American Journal of Emergency Medicine 37 (2019) 1254–1259

Contents lists available at ScienceDirect

American Journal of Emergency Medicine

journal homepage: www.elsevier.com/locate/ajem

Original Contribution

Predicting of neuropsychosis in carbon monoxide poisoning according to the plasma troponin, COHb, RDW and MPV levels☆ Neuropsychoses in carbon monoxide poisoning

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ARTICLE INFO

Article history:
Received 5 June 2018
Received in revised form 5 September 2018
Accepted 14 September 2018

Keywords:
Carbon monoxide poisoning
Emergency department
Red blood cell distribution width
Mean platelet volume
Neuropsychosis

ABSTRACT

Objective: Carbon monoxide (CO) poisoning is very common worldwide. In this study, we aimed to evaluate the predictivity of neuro psychosis in carbon monoxide poisoning by the admission levels of red cell distribution (RDW), mean platelet volume (MPV) and troponin I levels which can be measured quickly and easily in the emergency department (ED).
Patients and methods: This single center observational study included a total of 216 consecutive patients who presented to the ED due to CO poisoning between January 2009 and December 2013. The diagnosis of CO poisoning was made according to the medical history and carboxyhemoglobin (COHb) level of >5%. According to the carboxyhemoglobin levels, the patients were classified as mildly (COHb < 20%) and severely poisoned (COHb > 20%). In addition, patients were divided into 2 groups, i.e., those with positive (>0.05 ng/mL for our laboratory) and negative (<0.05 ng/mL for our laboratory) troponin levels.
Results: Patients mean age was 52.58 ± 10.58. 57.9% of the patients had high troponin levels and 51.9% were poisoned severely according to COHb levels. Patients with positive troponin and COHb had longer CO exposure time and higher neutrophil, lymphocyte, mean platelet volume (MPV), COHb and red cell distribution width (RDW) levels at the index admission following CO poisoning than patients with negative troponin ($p < 0.05$). Age, COHb level, CO exposure time, MPV and RDW ($p = 0.001$, $p < 0.05$) remained associated with an increased risk of troponin positivity following adjustment for the variables that were statistically significant.
Conclusions: In patients presenting to the ED with CO poisoning, RDW and MPV can be helpful for risk stratification of neuropsychosis.

1. Introduction

Carbon monoxide is a non-irritant odorless, tasteless and colorless gas with hydrocarbon composition. CO binds to hemoglobin with an affinity 240 times greater than that of oxygen and consequently formed carboxyhemoglobin impairs the oxygen release to tissues and oxygen transport. When one CO binds to the heme site of hemoglobin, allosteric change takes place. Binding of the remaining three oxygens to heme increases and oxygen release to tissues is reduced [1].

CO intoxication, which causes a major part of emergency department admissions, is the leading cause of deaths of toxic origin [2,3]. CO binds to hemoglobin with a higher affinity than that of oxygen, transforms into COHb and impairs oxygen transport and utilization. CO also induces lipid peroxidation in the central nervous system (CNS) and inflammatory cascade that results in delayed neurological sequelae [4,5]. Morbidity due to CO intoxication is essentially associated with delayed neurocognitive impairment. After the first stabilization, it might be seen in high rates, up to 40% of the severely affected individuals. Severe delayed encephalopathy and cognitive sequela are rarer and have been reported at a rate of 0.06–11% in various series [6-9]. The clinical picture consists of the signs and symptoms of CNS disorder a few weeks after the recovery period following acute intoxication. Although the pathological mechanism cannot be explained exactly, the underlying pathological lesion is thought to be diffuse demyelination of the cerebral white matter [9]. CO poisoning causes neuropathological changes, along with other biochemical mechanisms apart from anoxia. The suggested mechanisms include cellular hypoxia due to various CO binding intracellular proteins, neurotoxicity due to excessive release of

☆ No support in the form of equipment, drugs, or grants.
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https://doi.org/10.1016/j.ajem.2018.09.017
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excitatory amino acids such as glutamate, development of neutrophil activation due to lipid peroxidation, accumulation of peroxynitrates that cause damage in blood vessel endothelium and apoptosis or programmed cell death [10-15].

RDW can be used in the differential diagnosis of some hematologic diseases including anemia by measuring the variation of erythrocytes in circulation [16].

Neutrophil and lymphocyte (NL) is a simple commonly used laboratory determinant of systemic inflammation [17,18]. NL is a combination of 2 independent inflammation markers. Neutrophils indicate a continuing non-specific inflammation. Lymphocytes are a combination of these 2 markers as an indicator of the regulatory pathway. NL has been proven to be a strong marker of inflammation [19,20].

MPV is a marker of platelet activation. Recent reports suggest that there is a correlation between increased MPV and inflammation as well as the risk of arterial and venous thrombosis [21,22].

According to the studies in the literature, in patients that presented to the ED due to CO poisoning, the relationship between RDW, MPV, neutrophil, lymphocyte, troponin, COHb levels at the time of admission and neuropsychosis has not been evaluated until today. The aim of this study was to evaluate the effects of elevated troponin levels as well as RDW and MPV levels, routinely measured in each patient that present to the ED with CO poisoning, on the brain.

2. Patients and methods

Data about 216 patients (86 males, 130 females; mean age 52.58 ± 10.58 years; distribution 31–75 years, 60.2% females) older than 18 years, who presented to the ED with CO poisoning between January 2009 and December 2013 were analyzed from the charts retrospectively. The clinical classification for carbon monoxide poisoning patients was made as mild-moderate or severe poisoned. While the patients with a carboxyhaemoglobin level between 10%–20% without any symptoms or with headache, lethargy or fatigue created the mild-moderate group, patients with a higher carboxyhaemoglobin level than 20% with loss of consciousness, confusion or cardiac ischaemia were accepted as the severely poisoned group. In addition, patients were divided into 2 groups, i.e., those with positive and negative troponin levels. Neurology and psychiatry admissions were followed up for 5 years by using the hospital's automation system after the patients were discharged from the clinics they had been treated.

At the time of admission, outpatients, patients with cerebrovascular disease, psychiatric diagnosis, chronic liver diseases, patients on dialysis for chronic kidney failure, patients diagnosed previously with infectious, inflammatory disease or malignancy, patients that previously had severe anemia or other hematologic diseases or received anemia treatment and patients who were administered erythrocyte transfusion within the last six months were excluded from the study.

The study was made in accordance with the Declaration of Helsinki for Human Research and was approved by the institutional review board. Demographic, clinical, and laboratory data from the date of presenting to the ED due to CO poisoning, including the COHb, RDW, MPV, neutrophil, lymphocyte and troponin levels, were assessed using review of the hospital's medical records.

Blood was collected from patients at the admission time to the emergency department for measuring COHb, troponin, biochemical levels and CBC. The patients' COHb levels were obtained from arterial blood gas analyses using the Acobas® b221 Blood Gas system (Roche, Basel, Switzerland). RDW, MPV, neutrophil and lymphocyte were measured using a Beckman Coulter Automated CBC Analyzer (Beckman Coulter, Inc., Fullerton, CA, USA). The normal reference range for RDW in our laboratory is 11.5–14.5. Neutrophil, lymphocyte and MPV normal reference intervals are 41–73%, 19–45%, 6,85–11 fL, respectively.

Troponin I levels, which were evaluated within fifteen minutes after the patients were admitted to ED, were measured with a one-step immunofluorometric assay sandwich method using three monoclonal antibodies (AQ90 Flex, Radiometer Medical ApS, Brønshøj, Denmark). The conventional definition of elevated troponin level is when this value exceeds the 99th percentile value of a healthy reference population and elevated test level, which is >0.05 ng/mL, for our laboratory, was accepted as positive. Additionally, non-elevated test level, which is ≤0.05 ng/mL, was accepted as negative.

A diagnosis of CO poisoning was made according to the medical history and a COHb level >5%. CO exposure time was defined as the approximate duration of CO inhalation.

2.1. Statistical analysis

The Kolmogorov-Smirnov test was used to verify the normality of the distribution of continuous variables. Continuous variables were expressed as mean ± SD or median (min-max) in the presence of abnormal distribution, and categorical variables as percentages. Receiver operator characteristic (ROC) curve analysis was performed to identify the optimal cut-off point of RDW and MPV (at which sensitivity and specificity would be maximal) for the prediction of troponin positivity. Comparisons between the groups of patients were made by the χ^2-test for categorical variables, the independent samples *t*-test for normally distributed continuous variables, and the Mann-Whitney *U* test when the distribution was skewed. The correlation was evaluated by Spearman correlation test. All statistical procedures were performed using SPSS software version15.0 (SPSS Inc., Chicago, IL, USA). A p-value of 0.05 was considered statistically significant.

3. Results

51.9% of the patients were severely poisoned according to COHb, and 59.7% of the patients had high troponin levels. RDW, MPV, neutrophil and lymphocyte levels were higher in patients with high troponin and COHb levels, as compared to those with low levels thereof. In addition, mean age, CO exposure time, COHb, AST, ALT and ALP were higher in the group with high troponin and COHb levels as compared to the group with low levels thereof ($p < 0.05$) (Tables 1A, B).

Moreover, RDW and MPV levels, troponin, COHb, glucose, amylase, CRP, WBC, MCHC (mean corpuscular hemoglobin concentration) and CO exposure time levels exhibited Spearman correlation (Table 2).

COHb and troponin positive and negative groups were compared in terms of gender, late neuropsychoses and mortality. χ^2 test was used for the comparison of two numerical values and the results were statistically significant ($p = 0.001$, $p < 0.05$) (Table 3A, B). While the mean troponin value among the 19 mortal patients was 2.58 ± 2.12 ng/dL, the mean value for the discharged patients was 0.67 ± 1.03 ng/dL ($p < 0.001$).

According to ROC curve analysis, to determine troponin positivity, RDW and MPV optimal cutoff values were as follows; RDW: >12.65 sensitivity 82% and specificity 53%; MPV: >9.95 fL sensitivity 87% and specificity 51% ($p = 0.001$, $p < 0.05$) (Fig. 1).

According to ROC curve analysis, to determine COHb positivity RDW and MPV optimal cutoff values were as follows, RDW: >13.14 sensitivity 84% and specificity 52%; MPV: >12.02 fL sensitivity 86% and specificity 54% ($p = 0.001$, $p < 0.05$) (Fig. 2).

4. Discussion

We demonstrated that high RDW, MPV, neutrophil, lymphocyte and COHb levels on admission, long CO exposure time, and advanced age could independently predict the neuropsychosis in patients who admitted to the ED with CO poisoning.

Changes due to acute CO poisoning are primarily manifested in organs such as the brain and heart, which have a high need for oxygen [23]. CO is also known to bind to heme proteins such as neuroglobin, cytochrome oxidase, cytochrome P-450, dopamine beta hydroxylase, tryptophan oxidase in brain structures such as the globus pallidus and

Table IA
Analyzing of the baseline characteristics and laboratory results according the troponin and severity classification of the poisoning.

	All patients n:216	Patients with (+) Troponin n:129	Patients with (-)Troponin n:87	p-value
Mean age(y)	52,58 ± 10,58	55,23 ± 10,80	48,48 ± 8,67	**0,001**
Famale	130(60,2%)	92(69,2%)	41(30,8%)	**0,001**
Male	86(39,8%)	37(44,6%)	46(55,4%)	
CRP	3,84 ± 4,42	6,01 ± 4,56	0,63 ± 0,67	**0,001**
CO ET (h)	3,69 ± 2,61	4,29 ± 2,36	3,28 ± 2,86	**0,005**
WBC(10^3/uL)	9,61 ± 3,22	10,87 ± 3,39	7,73 ± 3,39	**0,001**
RDW (%)	14,93 ± 1,68	15,66 ± 1,55	14,44 ± 1,59	**0,001**
MPV fL	8,52 ± 1,02	8,93 ± 0,86	8,24 ± 1,03	**0,001**
MCHC g/dL	32,57 ± 1,58	32,84 ± 1,54	32,17 ± 1,54	**0,002**
MCV	88,14 ± 5,46	87,83 ± 5,91	88,61 ± 4,71	0,304
MCH	29,53 ± 2,52	29,52 ± 2,55	29,54 ± 2,50	0,944
Neu %	4,84 ± 3,28	5,74 ± 3,86	3,50 ± 1,32	**0,001**
Lymph %	1,67 ± 1,33	2,01 ± 1,52	1,18 ± 0,77	**0,001**
Glu (mg/dl)	127,25 ± 43,25	134,37 ± 50,59	118,12 ± 31,67	**0,008**
AST (U/L)	34,36 ± 25,40	38,97 ± 28,31	27,29 ± 18,38	**0,001**
ALT (U/L)	33,02 ± 27,65	38,74 ± 30,66	24,54 ± 19,75	**0,001**
ALP (U/L)	117,30 ± 63,43	129,30 ± 62,64	99,50 ± 60,68	**0,001**
CK (U/L)	142 ± 93,49	143,28 ± 98,80	140,09 ± 85,53	0,806
CK-M(U/L)	30,93 ± 21,43	30,93 ± 23,66	30,92 ± 17,74	0,995
Amylase U/L	125,08 ± 69,68	164,75 ± 62,57	66,26 ± 21,21	**0,001**
COHb (%)	26,10 ± 14,01	34,08 ± 13,02	14,41 ± 2,72	**0,001**
Tn(ng/dL)	0,84 ± 1,28	1,40 ± 1,40	0,16 ± 0,01	**0,001**

(+): positive; (-): negative; CRP: C reactive protein; CO ET: carbon monoxide exposure time; WBC: white blood cell; RDW: red cell distrubition width; MPV: mean platelate volume; MCHC: mean corpuscular hemoglobin concentration; MCV; mean corpuscular volume; MCH: mean corpuscular hemoglobin; Neu: neutrophil; Lymph: lymphocyte; Glu: glucose; AST: aspartate aminotransferase; ALT: alanine aminotransferase; ALP: alkaline phosphatase; CK: creatine kinase; CK-MB: creatine kinase-muscle brain; COHb: carboxyhemoglobin; Tn: troponin.

Table 2
Spearman correlation coefficients for RDW and MPV.

	RDW rp-value		MPV rp-value	
Troponin	**−0,394**	**<0,001**	**−0,358**	**<0,001**
COHb	**−0,425**	**<0,001**	**−0,395**	**<0,001**
Amylase	**−0,384**	**<0,001**	**−0,394**	**<0,001**
CRP	**−0,411**	**<0,001**	**−0,401**	**<0,001**
WBC	**−0,179**	**<0,008**	**−0,270**	**<0,001**
MCHC	**−0,294**	**<0,001**	**−0,280**	>0,687
Blood sugar	**0,180**	>0,797	**0,003**	>0,965
CO exposure time	**0,116**	>0,088	**0,175**	**<0,010**

Bold values indicates significance at p < 0.05.

substantia nigra that are rich in iron and cause direct toxic effect [24-28]. In some cases, hippocampus and thalamus damage, cortical atrophy, loss of cerebellum Purkinje and internal granular layer cells may be observed. CO poisoning causes demyelination and degradation of the cerebral white matter in addition to gray matter lesions [24-30]. Although white matter lesions are seen in regions such as the frontal and parietal cortex, centrum semiovale and brain stem, the most frequently affected region is reported to be the parieto-occipital region [28]. 10–30% of the patients after CO poisoning were reported to develop delayed neuropsychiatric syndrome primarily characterized by cognitive changes, personality changes, parkinsonism, incontinence, dementia and psychosis [26,29], whereas young adults also had peripheral neuropathy in addition to these signs [29]. In our study, only 1% of the mild-moderately poisoned patients exhibited the neuropsychiatric syndromes mentioned in the literature, whereas 32.7% of the severely poisoned patients showed the aforementioned syndromes. In groups with high levels of RDW, MPV and troponin, a negative correlation was found using the Spearman analysis.

Our study included 130 (60.2%) females and the mean age was 52.58. In literature, there are gender and age differences in many studies. The percentage of males and the mean age were as follows, respectively; 61.62% and 37.73 in a study by Türkmen et al. [31], 71.83% and 33.39 in a study by Durak [32], 76.60% and 32.60 in a study by Azmak et al. [33]. On the other hand, the meta analysis study by Hosseininejad et al. [34] included 4620 (40.12%) males and 3057 (59.88%) females. The mortality rate was 9.50% (485) including 259 (5.07%) males and 226 (4.43%) females. Data from all CO poisoning cases in four studies were used in order to determine the mean age of all CO poisoning patients and the mean age was found to be 31.68. In the other six studies that only included fatal CO poisoning cases, the mean age of victims was 30.29. Considering all the studies, the mean age of fatal CO poisoning cases was found to be 31.24. The majority of patients were between the ages 15–44 in all studies. The mean age in our study was higher than the mean age indicated in literature. This may have stemmed

Table IB
Baseline characteristics of study patients.

	Severely Carbonmonoxide Poisoning patients COHb (>20%) n:106	Mild-moderate Carbonmonoxide poisoning Patients COHb (10%-20%) n:110	p-value
Meanage(y)	55,64 ± 11,18	49,14 ± 8,60	**0,001**
Famale	81(60,9%)	52(39,1%)	**0,001**
Male	25(30,1%)	58(69,9%)	
CRP	4,40 ± 0,41	0,68 ± 0,71	**0,001**
CO ET (h)	4,26 ± 2,72	3,10 ± 2,36	**0,001**
WBC(10^3/uL)	11,29 ± 3,36	7,79 ± 1,74	**0,001**
RDW (%)	15,69 ± 1,66	14,22 ± 1,37	**0,001**
MPV fL	8,95 ± 0,96	8,11 ± 0,90	**0,001**
MCHC g/dL	32,94 ± 1,48	32,17 ± 1,59	**0,001**
MCV	87,84 ± 5,98	88,46 ± 4,85	0,406
MCH	29,41 ± 2,32	29,65 ± 2,73	0,503
Neu %	6,09 ± 4,01	3,49 ± 1,27	**0,001**
Lymph %	2,17 ± 1,56	1,14 ± 0,74	**0,001**
Glu (mg/dl)	136,99 ± 53,01	117,97 ± 30,54	**0,002**
AST (U/L)	41,38 ± 29,45	26,60 ± 17,25	**0,001**
ALT (U/L)	41,36 ± 31,72	24,04 ± 18,82	**0,001**
ALP (U/L)	134,62 ± 63,04	98,64 ± 58,65	**0,001**
CK (U/L)	143,38 ± 90,18	140,50 ± 97,34	0,822
CK-M(U/L)	31,42 ± 24,46	30,40 ± 17,69	0,729
Amylase U/L	177,91 ± 55,86	68,18 ± 21,62	**0,001**
COHb (%)	36,88 ± 11,61	14,61 ± 2,71	**0,001**
Tn(ng/dL)	1,59 ± 1,41	0,03 ± 0,05	**0,001**

Table 3
COHb and troponin positive and negative groups were associated with gender, neuropsychosis and mortality.

A

		COHb		χ^2	p-Value
		>20%	10%–20%		
Gender	Male	25 (30,1%)	58 (69,9%)	**19,37**	**<0,001**
	Female	81 (60,9)	52 (39,1%)		
Neuropsychosis	No	72 (40,4%)	106 (59,6%)	**30,11**	**<0,001**
	Yes	34 (89,5%)	4 (10,5%)		
Mortality	No	89 (45,2%)	108 (54,8%)	**13,60**	**<0,001**
	Yes	17 (89,5%)	2 (10,5%)		

B

		Troponin		χ^2	p-Value
		Positive	Negative		
Gender	Male	37 (44,6%)	46 (55,4%)	**12,85**	**<0,001**
	Female	92 (69,2%)	41 (30,8%)		
Neuropsychosis	No	91 (51,1%)	87 (48,9%)	**30,09**	**<0,001**
	Yes	38 (100%)	0		
Mortality	No	110 (55,8%)	87 (44,2%)	**14.50**	**<0,001**
	Yes	19 (100%)	0		

Bold values indicates significance at p < 0.05.

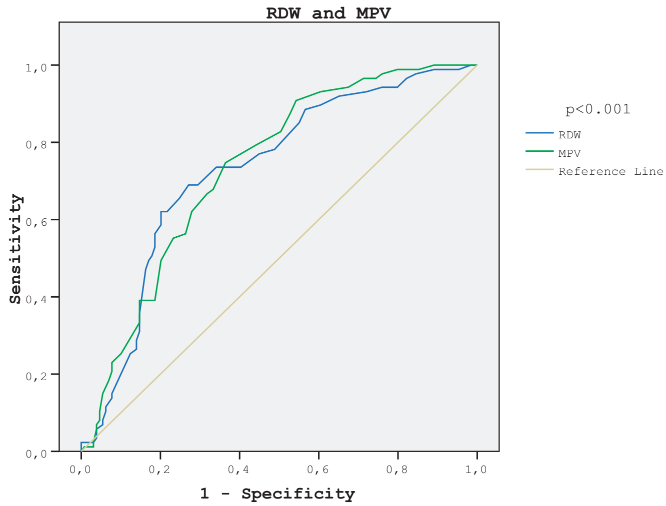

Fig. 1. ROC curve for troponin positivity.

from the family and community structure as well as the general increase in the mean age of the community.

According to literature reviews there are only several case presentations that directly investigates the inflammation marker in late complications due to CO poisoning. The pathophysiological mechanisms underlying these complications have still not been clearly understood. One of these mechanisms involves the activation of nitric oxide and other oxygen-free radicals by CO poisoning [35]. Oxygen-free radicals affect blood flow, thereby contributing to endothelium damage [36]. It is asserted that this oxidative damage substantially mediates leukocytes. In addition, when rats are rendered leukopenic, lipid peroxidation is inhibited after CO poisoning. Leukocyte sequestration increases significantly in brain microvasculature after exposure to CO [37]. Formation of oxygen radicals during reperfusion was found to be the main component of ischemic brain damage [38]. Previous studies also showed that neutrophils play a role in CO-mediated brain injuries in CO poisoning [37,39]. In this study, white blood cell count, as well as neutrophil and lymphocyte count in circulation, was significantly higher in the severely poisoned groups according to the COHb and troponin levels. These high levels in the acute stage were found to have positive correlation with increased mortality in the short-term, and neuropsychoses in the long-term.

It was reported that the second mechanism, i.e. thrombotic tendency, increased in patients with CO poisoning [40,41]. Thom et al. showed that acute CO poisoning caused intravascular neutrophil activation due to the interaction with platelets [42]. Similarly in our study, we found that MPV levels were significantly higher in patients with CO poisoning. We believe that the results of our study will implicitly support this study. Oxygen-free radicals can affect blood flow and platelet aggregation that contributes to endothelium damage [43]. In addition, free radicals can increase platelet adhesion and lead to changes in the fibrinolytic pathway [44]. Therefore, apart from oxidative stress, hypoxia may cause a direct increase in MPV in patients with CO poisoning [45,46]. In our study, MPV was significantly higher in the group with positive cardiac enzymes. According to these results, we can say that CO poisoning causes an increase in MPV, which is an indicator of platelet activation. In patients with CO poisoning, complications might be due to increased platelet activation in addition to hypoxia. Hence, antiplatelet drug use may be plausible in acute CO poisoning.

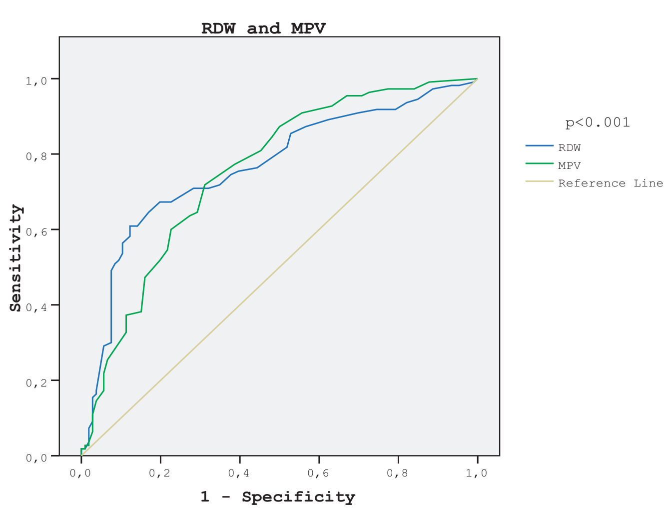

Fig. 2. ROC curve for COHb positivity.

In this study, laboratory tests revealed high positive values of COHb and troponin in 51.9% and 59.7% of the patients that presented to the ED with CO poisoning, respectively. COHb, CO exposure time and age were independent determinants of positive troponin. In a study by Satran et al. [47], the rate of cardiac biomarker positivity, which is defined as elevated CK-MB and/or troponin, was 44%. Kalay et al. [48] found the troponin positivity rate to be 30% in a study consisting of 20 patients. They observed that COHb levels and CO exposure time in troponin-positive patients were significantly higher as compared to troponin-negative patients, which is consistent with our study. While early mortality rate was 14.7% in the troponin-positive group, there were no mortalities in the troponin-negative group. In addition, late neuropsychosis was seen in 29.5% of the troponin-positive group and not seen in the troponin-negative group. Our study was similar to the studies of Satran [47] and Kalay [48].

Until recently, studies have shown that RDW is elevated in various pathologies, and is even directly related to mortality. Since it is associated with so many pathologies, RDW has been studied as a marker of inflammation, and Lippi et al. [49] reported a positive correlation with C-reactive protein (CRP) levels. Marinkovic et al. [50] reported that red blood cell life was shorter in conditions of oxidative stress, which resulted in increased hemolysis and subsequently elevated RDW. Our study also showed that CRP, RDW and MPV were significantly higher in the COHb and troponin-positive group as compared to the negative group and there was a negative Spearman correlation as seen in the literature.

For patients with COHb levels between 10% and 20% in the ED, the most common symptom was headache, which usually develops when COHb levels are around 25%. This suggests that these patients may have had initial COHb levels of between 20% and 30% [51]. Our study is consistent with the literature. In the group with COHb values lower than 20%, there were a few neuropsychiatric symptoms and no mortality, whereas the group with COHb higher than 20% had significantly high rates of neuropsychosis and mortality. Moreover, in our study, elevated troponin levels were observed in all patients in the group that had ≥40% COHb values.

Our research had one of the highest numbers of patients, including one in all studies on CO poisoning, RDW and MPV, were also found as an independent predictor of troponin positivity. RDW can be used in the differential diagnosis and prognosis follow-up of hematological diseases. However, recently there are several studies about its relation to cardiovascular diseases. It was shown that RDW is related to morbidity and mortality in acute coronary syndrome, coroner artery disease, acute pulmonary embolism, and heart failure [52-55].

In our report, RDW elevation in troponin-positive patients can be related to two pathological mechanisms. First, although patients with a known coronary artery disease were excluded, troponin-positive patients could have undiagnosed coronary artery disease, which could make the myocardium more sensitive to hypoxia. Therefore, in patients with asymptomatic atherosclerosis, and consequently high levels of RDW before CO poisoning, faster myocardial damage and a troponin increase can be observed when exposed to CO [56-58]. The second mechanism that can explain the higher levels of RDW in patients who have CO poisoning with troponin elevation can be related to the acute effect of COHb molecules on erythrocytes. COHb is generated by binding CO to the heme molecule in erythrocytes instead of oxygen, which may cause anisocytosis and RDW elevation by making structural changes in erythrocytes. RDW elevation in smokers compared to non-smokers was shown in a small case-control trial. In a study by Skjel-bakken et al. [59], a more powerful relationship between RDW and MI was noted in smokers when compared with non-smokers. In these studies, although the relation between smoking and RDW was connected to oxidative stress and increased inflammation, it should be kept in mind that smokers also have CO exposure and their COHb levels are higher than non-smokers [60].

There are some limitations in this study. This is a single center study and an observational evaluation was made, so there were follow-up data for the patients. Considering that the patients' basal RDW values before poisoning were not known, it cannot be estimated whether RDW values were elevated before poisoning or were elevated due to the acute effect of COHb on erythrocytes.

5. Conclusions

In CO poisoning cases, neuropsychological conditions that may develop in the future can easily go unnoticed, especially if the patient is asymptomatic. Physicians taking into account especially the COHb, Troponin, MPV and RDW levels evaluated in our study might be a guide in preventing neuropsychosis that can possibly develop in the future and in early diagnosis.

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