

## Original Research

**Cite this article:** Sabak M, Gulen M, Satar S, Yildirim C, Yildiz AF and Zengin S (2024). The Prognostic Effect of Air Pollution in Patients Presenting to the Emergency Department with Carbon Monoxide Poisoning. *Disaster Medicine and Public Health Preparedness*, **18**, e126, 1–9 <https://doi.org/10.1017/dmp.2024.135>

Received: 03 January 2024

Revised: 13 April 2024

Accepted: 30 May 2024

### Keywords:


Air Quality Index; carbon monoxide poisoning; climate factors; weather parameters

### Corresponding author:

Muge Gulen;

Email: [muge-gulen@hotmail.com](mailto:muge-gulen@hotmail.com)

# The Prognostic Effect of Air Pollution in Patients Presenting to the Emergency Department with Carbon Monoxide Poisoning

Mustafa Sabak<sup>1</sup>, Muge Gulen<sup>2</sup> , Salim Satar<sup>2</sup>, Cuma Yildirim<sup>1</sup>, Ahmet Faruk Yildiz<sup>1</sup> and Suat Zengin<sup>1</sup>

<sup>1</sup>Gaziantep University, Faculty of Medicine, Department of Emergency Medicine, Gaziantep, Turkey and <sup>2</sup>Adana City Training and Research Hospital, Department of Emergency Medicine, Adana, Turkey

## Abstract

**Objective:** Weather conditions such as low air temperatures, low barometric pressure, and low wind speed have been linked to more cases of carbon monoxide (CO) poisoning. However, limited literature exists regarding the impact of air pollution. This study aims to investigate the relationship between outdoor air pollution and CO poisoning in 2 distinct cities in Turkey.

**Methods:** A prospective study was conducted at 2 tertiary hospitals, recording demographic data, presenting complaints, vital signs, blood gas and laboratory parameters, carboxyhemoglobin (COHb) levels, meteorological parameters, and pollutant parameters. Complications and outcomes were also documented.

**Results:** The study included 83 patients (Group 1 = 44, Group 2 = 39). The air quality index (AQI) in Group 2 ( $61.7 \pm 27.7$ ) (moderate AQI) was statistically significantly higher (dirtier AQI) than that in Group 1 ( $47.3 \pm 26.4$ ) (good AQI) ( $P = 0.018$ ). The AQI was identified as an independent predictor for forecasting the need for hospitalization (OR = 1.192, 95% CI: 1.036 - 1.372,  $P = 0.014$ ) and predicting the risk of developing cardiac complications (OR: 1.060, 95% CI: 1.017 - 1.104,  $P = 0.005$ ).

**Conclusions:** The AQI, derived from the calculation of 6 primary air pollutants, can effectively predict the likelihood of hospitalization and cardiac involvement in patients presenting to the emergency department with CO poisoning.

Carbon monoxide (CO) is a toxic gas that is emitted into the atmosphere as a result of incomplete combustion of carbon-containing components. Even at high concentrations, it is challenging to detect due to its colorless, tasteless, and odorless nature.<sup>1</sup> The 2022 Annual Report of the National Poison Data System (NPDS) by the American Association of Poison Control Centers revealed that there were 13 760 isolated cases of CO poisoning, with 11 857 being accidental. Among these cases, 5750 individuals received treatment at health centers and 63 resulted in death.<sup>2</sup> Although headache, dizziness, weakness, nausea, vomiting, and chest pain are common symptoms, patients may also present with nonspecific symptoms. This could lead to a misdiagnosis or a delayed diagnosis. The primary cause of poisonings in residential settings is the use of gasoline-powered appliances, including heating systems, cooking appliances, and electric generators.<sup>3</sup>

Another important feature of CO in the air is that it affects greenhouse gases associated with global warming and climate change. Greenhouse gases can lead to higher soil and water temperatures, which can result in severe weather conditions or storms.<sup>4</sup> The negative consequences of air pollution make it a top concern in the modern world, posing threats to both humans and the natural world. The World Health Organization (WHO) report identifies 6 primary air pollutants that contribute to air pollution, including particulate matter (PM<sub>2.5</sub>, PM<sub>10</sub>), sulfur dioxide (SO<sub>2</sub>), nitrogen dioxide (NO<sub>2</sub>), CO, and ozone (O<sub>3</sub>). The substantial amount of these substances presents a danger to the well-being of living creatures, as it can have severe impacts on both air quality and the surrounding environment, including soil and groundwater resources.<sup>5</sup> Air quality standards and guidelines have been adopted by the WHO and the Environmental Protection Agency (EPA) as tools for managing air quality and controlling different pollutants.<sup>6,7</sup> In concordance with this aim, the Ministry of Environment, Urbanization and Climate Change in Turkey has instituted a continuous monitoring center on the online platform. Its principal role is to facilitate easy retrieval of information for the populace on the air quality they are exposed to and to promote the comprehension of relevant data. The air quality index (AQI) was developed to classify the air quality of a particular location as *good*, *moderate*, *unhealthy for sensitive groups*, *unhealthy*, *very unhealthy*, or *dangerous*. This information is updated hourly based on data collected from the field.<sup>8</sup>

The purpose of this study was to determine the link between outdoor air pollution and the incidence of CO poisoning among patients admitted to emergency departments in 2 distinct cities in Turkey.

## Methods

### Study Design and Setting

This study was conducted prospectively at tertiary hospitals in 2 different cities. Patients in Adana province were labeled as Group 1, while patients in Gaziantep province were labeled as Group 2. The study was started after the approval of the local ethics committee. The study was conducted in accordance with the Declaration of Helsinki and good clinical practice. Written informed consent was obtained from the patients to participate in the study.

### Selection of Study Patients

The study included individuals who visited both emergency medicine clinics for CO poisoning and had COHb levels greater than 5% (for nonsmokers) and 10% (for smokers) in their blood gas results. The study did not include patients who were under 18 years old, pregnant, experiencing poisoning from multiple substances (cyanide, etc.), lost to follow-up, deceased upon arrival at the emergency department from CO poisoning, admitted more than 24 hours after exposure, intentionally poisoned, or had a history of nervous system disease (Figure 1).

### Data Collection

The study data were documented in the pre-established data collection form by 6 esteemed emergency medicine experts, namely MS, MG, SS, CY, AFY, and SZ. In addition to the demographic data of the patients, their complaints, time of poisoning, CO source, vital signs, blood gas and laboratory parameters, COHb levels, length of stay in the emergency department, and outcomes were recorded. Hyperglycemia (>200 mg/dL), leukocytosis (>11.0x10<sup>9</sup>/L), myocardial involvement (conditions affecting the heart, such as myocardial infarction, dysrhythmia, and heart failure), acute renal failure (an increase in serum creatinine level by 0.3 mg/dL or more within 48 hours), central nervous system depression, hypoxemia (<92%), and rhabdomyolysis complications were also recorded.

Venous blood gas COHb level was measured with a Radiometer ABL90 flex (Radiometer, Copenhagen, Denmark) brand blood gas analyzer.

Detailed weather forecast (temperature, humidity, and average wind speed) and climate parameters (AQI, PM<sub>2.5</sub>, PM<sub>10</sub>, CO, SO<sub>2</sub>, NO<sub>2</sub>, and O<sub>3</sub>) were collected for the days that the patients were admitted. In accordance with the Ministry of Environment, Urbanization, and Climate Change of the Republic of Turkey, the data were sourced from the subsequent websites: <https://www.mgm.gov.tr/tahmin/il-ve-ilceler.aspx?il=> and <https://www.havaizleme.gov.tr>, correspondingly (Figure 2).

## Outcomes

The primary outcome of the study was the prediction of the development of cardiac complications arising from CO poisoning by analyzing meteorological and air pollutant parameters.

The secondary outcome was the estimation of the correlation between meteorological and air pollutant parameters with blood COHb levels in patients who had CO poisoning.

## Statistical Analysis

The statistical evaluation of the data obtained in the study utilized the SPSS 22 software package (SPSS Inc, Chicago, Illinois, USA). Variables were divided into 2: categorical and continuous. Categorical data were presented as numbers and percentages and compared using the chi-square test. Whether continuous variables were normally distributed was determined using the Kolmogorov-Smirnov test. Continuous variables were presented with mean and standard deviation. The Kolmogorov-Smirnov test was used to compare the averages of the parameters examined. The paired sample *t* test was used to compare 2 groups when the variables were normally distributed in the evaluations made with the histogram, and the Mann-Whitney *U* test was used when the variables were not normally distributed. Multivariate analysis of air pollution and air temperature parameters was performed to predict the need for hospitalization of patients and to estimate the risk of developing cardiac complications. Pearson correlation analysis was used to explain the relationship between COHb level and meteorological and air pollutant parameters. The statistical significance level was set at *P* < 0.05.

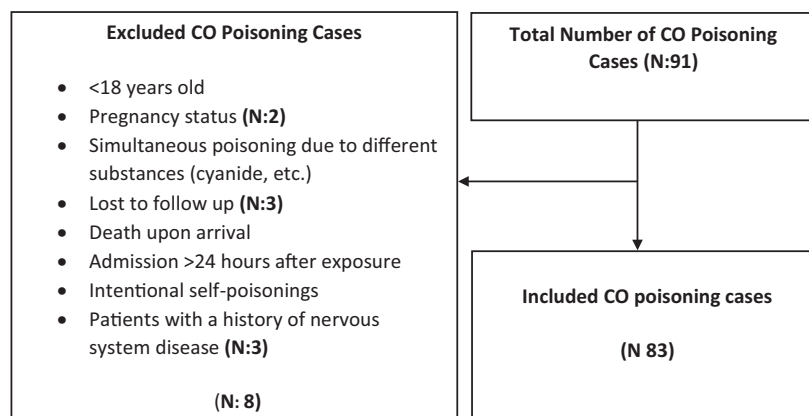


Figure 1. Flowchart of study.

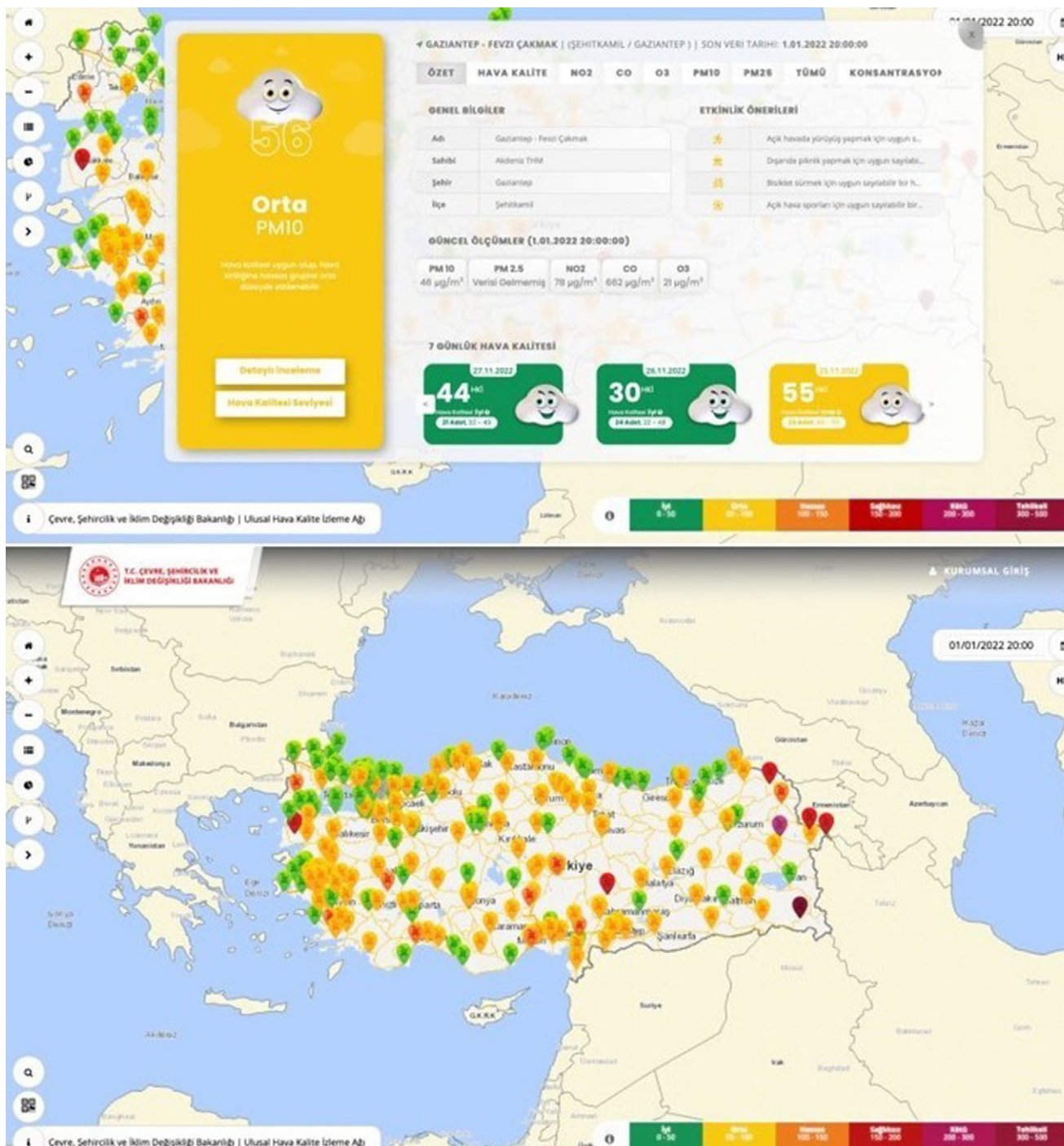


Figure 2. National Air Quality Monitoring Network and a sample of data pertaining to the province of Gaziantep on any given day.

**Results**

Eighty-three patients were included in the study. 50.6% (n = 42) of the patients were male, and the average age was 45.4 ± 18.6 years. Patients were most frequently admitted in January, 44.6% (n = 37) (P < 0.001), and equally during the day shift 39.8% (n = 33), and night shift, 39.8% (n = 33) (P = 0.260). The stove used for heating was the most common cause of poisoning, accounting for 78.3%. The most common presenting symptoms were headache (53%, n = 44), nausea and vomiting (48.2%, n = 40), and dizziness (37.3%, n = 31).

The most common comorbidity was hypertension (27.3%, n = 12) vs. 5.1%, n = 2), P = 0.007). When vital signs were examined, heart rate (96.2 ± 18.3 vs. 87.6 ± 17.4, P = 0.032), mean arterial pressure (93.4 ± 12.6 vs. 86.5 ± 11.1, P = 0.01), and respiratory rate (18.6 ± 3.4 vs. 16.9 ± 2.9, P = 0.016) were significantly higher in Group 1. The most common complications were hyperglycemia (25.3%, n = 21), leukocytosis (24.1%, n = 20), and myocardial involvement (24.1%, n = 20). The complication of acute renal failure was statistically significantly higher in Group 1 (15.9% vs. 2.6%, P = 0.04).

The length of stay in the emergency department ( $5.1 \pm 2.3$  vs.  $3 \pm 1$ ,  $P < 0.001$ ) and the ICU admission rate ( $22.7\%$  vs.  $5.1\%$ ,  $P = 0.023$ ) were statistically significantly higher in Group 1 (Table 1).

Upon admission, patients were evaluated for meteorological, pollutant, laboratory, and blood gas parameters, all of which are listed in Table 2. According to the meteorological parameters, the air temperature ( $7.8 \pm 4.7$  vs.  $4.8 \pm 3$ ,  $P = 0.001$ ) and wind speed ( $12.3 \pm 6.9$  vs.  $6.2 \pm 4.8$ ,  $P < 0.001$ ) of the center where Group 1 patients applied were statistically significantly higher than those in Group 2. The AQI cutoff values, which are determined based on the concentrations of pollutants in the air, are shown in Table 3. When the AQI was evaluated, the air quality in Group 2 ( $61.7 \pm 27.7$ ) (moderate AQI) was significantly higher than that in Group 1 ( $47.3 \pm 26.4$ ) (good AQI) ( $P = 0.018$ ). The data indicate that Group 2's AQI has a higher level of dirtiness. When the 2 groups

were compared in terms of pollutant parameters, Group 2 showed significantly higher levels of  $\text{NO}_2$  ( $89 \pm 38.5$  vs.  $43.5 \pm 36.6$ ,  $P < 0.001$ ) and  $\text{SO}_2$  ( $81.6 \pm 60.7$  vs.  $22 \pm 21.9$ ,  $P < 0.001$ ) and lower levels of  $\text{O}_3$  ( $41.7 \pm 22.5$  vs.  $27.9 \pm 22.2$ ,  $P = 0.006$ ) than Group 1. In Table 2, the analysis of air pollutants (CO,  $\text{PM}_{10}$ , and  $\text{PM}_{2.5}$ ) in different provinces is presented.

Multivariate analysis of meteorological and pollutant parameters to predict the need for hospitalization and the need for cardiac complications is shown in Table 4. It has been determined that the AQI is a reliable predictor for hospitalization (OR = 1.192, 95% CI: 1.036 - 1.372,  $P = 0.014$ ) and the risk of developing cardiac complications (OR = 1.060, 95% CI: 1.017 - 1.104,  $P = 0.005$ ) (Table 4).

The correlations between COHb levels and meteorological and air pollutant parameters are outlined in Table 5. When meteorological parameters were examined, it was found that the COHb

**Table 1.** Distribution of demographic and clinical data of CO poisoning cases by provinces

Variable	All patients (n = 83) n (%)	Group 1 (n = 44) n (%)	Group 2 (n = 39) n (%)	P value
<b>Male, n (%)</b>	42 (50.6)	22 (50)	20 (51.3)	0.907
<b>Age, mean<math>\pm</math>SD (years)</b>	45.4 $\pm$ 18.6	48.2 $\pm$ 20.2	42.2 $\pm$ 16.3	0.145
<b>Month of the CO poisoning, n (%)</b>				<b>&lt;0.001</b>
December	17 (20.5)	2 (4.5)	15 (38.5)	
January	37 (44.6)	25 (56.8)	12 (30.8)	
February	20 (24.1)	14 (31.8)	6 (15.4)	
March	9 (10.8)	3 (6.8)	6 (15.4)	
<b>Time of arrival at emergency room, n (%)</b>				0.260
Day shift (08:01–16:00)	33 (39.8)	19 (43.2)	14 (35.9)	
Evening shift (16:01–00:00)	17 (20.5)	11(25)	6 (15.4)	
Night shift (00:01–08:00)	33 (39.8)	14 (31.8)	19 (48.7)	
<b>Source of CO poisoning, n (%)</b>				0.140
Heating stove	65 (78.3)	37 (84.1)	28 (71.8)	
Barbecue	7 (8.4)	5 (11.4)	2 (5.1)	
Gas-fired combi boiler	6 (7.2)	1 (2.3)	5 (12.8)	
Fire area	4 (4.8)	1 (2.3)	3 (7.7)	
Water pipe	1(1.2)	0	1 (2.6)	
<b>Symptoms, n (%)</b>				
Headache	44 (53)	22 (50)	22 (56.4)	0.559
Nausea and vomiting	40 (48.2)	21 (47.7)	19 (48.7)	0.928
Dizziness	31 (37.3)	9 (20.5)	22 (56.4)	<b>0.001</b>
Syncope	16 (19.3)	8 (18.2)	8 (20.5)	0.788
Weakness	17 (20.5)	2 (4.5)	15 (38.5)	<b>&lt;0.001</b>
Dyspnea	12 (14.5)	4 (9.1)	8 (20.5)	0.140
Altered level of consciousness	10 (12)	7 (15.9)	3 (7.7)	0.251
Chest Pain	7 (8.4)	4 (9.1)	3 (7.7)	0.819
<b>Comorbidities, n (%)</b>				
Hypertension	14 (16.9)	12 (27.3)	2 (5.1)	<b>0.007</b>
Diabetes mellitus	10 (12)	5 (11.4)	5 (12.8)	0.839
Coronary artery disease	3 (3.6)	2 (4.5)	1 (2.6)	0.629

(Continued)



Table 1. (Continued)

Variable	All patients (n = 83) n (%)	Group 1 (n = 44) n (%)	Group 2 (n = 39) n (%)	P value
Chronic renal failure	2 (2.4)	2 (4.5)	0 (0)	0.178
Asthma	2 (2.4)	1 (2.3)	1 (2.6)	0.931
COPD	1 (1.2)	1 (2.3)	0 (0)	0.344
<b>Vital signs, mean±SD</b>				
HR, bpm	92.2±18.3	96.2±18.3	87.6±17.4	<b>0.032</b>
MAP, mmHg	90.2±12.3	93.4±12.6	86.5±11.1	<b>0.010</b>
RR, min	17.8±3.3	18.6±3.4	16.9±2.9	<b>0.016</b>
SO <sub>2</sub> , %	97.4±3	97.2±3.3	97.8±2.8	0.365
GCS	14.8±1.1	14.6±1.4	14.9±0.3	0.134
<b>Complications n (%)</b>				
Hyperglycemia	21 (25.3)	13 (29.5)	8 (20.5)	0.345
Leukocytosis	20 (24.1)	14 (31.8)	6 (15.4)	0.081
Myocardial involvement	20 (24.1)	14 (31.8)	6 (15.4)	0.081
Acute renal failure	8 (9.6)	7 (15.9)	1 (2.6)	<b>0.040</b>
CNS depression	7 (8.4)	6 (13.6)	1 (2.6)	0.070
Hypoxemia	4 (4.8)	3 (6.8)	1 (2.6)	0.366
Rhabdomyolysis	2 (2.4)	2 (4.5)	0 (0)	0.178
<b>Length of emergency room stay (hours)</b>	<b>4.1±2.1</b>	<b>5.1±2.3</b>	<b>3±1</b>	<b>&lt;0.001</b>
<b>Outcome</b>				
Discharge	71 (85.5)	34 (77.3)	37 (94.9)	<b>0.023</b>
Hospitalization ICU	12 (14.5)	10 (22.7)	2 (5.1)	
<b>Length of hospital stay (days)</b>	<b>5.8±2.4</b>	<b>5.5±2.6</b>	<b>7±1.4</b>	<b>0.449</b>

Group 1: Patients from Adana province. Group 2: Patients from Gaziantep province.

CO = carbon monoxide; COPD = chronic obstructive pulmonary disease; HR = heart rate; MAP = mean arterial pressure; RR = respiratory rate; SO<sub>2</sub> = oxygen saturation; CNS = central nervous system; GCS = Glasgow Coma Scale; ICU = intensive care unit.

Bold values indicates a statistically significant difference with a p value <0.05.

levels of the patients had a statistically significant but weak correlation with air humidity ( $r = 0.276$ ,  $P = 0.011$ ). When the pollutant parameters were examined, it was found that the COHb levels of the patients had a statistically significant but weak negative correlation with the air O<sub>3</sub> level ( $r = -0.218$ ,  $P = 0.048$ ).

## Discussion

This study reveals that AQI, calculated based on the concentration of pollutants in the air, is an independent predictor for determining the probability of hospitalization and cardiac complications in emergency department patients with CO poisoning.

The cause of CO poisoning cannot be attributed to a single factor. The severity of CO poisoning depends on the amount of CO exposure, the concentration of CO in the air breathed in, the duration of exposure, and the patient's breathing rate.<sup>9–11</sup> Both outdoor pollution (ambient air pollution) and indoor pollution (pollution produced by indoor fuels, exhaust, poor ventilation, leakage of outdoor air pollutants) affect the concentration of CO in inhaled air.<sup>12,13</sup> The presence of indoor smoke may result in an elevation of basal COHb levels.<sup>10,11</sup> This study indicates that despite the lower AQI in Adana province (Group 1), patients are

prone to extended stays in the emergency department and prolonged hospitalization due to the prevalence of indoor pollution.

The National AQI was developed by modifying the EPA AQI to national legislation and limit values. The AQI is calculated based on six basic pollutants: PM<sub>2.5</sub>, PM<sub>10</sub>, CO, SO<sub>2</sub>, NO<sub>2</sub>, and O<sub>3</sub>.<sup>8</sup> Particulate matter often forms in the atmosphere as a result of chemical reactions between various pollutants.<sup>14</sup> Particles with aerodynamic diameters of 2.5 μm (PM<sub>2.5</sub>) or 10 μm (PM<sub>10</sub>) seem to be the main cause of the cardiovascular effects observed from air pollution.<sup>15</sup> The review of several studies revealed that the cardiovascular death rates increased by about 1% for every 10 μg/m<sup>3</sup> rise in PM<sub>2.5</sub>.<sup>16</sup> The penetration of particles generally depends on their size. Particulate matter comprises small liquid or solid droplets that can be breathed in.<sup>17</sup>

After inhaling, particles with a diameter of 10 micrometers (PM<sub>10</sub>) can enter the lungs and even reach the circulatory system. Fine particles (PM<sub>2.5</sub>) pose a greater risk to health.<sup>18,19</sup> While it causes nasopharyngitis in the short term, it has been associated with cardiovascular diseases and infant deaths in the long term.<sup>12,15</sup> Individuals with asthma, pneumonia, diabetes, respiratory, and cardiovascular diseases may be more vulnerable to the effects of PM because it affects respiratory health and the immune system.<sup>20</sup> Ozone is a gas created from oxygen through high voltage electrical

**Table 2.** Distribution of meteorological parameters, pollutant parameters, and laboratory and blood gas parameters of CO poisoning cases by province

Variable	All patients (n=83)	Group 1 (n=44)	Group 2 (n=39)	P value
<b>Laboratory parameters, mean±SD</b>				
WBC	10.3±3.7	10.9±3.7	9.5±3.7	0.082
Glucose	129.4±54.7	131±65.3	127.6±40.2	0.779
Hs-cTn	817.3±3409.3	968.8±3520.8	650.4±3319.6	0.675
CRP	5.9±10.5	5.2±6.2	6.7±14	0.504
<b>Venous blood gas parameters, mean±SD</b>				
pH	7.35±0.1	7.34±0.1	7.36±0.1	0.513
HCO <sub>3</sub> mmHg	22.8±3.9	22.8±3.6	22.8±4.2	0.987
COHb %	20.5±8.2	21.8±8.5	19±7.7	0.125
Lactate mmol/L	2.5±1.7	2.6±1.9	2.3±1.4	0.447
<b>Meteorological parameters, mean±SD</b>				
Temperature °C	6.4±4.2	7.8±4.7	4.8±3	<b>0.001</b>
Humidity %	60.7±19.9	56.9±21.7	64.9±16.9	0.062
Wind speed m/s	9.4±6.7	12.3±6.9	6.2±4.8	<b>&lt;0.001</b>
<b>Pollutant parameters, mean±SD</b>				
Air quality index	54.1±27.8	47.3±26.4	61.7±27.7	<b>0.018</b>
CO µg/m <sup>3</sup>	1031.9±925.5	955.2±1032.1	1118.5±792.8	0.426
SO <sub>2</sub> µg/m <sup>3</sup>	50±53.4	22±21.9	81.6±60.7	<b>&lt;0.001</b>
NO <sub>2</sub> µg/m <sup>3</sup>	64.9±43.8	43.5±36.6	89±38.5	<b>&lt;0.001</b>
PM <sub>10</sub> µg/m <sup>3</sup>	57.7±61.2	63.9±68	50.7±52.4	0.330
PM <sub>2.5</sub> µg/m <sup>3</sup>	30.4±29.4	31.1±31.8	29.7±26.9	0.826
O <sub>3</sub> µg/m <sup>3</sup>	35.2±23.2	41.7±22.5	27.9±22.2	<b>0.006</b>

Group 1: Patients from Adana province. Group 2: Patients from Gaziantep province.

WBC = white blood cell; Hs-cTn = high sensitive cardiac troponin; CRP = C-reactive protein; HCO<sub>3</sub> = bicarbonate; COHb = carboxyhemoglobin; CO = carbon monoxide; SO<sub>2</sub> = sulfur dioxide; NO<sub>2</sub> = nitrogen dioxide; PM = particulate matter; O<sub>3</sub> = ozone.

Normal range of the parameters: WBC: 3.7-8.7x10<sup>3</sup> /µL, glucose: 74-106 mg/dL, Hs-cTn: 0-11.6 ng/L, CRP: 0-5 mg/L, pH: 7.35-7.45, COHb: 0.5-1.5%, HCO<sub>3</sub>: 21.8-26.2 mmol/L, lactate: 0.5-1.6 mmol/L. National cutoff values: Air quality index: good (0-50), moderate (50-100), unhealthy for sensitive groups (100-150), unhealthy (150-200), very unhealthy (200-300), hazardous (300-500) CO: 10.000 µg/m<sup>3</sup> (8h average) SO<sub>2</sub>: 350 µg/m<sup>3</sup> (1h average), NO<sub>2</sub>: 250 µg/m<sup>3</sup> (1h average), PM<sub>10</sub>: 50µg/m<sup>3</sup> (24h average), O<sub>3</sub>: 120 µg/m<sup>3</sup> (8h average).

Bold values indicates a statistically significant difference with a p value <0.05.

**Table 3.** Air Quality Index (AQI) cut-off values\*

AQI	SO <sub>2</sub> (µg/m <sup>3</sup> )		NO <sub>2</sub> (µg/m <sup>3</sup> )		CO (µg/m <sup>3</sup> )		O <sub>3</sub> (µg/m <sup>3</sup> )		PM <sub>10</sub> (µg/m <sup>3</sup> )	PM <sub>2.5</sub> (µg/m <sup>3</sup> )	
	1h average	24h average	1h average	8h average	8h average	1h average	8h average	1h average	24h average	24h average	
0-50	0-93		0-102	0-5.2	0-108				0-54	0.0-12.0	Good
51-100	94-200		103-192	5.3-11	109-248				55-154	12.1-35.4	Moderate
101-150	201-493		193-689	11.1-14.5			249-328		155-254	35.5-55.4	Unhealthy for Sensitive Groups
151-200	494-810		690-1242	14.6-18			329-408		255-354	55.5-150.4	Unhealthy
201-300		811-1609	1243-2380	18.1-35.5			409-808		355-424	150.5-250.4	Very Unhealthy
301-400		1610-2141	2381-3145	35.6-47.1			809-1008		425-504	250.5-350.4	Hazardous
401-500		2142-2674	3146-3910	47.2-58.8			1009-1208		505-604	350.5-500.4	Hazardous

\*The national air quality index (AQI) was created by adapting the Environmental Protection Agency (EPA) AQI to our national legislation and limit values. AQI is calculated for 6 basic pollutants: particulate matter (PM<sub>2.5</sub>, PM<sub>10</sub>), carbon monoxide (CO), sulfur dioxide (SO<sub>2</sub>), nitrogen dioxide (NO<sub>2</sub>), and ozone (O<sub>3</sub>).

**Table 4.** Multivariate analysis to predict the need for hospitalization of patients and the risk of developing cardiac complications

	Need for hospitalization			Cardiac complication		
	OR	95% CI	P value	OR	95% CI	P value
<b>Air quality index</b>	1.192	1.036–1.372	<b>0.014</b>	1.060	1.017–1.104	<b>0.005</b>
<b>SO<sub>2</sub></b>	0.991	0.956–1.026	0.607	1.015	0.998–1.032	0.094
<b>NO<sub>2</sub></b>	1.005	0.979–1.032	0.707	0.997	0.978–1.017	0.760
<b>CO</b>	0.999	0.995–1.003	0.526	0.999	0.996–1.001	0.210
<b>O<sub>3</sub></b>	1.074	0.985–1.171	0.106	1.006	0.965–1.048	0.788
<b>PM<sub>10</sub></b>	0.939	0.860–1.026	0.162	0.977	0.939–1.017	0.258
<b>PM<sub>2.5</sub></b>	0.925	0.804–1.063	0.272	0.976	0.910–1.047	0.495
<b>Humidity</b>	0.951	0.888–1.018	0.149	1.003	0.972–1.035	0.865
<b>Wind speed</b>	1.245	0.990–1.566	0.061	1.092	0.990–1.205	0.078
<b>Temperature</b>	1.418	0.915–2.196	0.118	1.159	0.956–1.405	0.133

OR = odds ratio; CI = confidence interval; COHb = carboxyhemoglobin; CO = carbon monoxide; SO<sub>2</sub> = sulfur dioxide; NO<sub>2</sub> = nitrogen dioxide; PM = particulate matter; O<sub>3</sub> = ozone. National cutoff values: Air quality index: good (0-50), moderate (50-100), unhealthy for sensitive groups (100-150), unhealthy (150-200), very unhealthy (200-300), hazardous (300-500) CO:10.000 µg/m<sup>3</sup> (8h average) SO<sub>2</sub>: 350 µg/m<sup>3</sup> (1h average), NO<sub>2</sub>: 250 µg/m<sup>3</sup> (1h average), PM<sub>10</sub>:50µg/m<sup>3</sup> (24h average), O<sub>3</sub>: 120 µg/m<sup>3</sup> (8h average).

**Table 5.** Correlation values between COHb levels and meteorological and pollutant parameters

	COHb Level	
	r	P
<b>Meteorological Parameters</b>		
Temperature °C	-0.079	0.476
Humidity %	0.276	<b>0.011</b>
Wind speed m/s	0.021	0.847
<b>Pollutant parameters</b>		
Air quality index	0.012	0.913
CO mg/m <sup>3</sup>	0.005	0.962
SO <sub>2</sub> µg/m <sup>3</sup>	-0.086	0.437
NO <sub>2</sub> µg/m <sup>3</sup>	0.082	0.460
PM <sub>10</sub> µg/m <sup>3</sup>	-0.039	0.726
PM <sub>2.5</sub> µg/m <sup>3</sup>	-0.019	0.862
O <sub>3</sub> µg/m <sup>3</sup>	-0.218	<b>0.048</b>

r: Correlation coefficient

COHb = carboxyhemoglobin; CO = carbon monoxide; SO<sub>2</sub> = sulfur dioxide; NO<sub>2</sub> = nitrogen dioxide; PM = particulate matter; O<sub>3</sub> = ozone.

Bold values indicates a statistically significant difference with a p value <0.05.

discharge.<sup>21</sup> It is taken by inhalation. It affects the upper layers of the skin, tear ducts, and lungs.<sup>22,23</sup> Ozone increases DNA damage in epidermal keratinocytes, leading to biochemical, morphological, functional, and immunological disorders.<sup>24</sup> Automobile engines emit NO<sub>2</sub>, a harmful pollutant associated with traffic.<sup>25,26</sup> Inhaling at high levels (above 0.2 ppm) can cause coughing, wheezing, difficulty breathing, bronchospasm, and even pulmonary edema. In addition, high concentrations may have an effect on T-cells that play a crucial role in immune response, including CD<sup>8+</sup> cells and natural killer cells. Chronic exposure can lead to chronic lung disease.<sup>27</sup> The release of SO<sub>2</sub> is a hazardous byproduct commonly associated with the burning of fossil fuels or industrial

processes. The level of the annual SO<sub>2</sub> standard is 0.03 ppm.<sup>28</sup> Inhaling can cause shortness of breath, wheezing, acute bronchitis, and increased mucus secretion. It can cause urticaria, lacrimation, corneal opacity, and worsening of pre-existing cardiovascular diseases.<sup>27</sup> Air quality index is calculated based on the proportions of these 6 harmful gases in the air.

Carbon monoxide concentrations usually range from 0.02 to 1.0 ppm in clean air. However, in urban areas, CO concentrations can increase 10-fold during periods of atmospheric stagnation, such as when temperature inversions occur in winter or in situ air masses form in summer.<sup>29,30</sup> Research in the literature has extensively explored the impact of meteorological factors such as temperature, humidity, and wind speed on CO poisoning.<sup>31</sup> In recent years, studies have been evaluating cases of out-of-hospital cardiac arrest and CO poisoning, as well as air pollutants such as PM<sub>2.5</sub>, PM<sub>10</sub>, CO, SO<sub>2</sub>, NO<sub>2</sub>, and O<sub>3</sub>, along with meteorological factors.<sup>9-15</sup> In the study on CO poisoning, the researchers attempted to estimate the elevated risk of poisoning by creating models that incorporated both meteorological and pollutant parameters. The model, which considers both atmospheric conditions and pollutant variables, is effective in predicting cases of CO poisoning. It outperforms the model based only on weather factors in terms of precision and forecasting potential for severe poisoning levels.<sup>9</sup> This study differs from previous studies in that it examined the potential of meteorological factors and air pollution to predict the onset of complications and the likelihood of hospitalization. This study concluded that AQI was a significant factor in predicting both hospitalization and myocardial involvement. This discovery has the potential to be advantageous in mitigating unintentional CO poisoning, which carries a significant fatality risk. By including AQI value estimates in weather forecasts, public awareness of potential CO poisoning can be heightened.<sup>32</sup> Moreover, the dissemination of public service warnings through public transportation, news releases, and government surveillance can effectively reduce the occurrence of poisoning incidents, particularly during storms.<sup>33</sup> Additionally, imparting training in schools to heighten cognizance about CO poisoning and techniques for diminishing CO production in both indoor and outdoor settings will serve as an efficacious measure in

preventing both atmospheric pollution and CO poisoning in its nascent stages. In the event of poisoning, the primary objective is to reduce the number of fatalities by promptly and precisely identifying and treating the condition.

### Limitations

This study had some limitations. The first limitation regards the sample group. The study gathered data from 2 tertiary hospitals situated in 2 distinct provinces. These 2 hospitals rank among the biggest in the area. Nevertheless, other hospitals in the same area may have also seen instances of poisoning. This situation might have impeded the availability of a comprehensive dataset. The second limitation was related to continuous monitoring centers. Meteorological observation stations provide information by collecting average measurements of specific districts within each metropolis. The data were procured from the surveillance hubs within the vicinities of the patient's residence or the immediate vicinity.

### Conclusion

This study highlights the critical importance of air pollution in emergency department visits due to CO poisoning. The identification of the AQI as an independent predictor for hospitalization necessity and cardiac complications in patients with CO poisoning presenting at the emergency department highlights the significant impact of environmental factors on public health. These findings provide a fundamental guide for the development of public health strategies and the implementation of measures to improve air quality. Combating air pollution is urgent not only for the environment but also to protect the health of individuals.

**Data availability statement.** The datasets generated and/or analyzed during the current study are available from the corresponding author on reasonable request.

**Acknowledgments.** None.

**Author contribution.** MG, MS: Concept, review, data searching, design, statistical extractions, analysis, and final approval. SS, CY, AFY, SZ: Concept, review, analysis, data searching, analysis, and final approval.

**Competing interest.** The authors have no conflicts of interest to declare.

**Ethical standard.** The Ethics Committee of Gaziantep University of Medical Sciences, (Meeting date: February 23, 2022, Ethics committee decision no:2022/09) approved this study.

### References

1. Tomaszewski C. Carbon monoxide. In: Nelson LS, Howland MA, Lewin NA, Smith SW, Goldfrank LR, Hoffman RS, eds. *Goldfrank's Toxicologic Emergencies*. Vol 1. 11th ed. McGraw Hill; 2019:1663–1675.
2. Gummin DD, Mowry JB, Beuhler MC, et al. 2022 Annual Report of the National Poison Data System<sup>®</sup> (NPDS) from America's Poison Centers<sup>®</sup>: 40th Annual Report. *Clin Toxicol (Phila)*. 2023 Oct;**61**(10):717–939. doi: 10.1080/15563650.2023.2268981
3. Sircar K, Clower J, Shin M kyong, et al. Carbon monoxide poisoning deaths in the United States, 1999 to 2012. *Am J Emerg Med*. 2015;**33**(9): 1140–1145. doi:10.1016/j.ajem.2015.05.002
4. Emberson LD, Pleijel H, Ainsworth EA, et al. Ozone effects on crops and consideration in crop models. *Eur J Agron*. 2018;**100**:19–34. doi:10.1016/j.eja.2018.06.002
5. World Health Organization. WHO Global Air Quality Guidelines. Particulate Matter (PM<sub>2.5</sub> and PM<sub>10</sub>), Ozone, Nitrogen Dioxide, Sulfur Dioxide and Carbon Monoxide. Geneva. Published 2021. Accessed December 2, 2022. <https://iris.who.int/bitstream/handle/10665/345329/9789240034228-eng.pdf>
6. World Health Organization. Type of Pollutants. Publishing Date: 22 September 2021. Accessed October 5, 2023. <https://www.who.int/teams/environment-climate-change-and-health/air-quality-and-health/health-impacts/types-of-pollutants>
7. Newlands M. *Environmental Activism, Environmental Politics, and Representation: The Framing of the British Environmental Activist Movement*. University of East London; 2013. doi:<https://doi.org/10.15123/PUB.3046>
8. T.C. Republic of Türkiye Ministry of Environment U and CC. Ulusal Hava Kalite İzleme Ağı. Accessed December 3, 2023. <https://www.havaizleme.gov.tr>
9. Ruan HL, Deng WS, Wang Y, et al. Carbon monoxide poisoning: a prediction model using meteorological factors and air pollutant. *BMC Proc*. 2021;**15**. doi:10.1186/s12919-021-00206-7
10. Varon J, Marik PE, Fromm RE, et al. Selected topics: critical care carbon monoxide poisoning: a review for clinicians. *J Emerg Med*. 1999;**17**(1): 87–93. doi:[https://doi.org/10.1016/S0736-4679\(98\)00128-0](https://doi.org/10.1016/S0736-4679(98)00128-0)
11. Scherer G. Carboxyhemoglobin and thiocyanate as biomarkers of exposure to carbon monoxide and hydrogen cyanide in tobacco smoke. *Exp Toxicol Pathol*. 2006;**58**(2-3):101–124. doi:10.1016/J.ETP.2006.07.001
12. Manisalidis I, Stavropoulou E, Stavropoulos A, et al. Environmental and health impacts of air pollution: a review. *Front Public Health*. 2020;**8**. doi: 10.3389/fpubh.2020.00014
13. Exposure Guidelines for Residential Indoor Air Quality: A Report of the Federal. Minister of Supply and Services. 1995. Accessed December 3, 2023. <https://publications.gc.ca/collections/Collection/H46-2-90-156E.pdf>
14. Wilson WE, Suh HH. Fine particles and coarse particles: concentration relationships relevant to epidemiologic studies. *J Air Waste Manage Assoc*. 1997;**47**(12):1238–1249. doi:10.1080/10473289.1997.10464074
15. Dennekamp M, Akram M, Abramson MJ, et al. Outdoor air pollution as a trigger for out-of-hospital cardiac arrests. *Epidemiology*. 2010;**21**(4): 494–500. doi:10.1097/EDE.0b013e3181e093db
16. Pope CA, Dockery DW. Health effects of fine particulate air pollution: lines that connect. *J Air Waste Manage Assoc*. 2006;**56**(6):709–742. doi: 10.1080/10473289.2006.10464485
17. Cheung K, Daher N, Kam W, et al. Spatial and temporal variation of chemical composition and mass closure of ambient coarse particulate matter (PM<sub>10-2.5</sub>) in the Los Angeles area. *Atmos Environ*. 2011;**45**(16): 2651–2662. doi:10.1016/J.ATMOENV.2011.02.066
18. Kelishadi R, Poursafa P. Air pollution and non-respiratory health hazards for children. *Arch Med Sci*. 2010;**6**(4):483. doi:10.5114/AOMS.2010.14458
19. Zhang L, Yang Y, Li Y, et al. Short-term and long-term effects of PM<sub>2.5</sub> on acute nasopharyngitis in 10 communities of Guangdong, China. *Sci Total Environ*. 2019;**688**:136–142. doi:10.1016/j.scitotenv.2019.05.470
20. Current and Forecasted Air Quality in New Hampshire. Environmental Fact Sheet. New Hampshire Department of Environmental Services. Published 2019. Accessed December 2, 2023. <https://www.des.nh.gov/organization/commissioner/pip/factsheets/ard/documents/ard-16.pdf>
21. Bezirtzoglou E, Alexopoulos A. Ozone history and ecosystems: a goliath from impacts to advance industrial benefits and interests, to environmental and therapeutical strategies. In: Bakker SH, ed. *Ozone Depletion, Chemistry and Impacts*. Nova Science Publishers; 2009:135–145.
22. Amann M, Derwent D, Forsberg B, et al. Health Risks of Ozone from Long-Range Transboundary Air Pollution. 2008. Accessed December 2, 2023. <https://iris.who.int/bitstream/handle/10665/326496/9789289042895-eng.pdf?sequence=1>
23. Hatch GE, Slade R, Harris LP, et al. Ozone dose and effect in humans and rats. A comparison using oxygen-18 labeling and bronchoalveolar lavage. *Am J Respir Crit Care Med*. 1994;**150**(3):676–683. doi:10.1164/ajrccm.150.3.8087337
24. McCarthy JT, Pelle E, Dong K, et al. Effects of ozone in normal human epidermal keratinocytes. *Exp Dermatol*. 2013;**22**(5):360–361. doi:10.1111/exd.12125
25. Richmond-Bryant J, Chris Owen R, Graham S, et al. Estimation of on-road NO<sub>2</sub> concentrations, NO<sub>2</sub>/NO<sub>x</sub> ratios, and related roadway



- gradients from near-road monitoring data. *Air Qual Atmos Health*. 2017;**10**(5):611–625. doi:10.1007/s11869-016-0455-7
26. **Hesterberg TW, Bunn WB, McClellan RO**, et al. Critical review of the human data on short-term nitrogen dioxide (NO<sub>2</sub>) exposures: evidence for NO<sub>2</sub> no-effect levels. *Crit Rev Toxicol*. 2009;**39**(9):743–781. doi:10.3109/10408440903294945
27. **Chen TM, Kuschner WG, Gokhale J**, et al. Outdoor air pollution: nitrogen dioxide, sulfur dioxide, and carbon monoxide health effects. *Am J Med Sci*. 2007;**333**(4):249–256. doi:10.1097/MAJ.0b013e31803b900f
28. **U.S. Environmental Protection Agency**. Reviewing National Ambient Air Quality Standards (NAAQS). Accessed December 2, 2023. [https://www3.epa.gov/ttn/naaqs/standards/so2/s\\_so2\\_history.html](https://www3.epa.gov/ttn/naaqs/standards/so2/s_so2_history.html)
29. **National Ambient Air Quality Objectives for Carbon Monoxide: Desirable, Acceptable and Tolerable Levels**. CEPA/FPAC Working Group on Air Quality Objectives and Guidelines. 1994. Accessed December 2, 2023. <https://publications.gc.ca/Collection/En42-17-8-1994E.pdf>
30. **Campbell ME, Benson BA, Muir MA**. Urban air quality and human health: a Toronto perspective. *Can J Public Health*. 1995;**86**(5):351–357.
31. **Lin CM, Liao CM**. Temperature-dependent association between mortality rate and carbon monoxide level in a subtropical city: Kaohsiung, Taiwan. *Int J Environ Health Res*. 2009;**19**(3):163–174. doi:10.1080/09603120802460384
32. **Gijsenbergh FP, Vispoel M, Poppe H**, et al. Weather influence on the prevalence of carbon monoxide intoxications. *Acta Clin Belg Suppl*. 1990;**13**:96–97.
33. **Hampson NB, Piantadosi CA, Thom SR**, et al. Practice recommendations in the diagnosis, management, and prevention of carbon monoxide poisoning. *Am J Respir Crit Care Med*. 2012;**186**(11):1095–1101. doi:10.1164/rccm.201207-1284CI