical	3 (50%)
ventilation + HBO	3 (50%)
PSS (Frequency, %)	
Grade III	6 (100%)
ABG (mean ± SD) pH	7.1 ± 0.02
$PaCO_2$ mmHg	42.3 ± 14.5
PaO_2 mmHg	42.1 ± 9.3
HCO_3(mmol/L)	20.2 ± 3.4
COHb%	29.6 ± 12.1
Glucose(mean ± SD) (mg/dl)	140.8 ± 11.1
Na(mean ± SD) (mEq/L)	144 ± 8.6
K(mean ± SD) (mEq/L)	4.1 ± 0.6
ALT(mean ± SD) (U/L)	40.5 ± 40.4
CPK(mean ± SD) (U/L)	609.7 ±330.6
Creatinine(mean ± SD(mg/dl)	1.1 ± 0.6
Urea(mean ± SD) (mg/dl)	40.5 ± 20.5
WBCs(mean ± SD)	13.6 ± 4.9
RBCs(mean ± SD)	4.9 ± 0.8
Hb%(mean ± SD)	12.9 ± 3.1
AST(mean ± SD) (U/L)	43.8 ± 21.3
CPK MB(mean ± SD) (U/L)	75 ± 46.4
LDH(mean ± SD(U/L)	343.5 ± 89.5
Cardiac troponin I (mean ± SD) (ng / ml)	3± 2.2

Table 12. Descriptive table showing variables for in-hospital major complication cases in the studied acute CO poisoned patients

Variables	
Age (mean ± SD)	22.8±8 years
Gender (Frequency, %)	
Male	4(50%)
Female	4(50%)
Delay time (mean ± SD)	4.1±2.4 h.
Temperature (Frequency, %)	
Normal	5 (62.5%)
Hypothermia	1(12.5%)
hyperthermia	2 (25%)
Skin (Frequency, %)	
Normal	7(87.5%)
Cyanosis	1(12.5%)
Pulse (Frequency, %)	
Bradycardia	1 (12.5%)
Tachycardia	7(87.5%)
Blood pressure (Frequency, %)	
Normal	6(75%)
Hypotension	1(12.5%)
hypertension	1(12.5%)
CVS manifestations (Frequency, %)	
Dyspnea	2(25%)
Respiratory rate (Frequency, %)	
Normal	1 (12.5%)
Tachypnea	7 (87.5%)
Respiratory manifestations (Frequency, %)	
Mechanical ventilation	8(100%)
Respiratory distress	3(37.5%)
Pulmonary edema	2(25%)
Neurological manifestations (Frequency, %)	
Headache	3 (37.5%)
Flu-like symptoms	1(12.5%)
Dizziness	2(25%)
Confusion	3(37.5%)
Convulsion	3(37.5%)
Agitation	2(25%)
GCS (mean ± SD)	6.8± 2.4
Gastrointestinal manifestations (Frequency, %)	
Nausea	1(12.5%)
Vomiting	5 (62.5%)
Colic	1 (12.5%)
Musculo-skeletal manifestations (Frequency, %)	
Rhabdomyolysis	3(37.5%)
Weakness	1(12.5%)
Asthenia	1(12.5%)
Site of admission (Frequency, %)	
Inpatient	1 (16.7%)
ICU	5 (83.3%)
Hospital stay (mean ± SD)	10.3±9.9 Days
Treatment (Frequency, %)	
Mechanical ventilation	2(25%)
Mechanical ventilation + HBO	6(75%)
PSS (Frequency, %)	
Grade III	8(100%)
ABG (mean ± SD)	
pH	7.1 ± 0.04
$PaCO_2$ mmHg	36.6±12.8
PaO_2 mmHg	62.9± 53.1
HCO_3(mmol/L)	19.2± 3.7
COHb%	20.4 ± 18.3

Variables	
Glucose(mean ± SD) (mg/dl)	131.6± 18.4
Na(mean ± SD) (mEq/L)	137.5±4.2
K(mean ± SD) (mEq/L)	4 ± 0.8
ALT(mean ± SD) (U/L)	47.2± 22.8
CPK(mean ± SD) (U/L)	1199±1351.5
Creatinine(mean ± SD(mg/dl)	0.9± 0.3
Urea(mean ± SD) (mg/dl)	28.9± 10.4
WBCs(mean ± SD)	12.8± 4.1
RBCs(mean ± SD)	4.5± 0.2
Hb%(mean ± SD)	11.4±2.4
AST(mean ± SD) (U/L)	97.6± 31.9
CPK MB(mean ± SD)(U/L)	38.8± 7.9
LDH(mean ± SD(U/L)	304.8± 64.5
Cardiac troponin I (mean ± SD) (ng / ml)	1.9± 0.6

Table 13. Wald statistics and significant variables in the model

Variable (Parameter)	Wald statistic	*P*-value
Age	19.3	<0.001*
Delay time	21.8	<0.001*
PSS grade	48.9	<0.001*
Glucose	8.2	0.003*
AST	11.2	<0.001*
CPK MB	30.7	<0.001*
Cardiac Troponin I	17.2	<0.001*

**: Significant at P ≤ 0.05*

4. DISCUSSION

As regard temperature, grade 1 patients showed higher percentage of cases with normal temperature (93.1%) while grade 2 and grade 3 showed higher percentages of cases with hypothermia and hyperthermia. Hypothermia actually increases mortality in animals with CO poisoning [7]. As regard the pulse in the present study, the highest percentages of both tachycardia (70%) and bradycardia (20%) were seen in grade 3 patients. For the outcome, a significant percentage of mortality and morbidity in the form of IHMC, were associated with tachycardia seen in grade 3 patients while recovery was met in patients with normal pulse. These results were in agreement with those of Cevik, who reported that tachycardia showed significant relation with higher poisoning severity scores (PSS) and mortality [8].

Meert, reported that tachycardia was one of the most common presenting signs in CO poisoning [9]. Swank, stated that CO-induced sinus tachycardia was reported in a study in Ruby Memorial Hospital in the United States [10]. Aslan found that sinus tachycardia was present in 26.5% of patients with CO poisoning [11].

Hampson and Zmaeff, stated that bradycardia was found among 10 cases out of 18 cases (55.5%) in a study done to examine the outcome of group of patients with extreme CO poisoning in the United States [12]. Gandini reported that, although tachycardia is a common finding in CO poisoning and usually considered as a compensatory response to systemic hypoxia and cardiac dysfunction, yet bradycardia may be present, indicating rhythm disturbances [13].

Hypotension in the present study was detected in 2 cases (6.9%) in grade 1, 4 cases (19%) in grade 2 and 10 cases (33.3%) in grade 3. These results agreed with Raub, who stated that, severe CO poisoning may result in marked hypotension [14]. The results of the present study coincided with those of a study done by Chamberland, who reported, hypotension in a study describing the cardiac dysfunction in acute CO poisoning in the United States [15]. Kourembanas, reported that, CO induced smooth muscle relaxation and vasodilatation of blood vessels as well as inhibition of platelet aggregation. This could be due to activation of guanyl cyclase enzyme and generation of cyclic GMP [16]. Another explanation of hypotension was reported by Yanir who said that hypotension may result from myocardial injury secondary to hypoxia, ischemia, and direct myocardial depressant activity from myoglobin binding, peripheral vasodilatation or a combination of the aforementioned and may persist even after neurologic and metabolic symptoms have resolved [17].

With respect to hypertension it was detected in 3 cases who equally distributed among different PSS grades, this agreed with Meert who observed hypertension in their study to evaluate the clinical characteristics and outcome of patients exposed to CO poisoning [9]. Also,

Koskela observed that the blood pressure was slightly higher in patients exposed to CO poisoning than those who were not exposed in their study on 931 males' foundry workers in Finland to determine the predictors of ischemic heart disease among them [18].

Concerning the respiratory system examination in the present study, grade 3 showed the highest percentage of cases (80%) with tachypnea and (13.3%) bradypnea. The highest significant percentages of cases with respiratory distress, pulmonary edema and that were in need to mechanical ventilation, (86.7% 75% and 83.3% respectively) were recorded in grade 3. Cevik reported that, higher respiratory rate were found in cases categorized in higher PSS grades [8].

These findings also coincided with the results founded by Targosz and Pach, in their study; they stated that, the respiratory tract affection was the second predominant early complication following the toxic myocardial injury in their studied cases comparing the frequency and clinical course of CO Poisonings in Poland [19]. Furthermore, Handa and Tai, found that (8.3%) of their studied CO poisoned patients showed manifestations of acute respiratory distress syndrome in the form of profound dyspnea, intercostal retractions and marked hypoxemia [20].

Regarding to the neurological manifestations in the current study, they were in the form of headache, flue like symptoms, confusion, dizziness, seizures, blurring of vision and agitation, headache was the most outstanding feature presented in fifty-four patients (67.5%). And this coincided with that mentioned by Hampson and Hampson, who described headache as the most commonly reported symptom in acute CO poisoning which was often throbbing, continuous, diffuse and mostly located in the frontal area [21]. In addition, Kao and Nanagas, reported that, headache, particularly frontal and flu-like illness in the winter time with symptomatic cohabitants should raise the suspicion for CO poisoning [22].

In the present study, grade 1 patients showed significant percentages of headache (50%), Flu-like symptoms (66.7%) and dizziness (51.7%) compared to grade 2 and grade 3. Grade 3 showed the highest significant percentage of convulsions and agitation (53.8% and 64.3% respectively) compared to grade 2 and grade 1. Grade 2 patients showed the highest percentage (50%) of blurring of vision compared to grade 1 and grade 3.

Headache induced by CO poisoning may be attributed to reflex vasodilatation secondary to CNS hypoxia as recorded by Ernst and Zibrak [23]. In addition, CO-induced acute brain injury could be another cause of headache in CO exposed patients as described by Gorman who suggested that tissue hypoxia may facilitate the production of partially reduced oxygen species, resulting in reperfusion injury [24].

Regarding grading of the clinical picture by Glasgow coma scale (GCS), in the present study, grade 1 patients had the statistical significant highest mean GCS (13.6 ± 1.8); this was followed by grade 2 patients (9.1 ±3.5). Grade 3 patients showed significant lowest mean GCS (6.7± 2.7).

The above mentioned findings were in agreement with those of Kao and Nanagas, who reported that early neurologic manifestations include dizziness and headache; increasing exposure may produce altered mental status, confusion, syncope, seizure, acute stroke like syndromes, and coma [22]. This also agreed with Cevik, who reported that decreased level of consciousness showed significant relation with higher PSS [8].

Concerning the GIT affection in the presented CO poisoned patients, grade 1 patients had highest significant percentage of nausea (55.6%). These findings agreed with those of Felta-Zaragozano who recorded that, gastrointestinal symptoms such as nausea, vomiting and abdominal pain were observed in children hospitalized for acute CO poisoning, also, stated that vomiting and nausea were considered manifestations of both CNS and gastrointestinal tract involvement due to hypoxic effect of CO poisoning [25].

In the present study, there was no statistical significant difference of pH for patients within grade 1, grade 2 and control groups. Grade 3 showed significant lowest mean of pH (7.1±0.1) (acidosis) when compared with grade 1(7.4±0.06), grade 2 (7.4±0.05) and control group (7.4±0.02). Recovered cases showed the statistical significant highest mean of pH (7.3±0.07). IHMC and mortality cases were associated with acidosis. Concerning HCO3 in the present study, grade 1 showed significant highest mean of HCO3 as compared with grade 2 and grade 3. The significant lowest mean of

HCO3 was showed with grade 3. This agreed with Cevik, who reported that, the pH level of cases in severe PSS grade was significantly lower than that of other grades. There was moderate correlation between pH and PSS. Mean pH of mortal (7.22 ± 0.24) and IHMC cases (7.27 ± 0.09) was significantly lower than that of recovered cases (7.40 ± 0.07) ($p < 0.001$). Initial acidosis is a better predictor than COHb level [8]. Regarding COHb%, in the present study, there was no statistically significant difference between the three studied PSS grades but they had statistically significant highest mean COHb% when compared with control group. The emerged results in the present work find agreement with Hampson and Hauff, who concluded that, despite the fact that statistically significant difference in average COHb measurements were seen with regard to a number of variables, the clinical significance of these differences appeared to be minimal. Moreover, the utility of COHb measurements as predictors of clinical status in CO poisoning was not apparent. At least in part, this likely relates to delay and interval oxygen administration before obtaining COHb measurements [26]. In the present study, grade 3 patients showed significant highest mean glucose level (hyperglycemia) when compared with grade 1, 2 and control group. This agrees with Cevik, who reported that high glucose showed significant relation with higher PSS [8]. Penney, studied unanaesthetized rats exposed to carbon monoxide for 90 min to examine the effect of acute CO poisoning on plasma glucose and neurologic dysfunction. Glucose increased during CO exposure and after room air recovery. Neurologic deficit, behaviorally-assessed after 4 h of recovery, was strongly correlated with glucose increased [27]. Yarar found that hyperglycemia and glycosuria are commonly encountered during CO poisoning. In their study, hyperglycemia was found in 9.5% of patients and was regulated with appropriate insulin and fluid treatment [28]. Concerning serum Na levels in the present study, grade 3 patients showed significant highest means Na level (hypernatremia) followed by grade 2. There was no statistical significant difference between patients with grade 1 and control group. Hypernatremia usually occurs in water deprivation or in persons who cannot obtain sufficient water to replace losses (head trauma, carbon monoxide poisoning or diabetes insipidus). Sodium intoxication may result from excess intakes of NaCl or sodium bicarbonate [29]. As regards WBC in the present study, grade 3 patients showed significant highest mean WBC (leukocytosis) when compared with grade 1, grade 2 and control groups. The above mentioned results find agreement with those of Cevik, who reported that mean leucocyte level of cases categorized in severe grade was higher than that of other grades. Correlation of PSS grades and leucocyte level was powerful ($p < 0.001$, $r = 0.560$). Mean leucocyte level of dead (22.4 ± 5.6 × 103) and IHMC (21.2 ± 5.3 × 103) cases were significantly higher than that of recovered (11.4 ± 4.7 × 103) ($p < 0.001$) [8]. Concerning the relation between the cardiac markers (AST, LDH, CPK MB and cTnI) and PSS grades in the present study, it was found that for AST, LDH and CPK- MB, there was no statistically significant difference between patients with different PSS grades however grade 1, 2, and 3 showed statistically significant highest values when compared with control group. Regarding cardiac troponin I, grade 3 showed the statistically significant highest values when compared with grade 2, grade 1 and control group. Brogan has found that measuring serum CPK- MB was efficient to predict a wide range of short term adverse events in patients presented with chest pain [30]. Collinson stated that, the diagnostic criteria for myocardial injury have been classically based on the triad of history, ECG and measurement of cardiac enzymes. The choice of enzymes has been dictated by the evolution of laboratory techniques, starting from measuring serum AST and progressed to serum CPK- MB levels [31]. Zhang, in their study to detect cardiac injury in 62 cases with acute CO poisoning stated that determination of serum myocardial enzymes was helpful to detect myocardial injury. The abnormal rate of ECG was less than (10%), LDH was (58.3%) and CPK- MB was (37.1%) [32]. In addition, Gandini, stated that these conventional markers possess low specificity as indicators of cardiac necrosis in patients with conditions such as skeletal muscle necrosis or multiple organ failure which may occur in patients with severe CO poisoning [33]. With respect to treatment modalities in relation to PSS, COHb and outcome in the present study, grade 1 patients showed the highest significant percentage of cases that received oxygen by mask (59.2%). Grade 3 patients showed the highest significant percentage of cases that required mechanical ventilation (71.4%) and that required mechanical ventilation with hyperbaric oxygen (88.2%). The emerged results concerning treatment modalities in the current work were in agreement with those of Cevik, who reported, one hundred sixty-eight cases (92.3%) received oxygen by mask. Nine

cases (4.9%) required endotracheal intubation. Four of nine intubated cases did not receive HBO therapy. Ten cases (5.4%) received HBO therapy (with mask or intubated). Minor cases were mostly treated with oxygen by mask. Mean COHb levels of cases by treatment modalities (mask and oxygen, intubated with HBO and HBO) were different (20.9 ± 12.0, 32.5 ± 10.8 and 35.0 ± 16.5%, respectively, p = 0.001). All cases who received HBO were categorized moderate (one case) and severe (nine cases). In treatment, mortality was 0.6% (one case) in mask and oxygen group, 20% (two cases) in HBO group and 75% (three cases) in intubated with HBO group. Complications were 1.8% (three cases) in mask and oxygen group, 20% (two cases) in HBO group and 25% (one case) in intubated with HBO group. There is no significant correlation between treatment modalities and outcome [8]. The relation between PSS grades and outcome as revealed through the results of the present study revealed that, sixty-six patients were recovered, twenty-nine of them (43.9%) were in grade 1 PSS, 21 patients (31.8%) in grade 2 and 16 patients (24.2%) in grade 3. This difference was not statistically significant. Six cases were died while in-hospital major complications (IHMC) occurred in 8 patients (10%). All patients that died and those with IHMC were in Grade 3 PSS. Mortality was 7.5%. The 20% of severe cases died. The above mentioned results find agreement with those of Cevik that reported, in their study, there are 170 (93.4%) patients had full recovery, six (3.3%) mortalities and six (3.3%) IHMC (five seizure activity and a myocardial ischemia), 30% of severe cases died [8]. For the outcome in acute CO poisoned cases in the present study a special concern was put for studying the mortality and IHMC cases, through the emerged results, the indications of poor prognosis were described as higher mean of ages, higher mean delay time, tachycardia, bradypnea, cyanosis, respiratory distress, pulmonary edema, lowest mean GCS, musculoskeletal edema, tenderness, hypernatremia, leukocytosis, acidosis, hyperglycemia, high levels of CPK- MB, AST and cTnI, ECG changes, abnormal CT (infarction and brain edema) at presentation and the need of mechanical ventilation with hyperbaric oxygen. The results of regression analysis in the present study showed that age, delay time, PSS grade, glucose, AST, CPK- MB and cTnI are significant predictors of outcome. These results indicate that those variables are the most important factors for determining the outcome within the limitations of our study. Piantadosi, reported that indications of poor prognosis were described as altered consciousness at presentation, advanced age, patients with underlying cardiovascular disease and metabolic acidosis [34]. While Cevik, in their study found that, coma was found to have significant correlation with bad outcome, and mortal cases had significantly higher mean age and lower pH levels. They found also that acidosis in presentation has higher mortality and IHMC rate, and its correlation with PSS, COHb level and outcome was moderate [8]. Turner mentioned the importance of initial acidosis as a good predictor of severity of CO poisoning [35]. Pach, reported that PSS generally gives a good grade to poisonings. They concluded that the reason of less satisfying concordance for CO poisoning was mainly caused by discrepancies in the evaluation of neurological symptoms. Modification and additional criteria may be justified to increase reliability of the PSS [6]. Cevik found that, high COHb, leucocyte, glucose and low pH levels with higher PSS grades. In addition, tachycardia, high leucocyte and glucose and low pH levels showed significant correlation with bad outcome. Because initial PSS showed moderate correlation with outcome, there is a question to be solved that accompanying the PSS with initial results of some laboratory tests (leucocyte, glucose, etc.) and/or underlying diseases and/or age may improve the role of PSS in prediction of severity of clinic situation and outcome. They concluded that, as an unreliable laboratory result, COHb levels may not be correlated with clinical severity and outcome. Decreased level of consciousness, acidosis, tachycardia, high glucose and leucocyte levels showed significant relation with higher PSS, COHb level and adverse outcome. For evaluation of the application of a PSS, there should be an adequate sample size and accurate and precise categorization of the grades with specified ranges of the measurements of physiological variables for each category. The results of their study showed that PSS is reliable severity score for CO poisoning cases [8]. However, they agreed with the modification by Pach and additional criteria may be justified to increase reliability of the current PSS. Use of PSS to predict severity, management and outcome needs further prospective studies [6].

5. CONCLUSION

From the results of outcome and study of mortality and IHMC cases, the indications of poor prognosis in the present study were described as higher mean of ages, higher mean delay time,

tachycardia, bradypnea, cyanosis, respiratory distress, pulmonary edema, lowest mean GCS, musculoskeletal edema, tenderness, hypernatremia, leukocytosis, acidosis, hyperglycemia, high levels of CPK- MB, AST and cTnI, ECG changes, abnormal brain CT (infarction and brain edema) at presentation and the need of mechanical ventilation with hyperbaric oxygen. The results of regression analysis showed that age, delay time, PSS grade, Glucose, AST, CPK-MB and Cardiac Troponin I are significant predictors of outcome. These results indicate that those variables are the most important factors for determining the outcome within the limitations of our study. So the results of the present study revealed that PSS is reliable severity score for diagnosis, management and prognosis of COP cases especially with the added modifications in the form of laboratory investigations (ABG, COHb, blood glucose, Na, K, ALT, CPK, urea, creatinine, RBCs, WBCs, HB, cardiac markers (AST, CPK-MB, LDH and quantitative serum cardiac troponin I), ECG and brain CT scan. Limitation of study was small number of patients so similar studies are recommended on large number of patients.

ETHICAL APPROVAL

It is not applicable.

COMPETING INTERESTS

Authors have declared that no competing interests exist.

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