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Could Vital Signs Predict Carbon Monoxide Intoxication?

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OBJECTIVE: This retrospective study examined the correlation between carboxyhaemoglobin (COHb) levels and vital signs in patients with carbon monoxide (CO) intoxication. METHODS: Over a 10-year period, patients > 16 years of age who presented to the emergency department due to CO intoxication were included. Age, gender, comorbidities, month/year of presentation, presenting symptoms, vital signs, blood pH, COHb level, treatment and outcome were recorded. RESULTS: In total, 476 patients were included. The mean ± SD age was 36.22 ± 13.65 years; 96.4% of the patients had a normal Glasgow Coma Scale score, 91.0% had normal blood pressure and 80.0% had a normal heart rate. COHb levels were stratified into three groups: < 10% (*n* = 39), 10 – 20% (*n* = 106) and > 20% (*n* = 205); levels could not be obtained in the remaining 126 patients. In patients with COHb levels > 20%, 34 (16.6%) had alkalosis and nine (4.4%) had acidosis. Among patients with COHb levels > 20%, 140 (68.3%) had normal vital signs. CONCLUSIONS: Vital signs cannot be used as a prognostic marker of CO intoxication and, therefore, patients must be monitored closely.

KEY WORDS: Carbon monoxide (CO) intoxication; Vital signs; Alkalosis; Acidosis; Prognosis

Introduction

Carbon monoxide (CO) is a tasteless, odourless and highly toxic gas that is produced by incomplete combustion of hydrocarbons.[1] The symptoms of CO intoxication are nonspecific, which means that diagnosis can only be made following suspicion.[2 – 4] Thus, the precise incidence of CO intoxication worldwide is unknown. CO has been described as "the unnoticed poison of the 21st Century".[5]

A wide spectrum of symptoms is associated with CO intoxication, and symptoms can mimic those of other common diseases such as nonspecific viral illness, flu-like syndrome and hypertensive attack.[6,7] The primary symptoms are headache/dizziness, vertigo, fatigue, nausea/vomiting, palpitation and angina pectoris.[8 – 11] Rarely, acute exposure to CO can cause myocardial infarction, cardiogenic shock or pulmonary oedema, whereas subacute exposure can cause mania or Parkinsonism.[12 – 18]

Hampson and Hauff[19] reported that there was no correlation between carboxyhaemoglobin (COHb) level and clinical presentation in cases of CO intoxication, whereas Çevik *et al.*[20] found a correlation

between the poisoning severity score and COHb level. The present study examined the correlation between COHb level and vital signs in patients with CO intoxication.

Patients and methods

STUDY POPULATION

This retrospective study included consecutive patients > 16 years of age who presented to the Department of Emergency Medicine, School of Medicine, Hacettepe University, Ankara, Turkey, due to CO intoxication, between January 2000 and December 2010. There were no other specific inclusion or exclusion criteria.

The study protocol was approved by the Hacettepe University School of Medicine Ethics Committee (HEK 08/130), Ankara, Turkey. As this was a retrospective study, informed consent from patients was not required.

STUDY ASSESSMENTS

Patient age, gender, comorbidities, the month and year of presentation, presenting symptoms, vital signs, blood pH, COHb level, treatment and outcome were retrieved from patients' records. Patients with a systolic and diastolic blood pressure of ≥ 140 mmHg and ≥ 80 mmHg, respectively, were considered to have hypertension. A heart rate of 60 – 100 beats/min was considered normal, < 60 beats/min was considered as bradycardia, and > 100 beats/min was considered tachycardia. A blood pH value of 7.35 – 7.45 was considered normal, < 7.35 was considered as acidosis and > 7.45 was considered as alkalosis.

Patients' COHb levels had been obtained from arterial blood gas analyses using a cobas® b 221 Blood Gas system (Roche, Basel, Switzerland), and patients were stratified into three groups according to their COHb levels as follows: < 10%, 10 – 20%, and > 20%. Data concerning the causes of CO intoxication were not contained in the patients' records. In patients in whom COHb levels could not be obtained, the diagnosis was based on anamnesis and findings of physical examination. The Glasgow Coma Scale (GCS) was used to assess each patient's neurological status.

STATISTICAL ANALYSES

Statistical analyses were carried out using the SPSS® software package, version 15.0 (SPSS Inc., Chicago, IL, USA) for Windows®. Numerical variables are shown as mean ± SD, and qualitative variables are shown as number and percentage. The χ^2-test and Pearson's correlation test (r) were used to determine differences between qualitative variables. A P-value of < 0.05 was considered to be statistically significant.

Results

A total of 476 patients were included in the study. The mean age ± SD was 36.22 ± 13.65 years and 290 patients (60.9%) were female; 278 (58.4%) patients presented between 20.00 h and 08.00 h, and 105 (22.1%) presented during January across the 10-year study period.

The most common complaints were headache/dizziness (298 patients; 62.6%); nausea/vomiting (79 patients; 16.6%), and syncope (76 patients; 16.0%). One patient was dead at the time of presentation to the emergency department. GCS scores were normal in 459 (96.4%) patients and below normal in 17 (3.6%) patients. Blood pressure was normal in 433 (91.0%) patients, low in 17 (3.6%), and high in 26 (5.5%) patients. In total, 381 (80.0%) patients had a normal heart rate, 91 (19.1%) had a heart rate > 100 beats/min, and four (0.8%) had a heart rate < 60 beats/min. Based on arterial blood gas analyses, pH was normal in 357 (75.0%) of the patients, 15 (3.2%) of the patients had acidosis and 55 (11.6%) had alkalosis.

Due to technical problems, arterial blood

gas analyses in 49 (10.3%) patients, and COHb levels in 126 (26.5%) patients, could not be obtained. In the patients whose COHb levels could not be obtained, diagnoses were based on anamnesis and physical examination. In patients in whom COHb levels were obtained (n = 350), the levels were: > 20%, n = 205 (58.6%); 10 – 20%, n = 106 (30.2%); < 10%, n = 39 (11.1%). Distribution of the type of presenting symptom according to the patient's COHb level is given in Table 1. Of the 205 patients with a COHb level > 20%, 140 (68.3%) had normal vital signs, 34 (16.6%) had alkalosis and nine (4.4%) had acidosis (Table 2).

Oxygen treatment was administered to all patients in the present study on presentation to the emergency department. Hyperbaric oxygen treatment was administered to 25 (5.3%) of the patients. In total, 458 (96.2%) of the patients were discharged from the emergency department and 16 (3.4%) were hospitalized. A correlation was observed between increased rate of hospital admissions due to syncope and increased COHb level (r = 0.63). There was no association between vital signs and COHb level. The mortality rate was 0.4% (two patients).

Discussion

As reported elsewhere,[1] the most common complaint among patients presenting to the emergency department and recruited to the present study was headache/dizziness, followed by syncope and nausea/vomiting. The hospital admission rate due to syncope increased as the COHb level increased, which was similar to the finding of Keleş *et al.*[21] Among patients included in the present study, 43.1% had a COHb level > 20% and the mortality rate was 0.4%, whereas Hampson and Hauff[19] reported rates of 62% and 2.6%, respectively. The most common period of admission to the emergency department was January, possibly as this is the coldest time of year in Ankara, and is the time when the use of stoves for heating is high.

Oxygen treatment was administered to all patients on presentation to the emergency department. Initiation of oxygen treatment during the early phase of CO intoxication – and hyperbaric oxygen treatment when indicated – decreases mortality by lowering the distribution of CO to the tissues. According to Hampson and Hauff,[19] acidosis increases in severity as the COHb level increases. In the present study, 4.4% and

TABLE 1:
Distribution of the type of presenting symptom according to the carboxyhaemoglobin (COHb) level among patients (n = 350) who presented to the emergency department with carbon monoxide intoxication

	COHb level		
Presenting symptom	**< 10% (n = 39)**	**10 – 20% (n = 106)**	**> 20% (n = 205)**
Headache/dizziness	24	76	117
Nausea/vomiting	10	13	33
Fatigue	0	1	2
Syncope	4	16	43
Coma	0	0	3
Cardiac arrest	0	0	1
Palpitation	0	0	4
Dyspnoea	1	0	2

Data presented as n patients.

TABLE 2:
Blood pressure, heart rate, Glasgow Coma Scale (GCS) score and blood pH according to carboxyhaemoglobin (COHb) level among patients (n = 350) who presented to the emergency department with carbon monoxide intoxication

Outcome measure	COHb < 10% (n = 39)	COHb 10 – 20% (n = 106)	COHb > 20% (n = 205)	χ^2-value	Statistical significance[a]
Blood pressure, mmHg					
80 – 140	35	95	176	3.426	NS
< 80	2	1	7		
> 140	2	8	10		
Heart rate, beats/min					
60 – 100	34	91	140	17.423	P = 0.002
< 60	0	0	2		
> 100	2	13	51		
GCS score					
= 15	39	104	193	8.993	P = 0.011
< 15	2	11	4		
Blood pH					
Normal (7.35 – 7.45)	35	93	160	11.062	P = 0.026
Acidosis (< 7.35)	1	0	9		
Alkalosis (> 7.45)	3	13	34		

Data presented as n patients.
[a]χ^2-test; comparison across COHb groups.
NS, not statistically significant (P > 0.05).

16.6% of the patients with COHb levels > 20% had acidosis and alkalosis, respectively. Metabolic changes may reflect the toxic effects of CO better than any particular COHb level. Mild CO cases may be accompanied by respiratory alkalosis to compensate for the reduction in oxygen-carrying capacity and delivery. Longer exposures, with decreased levels of consciousness, result in metabolic acidosis from the lactate production that accompanies tissue hypoxia.[22] This indicates that the patients in the present study presented to the emergency department during the early phase of CO intoxication.

Hypoxia, hypotension, metabolic acidosis and a high lactate level are the primary factors responsible for the neurological effects of CO intoxication.[23 – 25] Grieb *et al.*[26] reported a strong inverse relationship between the GCS score and the severity of CO intoxication. There was no association between vital signs and COHb levels in the present study. Moreover, 68.3% of the patients with a COHb level > 20% had normal vital signs. Thus, vital signs may be normal in most patients with CO intoxication who have neurological symptoms and high COHb levels.

In conclusion, the majority of patients with CO intoxication in the present study presented during the early phase of illness, during which vital signs and arterial blood gas analyses may be misleading. Diagnosis of CO intoxication can, therefore, be made based only on suspicion and patients must be monitored carefully.

Conflicts of interest

The authors had no conflicts of interest to declare in relation to this article.

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