MEDICAL RECORDS-International Medical Journal

Research Article

Systemic Inflammatory Markers (NLR/PLR) and Climatic Factors as Predictors of Severity in Pediatric Carbon Monoxide Poisoning

Sebahattin Memis[1], Mehmet Semih Demirtas[2], Huseyin Erdal[3], Gizem Sorkulu Memis[1], Mustafa Tusat[4], Emine Ozdemir Kacer[2]

[1]Aksaray University, Training and Research Hospital, Department of Pediatrics, Aksaray, Türkiye
[2]Aksaray University, Faculty of Medicine, Department of Pediatrics, Aksaray, Türkiye
[3]Aksaray University, Faculty of Medicine, Department of Medical Genetics, Aksaray, Türkiye
[4]Aksaray University, Faculty of Medicine, Department of Pediatric Surgery, Aksaray, Türkiye

Abstract

Aim: Carbon monoxide (CO) poisoning remains a global health challenge, particularly in developing nations. This study evaluates the prognostic role of neutrophil-to-lymphocyte ratio (NLR), platelet-to-lymphocyte ratio (PLR), and climatic parameters in pediatric CO poisoning severity.

Material and Method: In this retrospective cohort study, 132 pediatric patients admitted to a tertiary emergency department were stratified by carboxyhemoglobin (COHb) levels (mild: <5%; moderate: 5–25%; severe: >25%). Associations between NLR/PLR, meteorological data (temperature, wind speed, air pressure), and clinical outcomes were analyzed.

Results: Severe cases exhibited significantly elevated NLR (2.09 [IQR 1.24-3.54]; p=0.019) and PLR (106.2 [80.6-164.4]; p=0.032) compared to others, alongside higher COHb (31.45 [27.25-34.2]; p<0.001) and lactate (2.7 [1.8-3.45] mmol/L; p=0.07). Central nervous system symptoms (CNS) predominated in severe poisoning (28.6%, p=0.014). Maximum temperature was inversely correlated with severity (p=0.032).

Conclusion: NLR and PLR serve as early indicators of CO poisoning severity, while higher air temperatures reduce risk. These findings support using routine hemogram indices for risk stratification and underscore the need for climate-aware public health interventions.

Keywords: Carbon monoxide poisoning, air temperature, children, systemic immune/inflammatory index markers, temperature

INTRODUCTION

Carbon monoxide (CO), a colorless, odorless, and tasteless gas, is produced by the incomplete combustion of carbon-containing fuels. It is a significant air pollutant emitted from sources such as engine exhausts and fires. In many regions, especially in developing countries, CO poisoning often results from the inadequate burning of wood or coal used for heating during cold weather or for activities like cooking and baking (1). Once inhaled, CO binds to hemoglobin with an affinity approximately 250 times greater than oxygen, forming carboxyhemoglobin (COHb). This process reduces hemoglobin's oxygen-carrying capacity, leading to tissue hypoxia (2). Clinically, CO poisoning can present with nonspecific symptoms such as headache, dizziness, and nausea, or progress to severe neurological manifestations, including loss of consciousness and death (3). Notably, symptom severity does not always correlate with COHb levels, making early diagnosis and intervention critical (4). Children are particularly vulnerable due to their higher metabolic rates, increased respiratory rates relative to body surface area, and limited ability to articulate symptoms (5). Delayed recognition of CO poisoning can result in long-term neurological sequelae, underscoring the need for effective diagnostic and prognostic tools.

The neutrophil-to-lymphocyte ratio (NLR) and platelet-to-lymphocyte ratio (PLR), derived from routine complete blood counts, are emerging as systemic immune/

CITATION
Memis S, Demirtas MS, Erdal H, et al. Systemic Inflammatory Markers (NLR/PLR) and Climatic Factors as Predictors of Severity in Pediatric Carbon Monoxide Poisoning. Med Records. 2025;7(3):756-62. DOI:1037990/medr.1726333

Received: 24.06.2025 **Accepted:** 20.08.2025 **Published:** 09.09.2025
Corresponding Author: Sebahattin Memis, Aksaray University, Training and Research Hospital, Department of Pediatrics, Aksaray, Türkiye
E-mail: dr.sebahattinmemis@gmail.com

inflammatory markers. Initially studied in malignancies, these indices are increasingly used to assess inflammation and predict outcomes in pediatric diseases (6). Their accessibility and low cost make them promising candidates for evaluating CO poisoning severity (7).

CO poisoning exhibits seasonal patterns, with higher incidence during winter months when low temperatures, reduced wind speeds, and increased indoor heating contribute to gas accumulation in enclosed spaces (8). Despite global awareness, CO poisoning remains a persistent public health challenge, particularly in developing nations (9). Early prediction of poisoning severity is essential to mitigate adverse outcomes.

This study investigates the relationship between climate parameters (e.g., temperature, humidity, wind speed, air pressure), systemic inflammatory markers (NLR, PLR), and CO poisoning severity in pediatric patients. By analyzing these factors, we aim to identify accessible prognostic indicators and environmental risk factors to improve clinical management and prevention strategies.

MATERIAL AND METHOD

Study Design

This study is a retrospective cohort study conducted in the pediatric emergency department of Aksaray Training and Resarch Hospital, a tertiary level university hospital.

Study Population

The study included 235 pediatric patients aged 1 month to 18 years who were admitted to the Aksaray Training and Resarch Hospital Pediatric Emergency Department with CO poisoning between 2017 and 2024. A total of 103 patients whose climate parameters were not accessible before 2018 and after August 2023 were excluded from the study. A total of 132 patients were included in the study.

Data Collection

The data in the study were obtained retrospectively from the hospital data system. Demographic characteristics of the patients such as age, gender, and nationality, admission date, day, time, time zone, patient's arrival symptoms, COHb level at the time of admission date, hemoblobin (g/L), neutrophil (mm^3), lymphocyte (mm^3), platelet (mm^3), blood gas parameters, albumin (g/L), C-reactive protein (CRP) (mg/L), troponin (pg/mL), follow-up period, and control laboratory parameters were obtained retrospectively from the hospital information system and recorded. Climate parameters were obtained from the Meteorology Department of the Ministry of Environment and Urbanization of the Republic of Türkiye. Air temperature (°C), humidity (g/m^3), wind speed (Knot), wind direction, and air pressure (mb) on the days when the patients applied were recorded.

In our study, patients were stratified by age (<5 years and ≥5 years) and categorized into three exposure subgroups: stove-related, natural gas-related, and other sources (e.g., fires, vehicle exhaust, hookah). Clinical presentations were classified by system involvement: respiratory system (shortness of breath, cough, tachypnea), central nervous system (CNS) (seizures, confusion, dizziness, headache), or gastrointestinal system (GIS) (vomiting, nausea, abdominal pain).

Disease severity was defined by admission COHb levels: mild (COHb<5%), moderate (COHb: 5–25%), or severe (COHb>25%). This stratification enabled systematic analysis of demographic, clinical, and environmental correlations with poisoning severity (7).

Ethics

Aksaray University Health Sciences Scientific Research Ethics Committee approved the decision dated 05.12.2024 and numbered SAGETIK134.

Statistical Analysis

The normality of variable distributions was assessed through multiple approaches, including visual inspection of histograms and quantitative measures (kurtosis, skewness), supplemented by the Kolmogorov-Smirnov test. For non-normally distributed variables, we employed non-parametric tests: the Mann-Whitney U test for comparisons between two independent groups and the Kruskal-Wallis test for analyzing CO poisoning severity classifications. These non-parametric results are presented as medians with interquartile ranges (IQR-25-75). Categorical variables were evaluated using the Chi-square test, with post-hoc analyses (adjusted residuals with Bonferroni correction) to identify specific subgroup differences when significant overall effects were detected.

RESULTS

The study cohort comprised 132 pediatric patients, with a near-equal gender distribution (47% female [n=62]; 53% male [n=70]). The majority (92.4%) were Turkish nationals, while 7.6% were refugees. Patients had a mean age of 115.80±65.202 months (approximately 9.7 years), with 28.8% (n=38) aged ≤5 years and 71.2% (n=94) >5 years. When the application times of the patients were examined: 76% of admissions occurred during winter months, with February accounting for 40% of cases. Weekly distribution showed Wednesday as the most common admission day (peak frequency), while diurnal patterns demonstrated that 38.6% of presentations clustered between 08:00-16:00. Patients predominantly presented with respiratory (39.4%, n=52; cough/respiratory distress) or GIS (33.3%, n=44; nausea/vomiting) symptoms. Neurological sysmtomps occurred in 27.3% (n=36), ordered by decreasing frequency: confusion (n=14), headache (n=12), dizziness (n=8), and seizures (n=2), reflecting progressive CNS involvement. The average time between poisoning and hospital admission was 1.6±0.516 hours. Demographic data are summarized in Table 1.

Initial laboratory evaluation revealed the following key findings (presented as mean±SD): Hematologic parameters included elevated white blood cells (WBC 10.45±3.89/mm³), platelets (318.45±77.99/mm³), and neutrophil predominance (5.96±3.32/mm³), with corresponding systemic inflammatory indices (NLR 2.55±2.77; PLR 117.01±71.50). We found that there was a metabolic disorder in biochemical markers: lactic acidosis (lactate 2.36±1.23 mmol/L; pH 7.35±0.58), elevated cardiac enzymes (CK 182.9±441.7 U/L; CK-MB 29.91±16.61 U/L; troponin 28.02±205.04 pg/mL), and mild hypoalbuminemia (44.42±3.84 g/L). Blood gas analysis showed significant hypoxia (PaO_2 36.4±21.14 mmHg) with compensatory respiratory alkalosis (PCO_2 40.85± 7.44 mmHg; HCO_3^- 22.41±2.81 mmol/L). The mean COHb level confirmed substantial exposure (15.84±9.37%), while CRP (2.70±12.31 mg/L) indicated modest systemic inflammation.

Analysis of meteorological conditions during patient admissions revealed the following patterns (mean±SD): maximum wind speed (10.03±5.34 knots), average wind speed (2.42±1.36 knots), barometric air pressure (904.48±6.87 mb), and air temperatures (average: 6.54±7.84°C; maximum: 11.21±8.79°C; minimum: 2.08±6.78°C).

Under 5 years of age, WBC was 11.19 (9.78-13.75), whereas it was 9.01 (7.69-11.04) above 5 years of age (p<0.001). Under 5 years of age, NLR was 1.08 (0.47-1.85), whereas it was 2.07 (1.30-3.55) above 5 years of age (p<0.001). Under 5 years of age, PLR was 76.72 (48.9-99.1), whereas it was 117.25 (83.76-159.46) above 5 years of age (p<0.001). Under 5 years of age, troponin was 9.25 pg/mL (2.8-19.8), whereas it was 2.4 (1.65-3.65) above 5 years of age (p<0.001). CK level in female patients was 100 U/L (79.5-131.5) and 145.5 (103-200) in male patients (p<0.001). Under 2 hour of admission time NLR was 1.51 (0.81-2.52), whereas it was 2.26 (1.28-3.75) above 2 hours (p=0.01) (Table 2).

Patients stratified by COHb levels into mild (MG), moderate (MoG), and severe (SG) groups demonstrated significant differences in key parameters. The SG group exhibited significantly lower admission pH values compared to milder cases (p=0.030) and markedly higher COHb levels (p<0.001). The SG exhibited significantly elevated inflammatory markers compared to MG and MoG, with median NLR (2.09 [IQR 1.24-3.54]; p=0.019) and PLR (106.2 [80.6-164.4]; p=0.032), confirming their association with disease severity. Climatic analysis revealed higher maximum temperatures in MG versus SG (p=0.032) (Table 3).

No significant associations were found between poisoning severity and age (p=0.278), gender (p=0.433), nationality (p=0.432), admission season (p=0.413), time-to-admission (p=0.910), or wind direction (p=0.768). Etiological analysis revealed distinct exposure patterns: stove-related poisonings accounted for only 8.9% of SG cases, compared to 66.7% for natural gas and 60% for other sources (fire/hookah/exhaust; p=0.001). Clinical presentations varied significantly by severity: In the group with CNS symptoms, 28.6% of the cases were in the SG group, more than in the other groups (p=0.014). COHb levels showed no significant correlation with gender, age group, or time-to-hospitalization (Table 4).

Table 1. Sociodemographic characteristics of the patients who participated in the study

Parameters	Subgroups	All patient, % (n) n=132
Age	0-5 year	28.8 (38)
	>5 year	71.2 (94)
Gender	Female	47 (62)
	Male	53 (70)
Nationality	Turkish	92.4 (122)
	Refugee	7.6 (10)
Hospital admission season	Spring	29.5 (39)
	Summer	2.3 (3)
	Fall	10. 6 (14)
	Winter	57.6 (76)
Hospital admission day	Monday	10.6 (14)
	Tuesday	12.9 (17)
	Wednesday	24.2 (32)
	Thursday	12.1 (16)
	Friday	11.4 (15)
	Saturday	12.9 (17)
	Sunday	15.9 (21)
Admission hour	00-08	31.1 (41)
	08-16	38.6 (51)
	16-24	30.3 (40)
Location	Village	28.8 (38)
	District	25 (33)
	Center/city	46.2 (61)
Source	Stove	89.4 (118)
	Natural gas	2.3 (3)
	Others*	8.3 (11)
Incident admission time	0-2 hours	65.2 (86)
	>2 hours	35.8 (46)
Patient's arrival clinical symptoms	Respiration systemμ	39.4 (52)
	CNS**	27.3 (36)
	GIS£	33.3 (44)

*=Others: exposure to hookah, fire, car exhaust fumes, μ=shortness of breath, cough/choking **=CNS: central nervous system (headache, dizziness, drowsiness, confusion), GIS£: gastrointestinal system (vomiting, nausea, abdominal pain)

DOI: 10.37990/medr.1726333

Table 2. Evaluation of hemogram, biochemistry and climate parameters between age, gender and incident admission time

Parameters	Age*				Gender*				Incident admission time*			
	<5 year	>5 year	Z	p **	Female	Male	Z	p **	0-2 hour	>2 hour	Z	P**
Hemogram and biochemistry parameters												
WBC[1] (x1000 mm³)	11.19 (9.78-13.75)	9.01 (7.69-11.04)	-3.48	**0.001**	9.64 (7.64-11.31)	9.69 (8.22-12.92)	-1.06	0.288	9.66 (7.69-11.70)	9.45 (8.49-11.97)	-0.69	0.488
NLR[2]	1.08 (0.47-1.85)	2.07 (1.30-3.55)	-3.63	**<0.001**	1.79 (1.03-3.42)	1.67 (0.90-2.88)	-0.83	0.404	1.51 (0.81-2.52)	2.26 (1.28- 3.75)	-2.58	**0.01**
PLR[3]	76.72 (48.9-99.1)	117.25 (83.76-159.46)	-3.85	**<0.001**	104.65 (77.65-164.03)	96.76 (64.83-123.82)	-1.59	0.111	97.13 (62.15-138.46)	107.91 (89.55-171.13)	-1.56	0.118
CK (U/L)	113 (87-169)	109 (90-169)	-0.23	0.812	100 (79.5-131.5)	145.5 (103-200)	-3.59	**<0.001**	109 (85-173)	116 (95-165)	-0.64	0.517
CK-MB (U/L)	31 (25.5-40.5)	24.5 (19.25-33.75)	-2.46	**0.014**	27 (22-33.25)	26 (20-41)	-0.365	0.715	27 (22-36)	26.5 (19-34)	-0.45	0.651
pH	7.37 (7.31-7.40)	7.35 (7.31-7.39)	-0.67	0.501	7.37 (7.32-7.40)	7.34 (7.31-7.38)	-1.62	0.104	7.35 (7.31-7.39)	7.36 (7.32-7.38)	-0.03	0.975
COHb	12.65 (6.07-20.75)	16.6 (10.35-23.75)	-2.09	**0.036**	14.1 (7.9-22.2)	15.9 (9.1-22.6)	-0.82	0.409	14.3 (7.6-23.95)	15.6 (10.37-20.82)	-0.48	0.631
Laktat	2.15 (1.6-3.22)	2 (1.60-2.70)	-0.92	0.357	2 (1.67-2.82)	2.05 (1.57-2.70)	-0.02	0.982	2.1 (1.6-2.72)	1.95 (1.50-2.75)	-0.58	0.560
Albumin (g/L)	43.8 (40.75-46.5)	44.8 (41.17- 47.87)	-0.40	0.684	43.45 (41.17-46.55)	44.7 (41.2-48.35)	-0.59	0.550	43.1 (40.95-46.3)	44.65 (42.82-48.37)	-0.87	0.384
CRP[4] (mg/L)	0.32 (0.23-0.64)	0.56 (0.30-1.85)	-2.62	**0.009**	0.52 (0.28-1.38)	0.45 (0.25-1.47)	-0.29	0.767	0.49 (0.26-1.57)	0.44 (0.29-1.37)	-0.17	0.862
Troponin (pg/mL)	9.25 (2.8-19.8)	2.4 (1.65-3.65)	-3.77	**<0.001**	2.40 (1.55-6.12)	2.80 (1.90-5.00)	-0.75	0.454	2.5 (1.72-4.6)	3.2 (1.8-5.9)	-1.16	0.244
Climate parameters												
Max WS[4] (Knot)	10.3 (5.1-13.4)	7.95 (5.10-14.40)	-0.10	0.918	10.8 (6.45-14.4)	7.45 (5.10-13.02)	-1.77	0.076	7.7 (5.1-12.9)	10.3 (5.55-14.4)	-1.49	0.135
Mean WS[4] (Knot)	2.25 (1.37-3.8)	1.75 (1.17-3.72)	-0.73	0.463	2.4 (1.35-3.9)	1.4 (1.2-3.4)	-1.78	0.074	1.6 (1.2-3.7)	2.4 (1.37- 4.05)	-1.46	0.142
Air pressure (mb)	903.45 (898.6-909.85)	904.8 (898.6-909.8)	-2.61	0.794	902.8 (898.6-907.8)	905.6 (901.05-910.10)	-2.14	**0.032**	905 (898.7-909.3)	903.3 (897.5-910.8)	-0.19	0.845
Mean temperature (°C)	2.25 (1.37-3.8)	8.50 (1-12.2)	-1.20	0.229	8.7 (1.0-12.2)	7.45 (-0.1-12.2	-0.62	0.533	8.9 (0.52-12.25)	6.55 (0-10.9)	-1.17	0.241
Max temperature(°C)	11.2 (2.17-17.00)	12.3 (2.90-17.00)	-0.76	0.445	12.78 (2.82-17.0)	12.2 (2.82-17.00)	-0.15	0.880	12.3 (2.82-17.32)	10.9 (2.65-17.0)	-0.89	0.373
Min temprature (°C)	0.2 (-6.80-5.85)	3.05 (-1.10-7.62)	-1.81	0.069	2.4 (-0.8-7.6)	2.55 (-3.20-6.92	-0.64	0.517	3.5 (-1.8-8.25)	0.2 (-1.17-5.42)	-1.54	0.123

*All values are given as median (25-75 Percentile), **:Mann-Whitney U test was performed; 1– white blood cell, 2– neutrophil lymphocyte ratio 3– platelet lymphocyte ratio 4– maxiumum wind speed 5– C reactive protein

Table 3. Assessment of hemogram, biochemistry and climate parameters according to COHb classification

Parameters	Classification by COHb value			p*
	Mild	Moderate	Severe	
Hemogram and biochemistry parameters				
WBC[1](x1000 mm³)	9.02 (7.08-10.76)	9.44 (8.07-11.99)	11.05 (9.23-13.00)	0.170
NLR[2]	1.09 (0.46-2.92)	1.22 (0.6-2.47)	2.09 (1.24-3.54)	**0.019**
PLR[3]	70.6 (46.7-111.4)	100.6 (62.0-123.4)	106.2 (80.6-164.4)	**0.032**
CK(U/L)	102 (86.25-177.25)	114.5 (88.75-171.0)	126 (91-180)	0.815
CK-MB (U/L)	20 (17.5-36.5)	27 (22-33)	34 (23-43)	0.181
pH	7.38 (7.33-7.41)	7.36 (7.31-7.39)	7.32 (7.29-7.37)	**0.03**
COHb	2.1 (1.5-2.7)	15.45 (10.55-20.65)	31.45 (27.25-34.2)	**<0.001**
Lactate	1.7 (1.25-2.3)	2 (1.6-2.72)	2.7 (1.8-3.45)	0.07
Albumin (g/L)	48.95 (44.6-49.0)	44.7 (41.7-47.9)	41.75 (39.17-44.62)	0.239
CRP[4] (mg/L)	0.41 (0.19-6.22)	0.51 (0.29-1.36)	0.45 (0.22-1.87)	0.879
Troponin (pg/mL)	2.5 (1.7-4.8)	2.9 (1.8-6.37)	2.1 (1.8-4.6)	0.712
Climate parameters				
Maximum WS[5] (Knot)	6.7 (6.2-10.8)	9 (5.1-14.4)	6.95 (5.1-11.55)	0.694
Average WS (Knot)	1.4 (1.0-2.4)	2.15 (1.27-3.8)	1.55 (1.4-3.97)	0.203
Air pressure (mb)	904.7 (901.6-909.3)	903.95 (898.6-910.1)	905.6 (898.3-909.7)	0.709
Average temperture (°C)	9.0 (7.4-12.3)	6.55 (-0.4-12.2)	10.3 (2.72-12.2)	0.198
Maximum temparture (°C)	14.2 (13.05-19.0)	12.3 (8.5-17.0)	10.8 (1.9-17.0)	0.032
Minimum temparture (°C)	3.5 (3.1-8.1)	0.45 (-3.2-6.55)	1.15 (-0.67-8.6)	0.103

*: All values are given as median (25-75 Percentile) and Kruskal-Wallis test was performed; 1– white blood cell, 2– neutrophil lymphocyte ratio, 3– platelet lymphocyte ratio, 4– C reactive protein

Table 4. Relationship between age, source of poisoning, time of event-admission and gender factors according to COHb value classification

Features		Severe (COHb>25) n (%)*	Moderate (COHb: 25-5) n (%)	Mild (COHb<5) n (%)	p
Age	**0-5 year**	3 (2.4)	26 (20.8)	7 (5.6)	0.278
	>5 year	15 (12)	64 (51.2)	10 (11.2)	
Source	**Stove**	10 (8.9)	87 (77.7)	15 (13.4)	**0.001**
	Natural gass	2 (66.7)	0 (0.0)	1 (33.3)	
	Others¥	6 (60.0)	3 (30.0)	1 (10.0)	
Incident submission time	**0-2 hour**	15 (12)	54 (43.2)	12 (9.6)	0.144
	>2 hour	3 (2.4)	36 (28.8)	5 (4)	
Gender	**Female**	6 (4.8)	45 (36)	8 (7.2)	0.433
	Male	12 (9.6)	45 (36)	9 (6.4)	
Clinical symtomps	**Respiration**	4 (8.5)	32 (68.1)	11 (23.4)	**0.014**
	CNS**	10 (28.6)	24 (68.6)	1 (2.8)	
	GIS£	5 (11.6)	33 (76.7)	5 (11.6)	

*: n (%); n=number, %: percentile, ¥Others: exposure to hookah, fire, car exhaust fumes; **=CNS: central nervous system (headache, dizziness, drowsiness, confusion), GIS£: gastrointestinal system (vomiting, nausea, abdominal pain)

DISCUSSION

In this study, the relationship between pediatric cases of CO poisoning and systemic/immunoinflammatory markers and climate parameters was evaluated. Under 5 years of age, NLR was 1.08 (0.47-1.85), whereas it was 2.07 (1.30-3.55) above 5 years of age (p<0.001). Under 2 hour of admission time NLR was 1.51 (0.81-2.52), whereas it was 2.26 (1.28-3.75) above 2 hours (p=0.01). Severe Co poisoning appeared to be more common in the group with CNS findings (p=0.014). In relation to these data, a study conducted in Türkiye in 2013 (10) showed that symptoms were more severe in adolescents than in younger patients. A prospective study by Yalçın

et al. (11) demonstrated that an NLR value above 4 was significantly associated with the development of delayed neuropsychiatric syndrome (DNS), with an area under the curve (AUC) of 0.828, sensitivity of 78.2%, and specificity of 75.5 ($p<0.001$). These findings support the potential use of NLR as a low-cost, accessible biomarker not only for evaluating acute disease severity but also for identifying children at higher risk of long-term complications. When interpreted alongside patient age, time to hospital admission, and symptom presentation, NLR may enhance early risk assessment and guide management strategies in pediatric CO poisoning cases.

Our study found no statistically significant association between gender and poisoning severity ($p=0.433$). This finding aligns with existing literature reporting either comparable or slightly higher incidence rates among males (12,13). This likely reflects gender-specific exposure patterns rather than biological factors, as males in our region more frequently engage in high-risk activities like vehicle maintenance and outdoor combustion-related work. The lack of statistical significance despite numerical male predominance suggests that while exposure opportunities may vary by gender, clinical outcomes do not. These findings emphasize the need for prevention strategies targeting specific high-risk activities rather than demographic groups.

Our findings demonstrate a pronounced seasonal trend in CO poisoning incidence, with the majority of cases occurring during winter months (particularly in February) mirroring epidemiological patterns observed across diverse geographical regions. This temporal distribution aligns consistently with studies from Saudi Arabia (12), China (14) where 70% of cases clustered in winter months. The robust cross-cultural reproducibility of this phenomenon underscores its fundamental linkage to behavioral and environmental factors rather than regional peculiarities. The winter predominance reflects a confluence of risk determinants: heightened reliance on combustion-based heating systems, increased indoor confinement with inadequate ventilation, and the combustion inefficiency of fuels under low-temperature conditions. Particularly noteworthy is the February peak in our cohort, which may correlate with both the seasonal temperature nadir and cultural practices such as prolonged indoor heating during traditional winter periods. These observations collectively substantiate that CO poisoning represents not merely a clinical entity, but a socioclimatic phenomenon where environmental determinants interact with human behavioral patterns to create predictable risk windows. This evidence reinforces the imperative for targeted public health interventions including community education on ventilation practices and heating system maintenance to be intensified during these high-risk periods.

Our laboratory findings revealed a distinct biochemical progression associated with worsening clinical severity, characterized by elevated WBC counts, COHb, and lactate levels, coupled with decreasing pH, partial pressure of carbon dioxide (PCO_2), and oxygen (PaO_2). This constellation of abnormalities paints a comprehensive picture of the dual pathological mechanisms underlying severe CO intoxication: Firstly, profound tissue hypoxia resulting from CO's competitive binding to hemoglobin, and secondly, a consequent systemic inflammatory response. The marked lactate elevation (2.7 [1.8-3.45] mmol/L in severe cases) serves as a particularly sensitive indicator of cellular hypoxia, reflecting the shift to anaerobic metabolism when oxygen delivery becomes critically impaired. These findings align precisely with the work of Keleş et al. (15), who similarly demonstrated the strong predictive value of hyperlactatemia in assessing poisoning severity. The concordance between our results and established literature not only validates our methodological approach but also reinforces lactate's role as a key biomarker for both diagnostic and prognostic evaluation in CO poisoning. Furthermore, the observed leukocytosis and acid-base disturbances suggest that secondary inflammatory cascades significantly contribute to the pathophysiology, potentially explaining why some patients develop delayed neurological sequelae despite normalization of COHb levels. This biochemical profile underscores the importance of monitoring both oxygenation parameters and inflammatory markers in the acute management of CO intoxication.

Our study demonstrated significantly higher NLR and PLR ratios in severe CO poisoning ($p=0.019$ and 0.032, respectively), consistent with findings by Bağcı et al. (7). who reported similar increases in these inflammatory indices with worsening severity. NLR and PLR, derived from routine complete blood counts, have emerged as valuable systemic immune-inflammation markers (SIIs) for assessing disease progression in pediatric populations (6). Their rapid availability, cost-effectiveness, and widespread accessibility make them particularly useful in emergency settings. The strong association between elevated NLR/PLR and CO poisoning severity suggests these ratios may serve as early, practical biomarkers to guide clinical decision-making, especially in resource-limited environments where advanced diagnostics may be unavailable.

Our study revealed that higher maximum air temperatures were associated with milder CO poisoning cases ($p=0.032$), aligning with global observations that colder climates elevate poisoning risk (8,16,17). Notably, research from Taiwan quantifies this relationship, demonstrating a 10% reduction in CO poisoning risk per 1°C temperature increase (17). While our meteorological data—limited to daily averages rather than real-time measurements recluded precise temporal correlations, the consistent inverse trend between temperature and poisoning severity persists. This phenomenon likely stems from behavioral adaptations to cold weather: increased indoor heating, reduced ventilation, and prolonged occupancy in enclosed spaces, all of which exacerbate CO accumulation. These findings underscore temperature as a modifiable

environmental risk factor, reinforcing the need for targeted public health interventions during colder months.

Strength and Limitations

This study advances the understanding of CO poisoning in pediatric populations by integrating both biochemical markers and climate parameters a novel approach that extends beyond the conventional focus on laboratory values prevalent in prior research within our region. The inclusion of meteorological data provides a more comprehensive assessment of environmental risk factors, offering valuable insights for region-specific prevention strategies. However, several limitations must be acknowledged. The single-center design may restrict generalizability, while the modest sample size limits the power to detect subtler associations. Additionally, the reliance on daily climate averages rather than real-time measurements introduces potential temporal variability in exposure assessment. Future multicenter studies with larger cohorts and granular environmental data would help validate these findings and further elucidate the interplay between climatic factors and CO poisoning severity.

CONCLUSION

This study provides valuable insights into the relationship between systemic inflammatory markers (NLR, PLR), climatic factors, and the severity of pediatric CO poisoning. The findings demonstrate that elevated NLR and PLR ratios are strongly associated with disease severity, reinforcing their potential as accessible, cost-effective prognostic tools in emergency settings. Furthermore, the inverse correlation between maximum temperature and poisoning severity aligns with existing literature, highlighting the role of environmental conditions in modulating exposure risk. The study's results contribute meaningfully to the understanding of CO poisoning pathophysiology and risk stratification.

Financial disclosures: *The authors declared that this study has received no financial support.*

Conflict of interest: *The authors have no conflicts of interest to declare.*

Ethical approval: *Aksaray University Health Sciences Scientific Research Ethics Committee approved the decision dated 05.12.2024 and numbered SAGETIK134.*

REFERENCES

1. Rose JJ, Wang L, Xu Q, et al. Carbon monoxide poisoning: pathogenesis, management, and future directions of therapy. Am J Respir Crit Care Med. 2017;195:596-606. Erratum in: Am J Respir Crit Care Med. 2017;196:398-9.
2. Yonetani T, Park SI, Tsuneshige A, et al. Global allostery model of hemoglobin. Modulation of O(2) affinity, cooperativity, and Bohr effect by heterotropic allosteric effectors. J Biol Chem. 2002;277:34508-20.
3. The Lancet Public H. Carbon monoxide poisoning: largely preventable. Lancet Public Health. 2023;8:e827.
4. Hampson NB, Hauff NM. Risk factors for short-term mortality from carbon monoxide poisoning treated with hyperbaric oxygen. Crit Care Med. 2008;36:2523-7.
5. Teksam O, Gumus P, Bayrakci B, et al. Acute cardiac effects of carbon monoxide poisoning in children. Eur J Emerg Med. 2010;17:192-6.
6. Demirtas MS, Memiş S, Kocagözoğlu SG. Evaluation of hemoglobin, albumin, lymphocyte, and platelet score, neutrophil-to-lymphocyte ratio, and platelet-to-lymphocyte ratio in pediatric patients with rotavirus enteritis. J Clin Pract Res. 2025;47:28-35.
7. Bagci Z, Arslan A, Arslan D. The value of neutrophil: lymphocyte ratio and platelet: lymphocyte ratio in predicting clinical severity in children with carbon monoxide poisoning. Indian J Pediatr. 2021;88:1121-6.
8. Ruan HL, Deng WS, Wang Y, et al. Carbon monoxide poisoning: a prediction model using meteorological factors and air pollutant. BMC Proc. 2021;15:1.
9. Chen F, Ye Y, Wei Q, et al. Non-fire related carbon monoxide poisoning in Sichuan, China: a 9-year study (2008-2016). Iran J Public Health. 2019;48:458-64.
10. Kurt F, Bektaş Ö, Kalkan G, et al. Does age affect presenting symptoms in children with carbon monoxide poisoning?. Pediatr Emerg Care. 2013;29:916-21.
11. Yalçın G, Yalçın KE, Atasoy Hİ. Predictive value of complete blood count, venous blood gas measurements, and glucose/potassium ratio for delayed neuropsychiatric syndrome in children with acute carbon monoxide poisoning due to coal-burning stove. Turk Arch Pediatr. 2023;58:328-35.
12. Alharthy N, Alanazi A, Almoqaytib A, et al. Demographics and clinical characteristics of carbon monoxide poisoning for patients attending in the emergency department at a tertiary hospital in Riyadh, Saudi Arabia. Int J Emerg Med. 2024;17:25.
13. Al-Moamary MS, Al-Shammary AS, Al-Shimemeri AA, et al. Complications of carbon monoxide poisoning. Saudi Med J. 2000;21:361-3.
14. Zhang Y, Lu Q, Jia J, et al. Multicenter retrospective analysis of the risk factors for delayed neurological sequelae after acute carbon monoxide poisoning. Am J Emerg Med. 2021;46:165-9.
15. Keleş A, Demircan A, Kurtoğlu G. Carbon monoxide poisoning: How many patients do we miss?. Eur J Emerg Med. 2008;15:154-7.
16. Roca-Barcelo A, Crabbe H, Ghosh R, et al. Temporal trends and demographic risk factors for hospital admissions due to carbon monoxide poisoning in England. Prev Med. 2020;136:106104.
17. Wang CH, Shao SC, Chang KC, et al. Quantifying the effects of climate factors on carbon monoxide poisoning: a retrospective study in Taiwan. Front Public Health. 2021;9:718846.