Research Article

Mild carbon monoxide poisoning impairs left ventricular diastolic function

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Abstract

Rationale: Carbon monoxide (CO) poisoning is associated with direct cardiovascular toxicity. In mild CO poisoning in which cardiovascular life support is not required, the effects of CO on left and right ventricular functions are unknown in patients without cardiac failure. **Objectives:** Echocardiography was used to determine whether or not mild CO poisoning impairs ventricular function. Twenty otherwise healthy patients with CO poisoning and 20 age- and gender-matched controls were studied. Echocardiographic examinations were performed at the time of admission and 1 week after poisoning. **Results:** The impairment observed in the left and right ventricular diastolic function at the time of admission was greater than the impairment 1 week after poisoning. Mild CO poisoning did not have a significant effect on systolic function. Carboxyhemoglobin levels were positively correlated with left ventricular diastolic dysfunction, whereas the levels were not correlated with right ventricular diastolic function. **Conclusions:** In CO intoxication, the development of left and right ventricular diastolic dysfunction precedes systolic abnormality. Patients with mild CO poisoning do not manifest cardiovascular symptoms; however, it should be borne in mind that most of these patients have myocardial involvement.

Keywords: Carbon monoxide, elasticity imaging techniques, left ventricular function, right ventricular function, tissue Doppler

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Introduction

Carbon monoxide (CO) poisoning results in several electrical, functional, and morphological alterations of the heart.[1] CO-associated cardiovascular damage is the result of tissue hypoxia and direct CO-mediated damage at the cellular level.[2] The frequent occurrence of cardiac disturbances during or after exposure to CO, including an increased frequency of anginal attacks, arrhythmias, and increased levels of cardiac enzymes has led to a search for morphological changes caused by CO.[3]

Impaired cardiac function after CO poisoning is usually related to acute myocardial ischemia,[4] and myocardial infarction induced by acute CO poisoning has been reported in a patient with normal coronary arteries.[5] A normal carboxyhemoglobin (COHb) level for a non-smoker is <2%.[6] Even slightly elevated levels of COHb (2-6%) have been shown to decrease exercise tolerance and worsen myocardial ischemia in patients with known coronary artery disease.[7,8] Reversible cardiac damage may occur with a COHb concentration >25%.[9]

CO poisoning has a complex pathophysiology that involves hypoxic stress stemming from the interference with oxygen transport to the cells and possibly also from the impairment of electron transport. It is also possible for carbon monoxide to affect leukocytes, platelets, and the endothelium, unleashing a chain of effects that result in oxidative injury. The amount of CO that must be inhaled to induce CO intoxication remains unknown.[10]

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In recent years, Doppler echocardiography in combination with tissue Doppler imaging has become a well-accepted, practical, safe, and non-invasive method for diagnosing left and right ventricular systolic and diastolic function in the clinical setting.[11,12]

In this study, considering the previous emphasis on the effects of CO poisoning on the cardiovascular system, we hypothesized that even mild CO poisoning would lead to impairment of left and right ventricular function.

Materials and Methods

Study population

This study included 20 consecutive patients with CO intoxication (11 females and 9 males; mean age, 27.45 ± 7.80 years) who were diagnosed in the Emergency Department of our University between December 2003 and March 2007. The diagnosis was based on medical history and a COHb level >8%. Twenty age- and gender-matched healthy volunteers from our hospital staff served as controls [Table 1].

On admission to the emergency department, a complete physical examination was performed, and we searched specifically for peripheral arterial pulses and carotid bruits. Blood pressure was measured in the sitting position. Alcohol consumption and the presence of major cardiovascular risk factors were recorded. Arterial and venous blood samples were obtained to determine the baseline levels of serum creatinine, blood urea nitrogen, sodium, potassium, chloride, calcium, alanine aminotransferase, aspartate aminotransferase, hemoglobin, creatinine phosphokinase-MB (CPK-MB) fraction, and blood glucose. Troponin I levels of 0.2-1.0 U/L, as measured with an Immulite 2000 Immunoassay System (Siemens Medical Solutions USA, Inc., Malvern, PA, USA), and creatine kinase-MB (Abbott Laboratories, Diagnostics Division, Abbott Park, IL, USA) levels of <24 U/L were accepted as normal. Carboxyhemoglobin levels were measured with an OSM3 Hemoximeter (Radiometer, Copenhagen, Denmark).

Table 1: Demographic and baseline echocardiographic measurements of patient and control groups

	Carbon monoxide (*n*=20)	Control (*n*=20)	*P*
Gender (Female/male)	11/9	11/9	0.355
Age (years)	27.45±7.79	25.80±5.88	0.455
SBP (mm Hg)	117.65±13.40	112.00±11.96	0.168
DBP (mm Hg)	71.95±9.33	68.50±6.70	0.188
Heart rate (beats/minute)	73.40±12.85	70.50±10.21	0.434
Rate×pressure product	8644.05±1821.53	7892.50±1357.31	0.147
LVMI	73.49±16.90	74.73±15.42	0.810
LVEF	65.29±7.45	68.80±4.51	0.080
Mitral E/A ratio	1.57±0.30	1.65±0.33	0.371
Mitral E deceleration time (ms)	183.65±26.46	175.70±25.48	0.339
Sm (cm/s)	14.85±2.73	16.15±4.22	0.255
Em/Am ratio	1.71±0.55	1.61±0.37	0.516
Tissue doppler IVRT (ms)	83.05±16.34	87.75±19.04	0.407
RV Sm (cm/s)	16.00±3.04	17.20±3.18	0.231
RV tissue doppler IVRT (ms)	79.95±29.22	80.85±19.51	0.909
RV Em/Am ratio	1.26±0.42	1.32±0.35	0.680
LV MPI	0.59±0.12	0.64±0.18	0.385
RV MPI	0.61±0.18	0.65±0.17	0.501
Tricuspid E/A ratio	1.55±0.31	1.62±0.39	0.553
Tricuspid E deceleration time (ms)	183.20±29.13	171.38±21.31	0.152
Hemoglobin (g/dL)	13.45±1.76	13.185±1.83	0.645

Sm: Systolic peak velocity; Em: Early peak velocity; Am: Atrial peak velocity; IVRT: Isovolumic relaxation time; IVCT: Isovolumic contraction time; ET: Ejection time; MPI: Myocardial performance index; SBP: Systolic blood pressure; DBP: Diastolic blood pressure; LV: Left ventricle; RV: Right ventricle; LVMI: Left ventricular mass index; LVEF: Left ventricular ejection fraction

After blood sampling, 100% oxygen was administered (2-5 L/min) to all patients via a face mask. None of the patients required hyperbaric oxygen therapy or artificial ventilation. Chest radiographs were obtained in all patients. A 12-lead electrocardiogram was taken at the time of admission and 1 week after poisoning. Transthoracic echocardiographic examinations, which included tissue Doppler imaging, were performed on admission and 1 week after discharge from the hospital. The study was conducted according to the recommendations set forth by the Declaration of Helsinki on Biomedical Research Involving Human Subjects. The Ethics Committee on Biomedical Research at our university Medical School approved the study protocol. Written informed consent was obtained from each subject.

Echocardiographic examination

Each subject was examined using an Acuson Sequoia C256 Echocardiography System (Acuson, Mountain View, CA, USA) equipped with 3V2c and 5V2c broadband transducers with second harmonic capability. Within 30 minutes of admission to the Emergency Department, 2-dimensional M-mode Doppler echocardiography, and standard and pulsed-tissue Doppler echocardiography were performed on each subject, with the patient in the lateral decubitus position. Echocardiographic images were recorded on videotape. M-mode images were used to measure diastolic and systolic interventricular septal thickness, posterior wall thickness, and left ventricular end-diastolic and left ventricular end-systolic diameters on the parasternal long-axis view.

The pulsed Doppler sample volume was placed at the mitral and tricuspid leaflet tips. Early diastolic peak

flow velocity (E), late diastolic peak flow velocity (A), E/A ratio, and E-wave deceleration time (DT) were measured by using trans-mitral and trans-tricuspid Doppler imaging.

Pulsed-wave Doppler tissue imaging was performed. Filters were set to exclude high-frequency signals. Gains were minimized to allow for a clear tissue signal with minimal background noise. Recordings were obtained during normal respiration. A 5-mm sample volume was placed at the apical 4-chamber view on the lateral corner of the mitral annulus.[13] The resulting velocities were recorded for 5-10 cardiac cycles at a sweep speed of 100 mm/s and stored on videotape for off-line analysis. As a measure of regional systolic function, peak velocity (cm/s) and time velocity integral of myocardial systolic (Sm) wave were determined. Diastolic measurements including myocardial early (Em) and atrial (Am) peak velocities (cm/s), Em/Am ratio, and Sm-Em duration (isovolumic relaxation time [IVRTL]) measured as the time interval between the end of Sm and the onset of Em, were performed. Left ventricular ejection time (LVET) was measured from the onset to the end of left ventricular (LV) outflow. Right ventricular ejection time (RVET) was measured from the onset to the end of right ventricular (RV) outflow. Isovolumetric contraction time of the left ventricle (IVCTL) was the interval from the cessation of mitral inflow to the onset of LV outflow.

Isovolumetric relaxation time of the right ventricle (IVRTR) was defined as the time interval from the cessation of RV outflow to the onset of tricuspid inflow. The isovolumetric contraction time of the right ventricle (IVCTR) was measured from the cessation of tricuspid inflow to the onset of RV outflow. The myocardial performance index of the right ventricle (MPIR) was calculated by the formula (IVCTR + IVRTR)/RVET. The myocardial performance index of the left ventricle (MPIL) was calculated by the formula (IVCTL + IVRTL)/LVET.[14] All diastolic parameters were measured in three consecutive cardiac cycles and averaged.

Left ventricular Sm and EF values were used to demonstrate left ventricular contractile (systolic) function. Right ventricular Sm was used to demonstrate right ventricular contractile function.

Trans-mitral and trans-tricuspid E wave velocity, A wave velocity, E/A ratios, Em, Am, Em/Am ratios, IVRT, IVCT, ET, and MPI values were used to demonstrate right and left ventricular relaxation (diastolic) function.

The echocardiography was performed by an investigator who was blinded for clinical data and the analysis of the echocardiographic recordings were performed by two cardiologists, blinded to the subjects' data. Inter-observer variability was 5.7%.

Statistical analyses

Statistical analyses were performed with Statistical Product and Services Solutions software (SPSS, version 10.0; SPSS Inc, Chicago, IL, USA). Numeric values are expressed as means ± SD. The difference between echocardiographic parameters obtained at the time of admission and discharge was assessed with a paired samples *t*-test. When parametric tests were not appropriate, the Wilcoxon signed rank test was used. The Spearman rank correlation test was used to determine the correlation between the echocardiographic parameters and COHb level. A value of $P < 0.05$ was considered statistically significant.

Results

Clinical characteristics of the study population

All the patients were delivered 100% oxygen and none of them required additional therapy. Moreover, none of the patients developed neurologic or other complications requiring artificial ventilation. The mean left ventricular mass index (LVMI) and hemoglobin values were 73.49 ± 16.90 g/m² and 13.45 ± 1.76 g/dL, respectively. The mean carboxyhemoglobin levels on admission and 1 week after poisoning were 15.81% ± 4.87% and 1.57% ± 0.24%, respectively. The mean HbO_2 saturation on admission and 1 week after poisoning were 94.60% ± 5.09% and 97.67% ± 1.55%, respectively. The mean Creatinine phospho-Kinase-myocardial band (CPK-MB) level on admission and 1 week after poisoning was 16.52 ± 6.30 U/L and 15.79 ± 6.68 U/L, respectively. The value for troponin-I on admission and 1 week after poisoning was 0.2 ± 0 and 0.2 ± 0.004, respectively.

Systolic blood pressure, diastolic blood pressure, heart rate, and rate-pressure product values were only slightly affected after mild CO intoxication and the differences regarding these measures between the time of admission and one week after poisoning were not significant [Table 1].

Findings of left ventricular relaxation

Mitral E/A ratios were significantly lower ($P = 0.047$), and mitral E wave DT ($P = 0.011$), tissue Doppler IVRT ($P = 0.014$), and MPIL ($P = 0.023$) were significantly higher after CO poisoning than 1 week after poisoning. Em/Am values were slightly lower

after CO intoxication (1.52 ± 0.46) than 1 week after poisoning (1.71 ± 0.56), but the difference was not statistically significant [$P = 0.233$; Table 2].

Right ventricular relaxation parameters

Tricuspid E wave DT was significantly higher after CO poisoning (226.10 ± 61.53 ms) than 1 week after poisoning (183.20 ± 29.14 ms; $P = 0.008$). Tricuspid E/A ratios were not significantly altered after CO poisoning (1.41 ± 0.37) compared with 1 week later (1.56 ± 0.32; $P = 0.172$). Right ventricular (RV) E/A ratios and MPIR did not change significantly after mild CO intoxication ($P = 0.404$ and $P = 0.235$, respectively). RV IVRT (101.85 ± 27.87 ms) was significantly higher on admission than 1 week after poisoning [79.95 ± 29.23 ms; $P = 0.020$; Table 2].

Left and right ventricular contractile function

No significant difference was detected in left ventricular ejection fraction obtained on admission (following CO poisoning) and 1 week later [Table 2]. Left (LV Sm) and right ventricular systolic function (RV Sm) were not significantly altered with CO poisoning $P = 0.766$ and $P = 0.332$, respectively [Tables 2 and 3].

Relationship between carboxyhemoglobin levels and left and right ventricular relaxation

Carboxyhemoglobin levels were significantly correlated with mitral A wave ($r = 0.335$, $P = 0.025$), mitral E wave deceleration time ($r = 0.512$, $P = 0.001$), MPIL ($r = 0.316$, $P = 0.047$), and tricuspid deceleration time ($r = 0.344$, $P = 0.030$). Carboxyhemoglobin levels were negatively correlated with mitral E/A ratios ($r = -0.320$, $P = 0.044$).

Table 2: Left ventricular systolic and diastolic function measurements

	At the time of admission	1 week after CO poisoning	P
Mitral E_{max} (cm/s)	84.55±13.02	81.35±12.14	0.426
Mitral A_{max} (cm/s)	64.75±15.88	53.05±9.25	0.007
Mitral E deceleration time (ms)	205.00±24.24	183.65±26.47	0.011
Mitral E/A ratio	1.36±0.33	1.57±0.30	0.047
Sm (cm/s)	14.60±2.54	14.85±2.74	0.766
Em (cm/s)	22.40±5.22	22.45±5.49	0.977
Am (cm/s)	15.50±4.56	13.90±3.72	0.232
Em/Am ratio	1.52±0.46	1.71±0.56	0.233
Tissue doppler IVRT (ms)	100.95±26.47	83.05±16.34	0.014
Tissue doppler IVCT (ms)	81.85±25.06	77.50±15.58	0.514
Tissue doppler ET (ms)	262.90±36.84	271.95±30.77	0.404
Let ventricular MPI	0.70±0.15	0.60±0.12	0.023
LVEF	65.29±7.45	65.02±6.02	0.785

Sm: Systolic peak velocity; Em: Early peak velocity; Am: Atrial peak velocity; IVRT: Isovolumic relaxation time; IVCT: Isovolumic contraction time; ET: Ejection time; MPI: Myocardial performance index; LVEF: Left ventricular ejection fraction

Table 3: Right ventricular systolic and diastolic function measurements

	At the time of admission	1 week after CO poisoning	P
SBP (mm Hg)	118.60±16.42	117.65±13.41	0.842
DBP (mm Hg)	73.05±11.53	71.95±9.34	0.742
Heart rate (beats/minute)	77.55±11.01	73.40±12.85	0.280
Rate×pressure product	9140.45±1486.60	8644.05±1821.54	0.351
Tricuspid E_{max} (cm/s)	61.65±17.13	59.10±12.70	0.596
Tricuspid A_{max} (cm/s)	46±16.29	39.10±10.45	0.119
Tricuspid E deceleration time (ms)	226.10±61.53	183.20±29.14	0.008
Tricuspid E/A ratio	1.41±0.37	1.56±0.32	0.172
RV Sm (cm/s)	16.95±3.07	16.00±3.04	0.332
RV Em (cm/s)	20.15±4.91	20.35±3.60	0.884
RV Am (cm/s)	19.90±5.04	17.00±3.54	0.042
RV Em/Am ratio	1.13±0.60	1.27±0.43	0.404
RV tissue doppler IVRT (ms)	101.85±27.87	79.95±29.23	0.020
RV tissue doppler IVCT (ms)	74.35±17.19	80.65±27.47	0.390
RV tissue doppler ET (ms)	263.75±36.48	264.00±27.92	0.981
RV MPI	0.68±0.14	0.62±0.18	0.235

SBP: Systolic blood pressure; DBP: Diastolic blood pressure; RPP: Rate pressure product; RV: Right ventricle; Sm: Systolic peak velocity; Em: Early peak velocity; Am: Atrial peak velocity; IVRT: Isovolumic relaxation time; IVCT: Isovolumic contraction time; ET: Ejection time; MPI: Myocardial performance index

Discussion

Effects of carbon monoxide poisoning result from the formation of COHb. A normal COHb level for a non-smoker is <2%[6] and even slightly elevated levels of COHb (2-6%) have been shown to decrease exercise tolerance and worsen myocardial ischemia in patients with known coronary artery disease.[7,8] Reversible cardiac failure may occur with a COHb concentration >25% after CO poisoning.[9] In the current study, the mean COHb concentrations of the patients was 15.81% ±4.87%, which was consistent with mild CO poisoning and no clinical or laboratory findings indicating left ventricular systolic impairment was observed.

The present study has revealed that even mild CO poisoning has acute unfavorable effects on left and right ventricular function. To our knowledge, this is the first study to investigate the hazardous effects of mild CO poisoning. None of our patients died or experienced severe complications, such as the need for artificial ventilation. These favorable outcomes possibly resulted from the mild exposure to CO; as demonstrated by the lack of cardiac enzyme or troponin elevations seen in moderate and severe CO intoxication. During the early period, there were no pronounced symptoms in the patients included in this study. However, to determine the cardiac problems that may be induced in the long run by CO poisoning, further long-term, randomized, and prospective studies with a greater number of patients are necessary.

As a measure of the combined systolic and diastolic function, conventional LV myocardial performance

index (LVMPI) frequently comes into use.[15] It has been reported to be a reliable parameter for the evaluation of the performances of the right[16,17] and left ventricles.[18]

The MPI, as an index of global myocardial performance, can be obtained in an easy and reliable manner;[19] is independent of heart rate and blood pressure.[20,21] High levels of it have been reported to be associated with adverse events like myocardial infarction.[22] In our study, MPIR did not change significantly after mild CO intoxication but were significantly higher after CO poisoning than 1 week after poisoning.

The major cause of death from CO poisoning is myocardial injury and dysfunction.[23] Left ventricular systolic dysfunction was determined in patients with moderate CO poisoning in several studies.[24,25] Although significant systolic dysfunction was observed with severe CO poisoning, we observed no significant difference regarding the contractile function of the left ventricle immediately and 1 week after CO poisoning.

CO binds to cytochrome-c oxidase of the electron transport chain *in vitro*, resulting in asphyxiation at the cellular level.[26] Oxygen radical formation and subsequent lipid peroxidation have also been implicated as mechanisms for cell death.[26] COHb levels as low as 5% can cause a considerable decrease in the blood's capacity to carry oxygen.[27] However, clinical studies in healthy humans suggest that modest levels of CO have little effect on exercise performance, cardiac output, heart rate, blood pressure, or minute ventilation.[27] Increased sympathetic activation and consequent hemodynamic effects of CO may have implications for understanding the relationship between atmospheric CO pollution and fatality rates in patients with acute myocardial infarction.[28,29] Blood pressure, heart rate, and rate-pressure products were similar in the patient and control groups [Table 2].

As tissue Doppler imaging is more sensitive than conventional Doppler methods in the assessment of LV function and is independent of filling pressure,[30,31] we have combined the two techniques in our study, and found significant relaxation abnormalities related with CO poisoning.

Echocardiographic evaluation of right ventricular function is difficult because of the geometry of the ventricle and its position beneath the sternum. It has been reported that the MPI is a useful index for evaluating RV function.[16,17] As our study group consisted of patients with mild CO intoxication, our results confirm a relaxation abnormality, despite preserved ventricular contractile function at relatively low levels of CO intoxication.

Study limitations

The possible clinical results of CO poisoning may be underestimated by the relatively small sample size of the study. Our study includes data from an earlier period. In order to determine the cardiac problems that may be induced in the long run by CO poisoning, further long-term, randomized, and prospective studies with a greater number of patients are necessary. It is believed that LV filling pressure, left ventricular disorder is reflected by the ratio of peak diastolic early velocity (E) of LV inflow to peak diastolic longitudinal velocity (e') of the mitral annulus (E/e'). Previous studies have shown that, E/é is significant predictor of AMI.[32] E/é value would improve scientific value of our paper, however, we have not measured E/é values due to the fact that it was not present in our initial working plan.

Conclusions

We conclude that CO intoxication leads to right and left ventricular relaxation abnormalities before the development of contractile dysfunction. Further studies with large number of subjects, investigating the possible chronic effects of mild CO poisoning on left and right ventricular function will aid in obtaining more clinically relevant results.

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