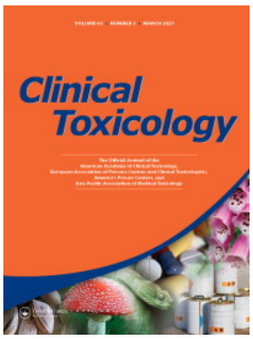

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Echocardiographic and clinical patterns in patients with acute carbon monoxide poisoning without cardiovascular and other chronic diseases

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ABSTRACT

Introduction: Severe carbon monoxide may impact the circulatory system, potentially leading to myocardial injury. This study aimed to assess left ventricular function via echocardiography in patients with acute carbon monoxide poisoning who were otherwise healthy.

Methods: We conducted an observational, single-centre study involving consecutive patients hospitalized with carbon monoxide poisoning.

Results: In a study of 112 consecutive patients with acute carbon monoxide poisoning, we identified a subset of 46 patients with moderate to severe poisoning. Among them, myocardial injury (defined by a peak high-sensitivity troponin T concentration >14.0 ng/L) was observed in 17 of 46 (36.9%) patients, forming the myocardial injury group. The remaining 29 patients formed the non-myocardial injury group. The echocardiographic assessment revealed no significant difference (P = 0.06) between the mean (±SD) left ventricular ejection fraction in the myocardial injury group (59.8 ± 5.4%), compared to the mean (±SD) in the non-myocardial injury group (62.9 ± 5.5%). However, the mean (±SD) left ventricular global longitudinal strain was significantly higher (P = 0.008) in the myocardial injury group (-20.1 ± 1.8%) compared to the non-myocardial injury group (-22.1 ± 2.4%). Patients in the myocardial injury group also exhibited significantly higher (P<0.001) mean heart rates (108.9 beats/min) compared to the non-myocardial injury group (87.6 beats/min). In addition, the mean plasma lactate concentration was significantly higher (P<0.001) in the myocardial injury group (1.95 mmol/L) compared to the non-myocardial injury group (1.2 mmol/L). There were no fatalities in either group.

Discussion: Healthy patients with carbon monoxide poisoning who have myocardial injury may show minor changes in echocardiography in contrast to patients with co-morbidities.

Conclusions: In patients with moderate to severe carbon monoxide poisoning, without concurrent chronic diseases, left ventricular global longitudinal strain was significantly lower in those with myocardial injury. However, these findings are based on a small cohort, necessitating further research.

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Carbon monoxide-induced cardiomyopathy; carbon monoxide poisoning; cardiac injury; echocardiography; global longitudinal strain

Introduction

Carbon monoxide poisoning continues to be a significant cause of hospitalization and mortality, with a global mortality rate from unintentional carbon monoxide poisoning of 0.366 per 100,000 in 2021. The highest age-standardized mortality rate of 2.12 deaths per 100,000 was in Eastern Europe [1–3]. The mechanism of impact of carbon monoxide on the human body is multifaceted and still under investigation. Carbon monoxide binds predominantly to intravascular space haemoglobin and cytochrome oxidase as well as to myoglobin, cytochrome P-450 oxidase, nitric oxide synthase, hydroperoxidase, guanylate cyclase, and nicotinamide adenine dinucleotide phosphate hydrogen reductase, impairing their function and producing critical organ damage, particularly in the brain and heart [4–6]. In the circulatory system, carbon monoxide can induce tachycardia, dysrhythmias, conduction disorders, hypo- or hypertension; myocardial injury; myocardial infarction both in the acute phase and months post-intoxication; tako-tsubo cardiomyopathy, tako-tsubo-like cardiomyopathy, or various forms of carbon monoxide-induced cardiomyopathy; pulmonary oedema; and sudden cardiac arrest [4,7–12]. Myocardial injury, defined as an increase in cardiac biomarkers, occurs in about 37% of patients with moderate to severe carbon monoxide poisoning and increases the risk of in-hospital and long-term mortality [4]. The definition of carbon monoxide-induced cardiomyopathy varies. It is often diagnosed when elevated cardiac biomarkers are present, along with ischaemic changes on the electrocardiogram or echocardiographic abnormalities pertaining to either systolic

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or diastolic function of the left ventricle or, occasionally the right ventricle. Carbon monoxide-induced cardiomyopathy, either global or regional (tako-tsubo-like syndrome), may be seen in approximately 74% of patients consecutively admitted with increased troponin I concentrations [9]. However, the status of the coronary circulation in these patients is not fully understood.

This study aimed to use echocardiography with an additional assessment of global longitudinal strain to detect left ventricular damage in patients with acute carbon monoxide poisoning, excluding those with chronic conditions, including arterial hypertension, that could influence left ventricular function.

Methods

Study group and protocol

This single-centre observational study included consecutive patients hospitalized for acute carbon monoxide poisoning at the Toxicology Clinic of the Medical University in Lublin, Poland, from June 2019 to July 2023. The exclusion criteria were: (1) patients under 18 years old; (2) patients with pre-existing chronic cardiac diseases (including hypertension, coronary syndrome, prior myocardial infarction, atrial fibrillation, chronic heart failure, significant valvular disease, congenital heart disease); (3) patients with clinically significant previously diagnosed diseases (such as diabetes mellitus, anaemia, sepsis); (4) patients with concurrent drug and alcohol poisoning; (5) cases of chronic carbon monoxide poisoning; (6) instances of poisoning with other gases (e.g., smoke inhalation from fires); (7) patients discharged before echocardiography could be performed; and (8) challenges in obtaining clear echocardiographic images of left ventricular function. Acute carbon monoxide poisoning was diagnosed when the carboxyhaemoglobin levels were >10% or there was confirmed exposure to high carbon monoxide concentrations, along with clinical features indicative of poisoning.

The protocol entailed a physical examination, electrocardiography, laboratory blood testing, and echocardiography. In patients with cardiac injuries, evaluations included analysis of the electrocardiogram for ST segment depression and elevation, T-wave inversion, QT interval prolongation, and bundle branch block. Key laboratory measurements included carboxyhaemoglobin, pH, and concentrations of high-sensitivity troponin T and N-terminal pro B-type natriuretic peptide. The plasma high-sensitivity troponin T concentration was measured on admission and repeated daily if the initial concentration was elevated, focusing on peak concentrations for analysis. Acute myocardial injury was diagnosed as a peak high-sensitivity troponin T concentration >14.0 ng/L [13]. High-sensitivity troponin T concentrations were determined using patented electrochemiluminescence technology (Cobas E 601, Roche Diagnostics, France).

Carbon monoxide poisoning was classified into three clinical grades:

- Grade 1 (minor): 10–25% carboxyhaemoglobin level and/or absence of clinical features.
- Grade 2 (moderate): 10–25% carboxyhaemoglobin level and/or symptoms such as weakness, headache, dizziness, or nausea.
- Grade 3 (severe): >25% carboxyhaemoglobin level and/or severe symptoms such as syncope, seizures, coma, pulmonary oedema, or myocardial infarction.

All participants provided written informed consent. In the study protocol, patients who were unable to sign the consent form due to acute poisoning could be included in the study, provided that written consent was obtained after they regained consciousness or from a legal guardian. The local Bioethics Committee approved the study (agreement number 71/2020/KB/VIII).

Echocardiography

Transthoracic echocardiography, performed using the GE Vivid E 95 system (GE Healthcare System), was conducted as soon as possible, ideally within 24 h of admission. Experienced cardiologists interpreted the results. Left ventricular systolic function was evaluated based on the ejection fraction and global longitudinal strain. The left ventricular ejection fraction was calculated using the Simpson's biplane method. Strain parameters and longitudinal strain analysis utilized a 17-segment left ventricle model. The area of interest was initially defined automatically and then verified and adjusted by the researcher if necessary.

The analysis of left ventricular diastolic function adhered to current echocardiographic recommendations [14]. Peak E-wave and A-wave velocities were measured in the apical four- chamber view at the tips of the mitral leaflets. Pulsed-wave tissue Doppler imaging e' velocities were assessed in the apical four-chamber view at the lateral and septal basal regions of the left ventricle. The average e' velocity was also determined. The left atrium area and volume measurements were taken in the four-chamber view, ensuring maximum left atrium length and transverse diameters. The disk method was applied to assess left atrial volume. The peak systolic jet velocity of tricuspid regurgitation was measured in the apical four-chamber view using continuous wave Doppler. All examinations were recorded digitally.

Statistical analysis

Continuous variables were tested for normal distribution using the Shapiro-Wilk test. Those with a normal distribution were presented as means and standard deviations, while those without normal distribution were expressed as medians and interquartile ranges. Quantitative results were reported as percentages. The Student's t-test was used to test for statistical significance of parametric data, while the Mann-Whitney U test was applied to non-normally distributed data. Categorical variables were analyzed using the chi-square or Fisher's exact tests. A P value of less than 0.05 was deemed statistically significant. Statistical analyses were performed using Statistica version 13.3 (TIBCO Software Inc.).

Results

In a cohort of 112 consecutive patients with acute carbon monoxide poisoning, 46 were selected for analysis based on their transthoracic echocardiography results. Twenty-nine

(63%) were women, and the median age of the 46 patients was 28 years (IQR: 22–42 years). Sixty-six patients were excluded from the study because of co-morbidities or inability to obtain echocardiography results (Figure 1).

Circumstances of poisoning

Faulty gas heating installations were the most common source of carbon monoxide, and all incidents were unintentional. None of the patients were treated for acute infections, and those examined during the COVID-19 pandemic tested negative using a polymerase chain reaction test.

Treatment

All participants received high-flow oxygen therapy with 100% oxygen via a non-rebreather mask until the carboxyhemoglobin level decreased below 5–7%. Then they continued oxygen therapy through a face mask with a 50% oxygen mixture for about 6 h, and then intermittently with a 30% oxygen mixture for 24 h. There were no deaths in either group during hospitalization, and all patients were discharged home.

Basic characteristics

Myocardial injury occurred in 17 (36.9%) of the 46 patients, constituting the myocardial injury group. The remaining 29 patients fell into the non-myocardial injury group. Patients in the myocardial injury group also exhibited significantly higher ($P<0.001$) mean heart rates (108.9 beats/min) compared to the non-myocardial injury group (87.6 beats/min). In addition, the mean plasma lactate concentration was significantly higher ($P<0.001$) in the myocardial injury group (1.95 mmol/L) compared to the non-myocardial injury group (1.2 mmol/L). Severe carbon monoxide poisoning was more frequent ($P=0.045$) in the myocardial injury group (13 of 17 [76.5%] patients) compared to the non-myocardial injury group (12 of 29 [41.4%] patients), while moderate poisoning was more common ($P=0.045$) in the non-myocardial injury group (17 of 29 [58.6%] patients) compared to the myocardial injury group (4 of 17 [23.5%] patients). Characteristics and laboratory findings of the carbon monoxide poisoned study group are presented in Table 1.

In the myocardial injury group, loss of consciousness (10 of 17 patients [58.8%]) and dizziness (10 of 17 [58.8%] patients) were the most prevalent symptoms of carbon monoxide poisoning, whereas, in the non-myocardial injury group, headache (19 of 29 [65.5%] patients) and dizziness (18 of 29

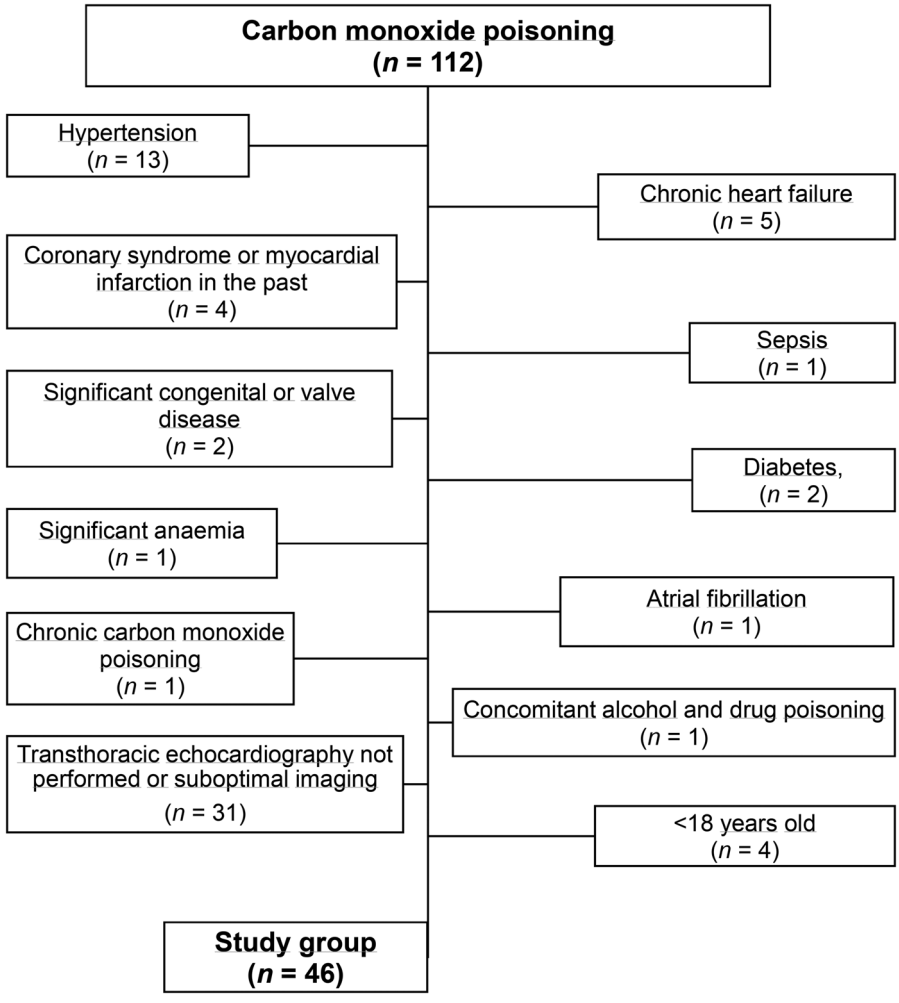

Figure 1. Selection of study group.

Table 1. Characteristics and laboratory findings of carbon monoxide poisoned patients.

Characteristic	Total ($n=46$)	Myocardial injury group ($n=17$)	Non-myocardial injury group ($n=29$)	P value
Age (years), median (IQR)	28 (22–42)	29 (21–40)	27 (22–45)	0.80
Female, n (%)	29 (63)	8 (47)	21 (72.4)	0.08
Body mass index (kg/m^2), median (IQR)	24 (21.1–27.3)	23.8 (20.7–27.7)	24 (21.1–27.1)	0.51
Systolic blood pressure (mmHg), median (IQR)	120 (110–130)	120 (105–145)	120 (110–129)	0.63
Diastolic blood pressure (mmHg), mean ± SD	78.2 ± 15.2	78.0 ± 19.2	78.4 ± 12.5	0.94
Heart rate (beats/min), mean ± SD	95.5 ± 21.1	108.9 ± 20.4	87.6 ± 17.4	<0.001
Tachycardia, n (%)	21 (45.6)	14 (82.3)	7 (24.1)	<0.001
Carboxyhaemoglobin level (%), mean ± SD	20.1 ± 9.0	22.2 ± 11.3	18.8 ± 7.3	0.22
pH, mean ± SD	7.36 ± 0.03	7.34 ± 0.03	7.37 ± 0.03	0.054
Lactate concentration (mmol/L), median (IQR)	1.4 (1.05–1.8)	1.95 (1.5–4.5)	1.2 (0.9–1.5)	<0.001
Creatinine concentration (mg/dL), mean ± SD	0.8 ± 0.16	0.8 ± 0.14	0.79 ± 0.18	0.66
Creatinine concentration (µmol/L), mean ± SD	70.7 ± 14.1	70.7 ± 12.4	69.8 ± 15.9	0.66
Troponin T max concentration (ng/L), median (IQR)	9.54 (7.2–16.4)	28.7 (14.7–70.3)	7.4 (5.2–8.6)	<0.001
N-terminal pro B-type natriuretic peptide concentration (pg/mL), median (IQR)	114 (56.8–167.5)	114 (11.7–183)	106.4 (57.1–139)	0.88
Clinical grade of carbon monoxide poisoning: Grade 2 (moderate), n (%)	21 (45.7)	4 (23.5)	17 (58.6)	0.045
Clinical grade of carbon monoxide poisoning: Grade 3 (severe), n (%)	25 (54.3)	13 (76.5)	12 (41.4)	0.045

Table 2. Symptoms in patients after carbon monoxide poisoning.

Symptom	Total ($n=46$)	Myocardial injury group ($n=17$)	Non-myocardial injury group ($n=29$)	P value
Dizziness, n (%)	28 (60.8)	10 (58.8)	18 (62.1)	1.0*
Headache, n (%)	25 (54.3)	6 (35.2)	19 (65.5)	0.07*
Loss of consciousness, n (%)	20 (43.4)	10 (58.8)	10 (34.5)	0.13*
Nausea, n (%)	14 (30.4)	3 (17.6)	11 (37.9)	0.19*
Weakness, n (%)	7 (15.2)	1 (5.9)	7 (24.1)	0.22*
Vomiting, n (%)	6 (13.0)	3 (17.6)	3 (10.3)	0.65*
Chest pain, n (%)	4 (8.6)	0	4 (13.8)	0.28*
Dyspnoea, n (%)	3 (6.5)	1 (5.9)	2 (6.9)	1.0*

*Fisher exact test was used.

[62.1%] patients) were more common. However, there was no statistical significance between groups. Detailed results are presented in Table 2. Notably, chest pain did not occur in any patient in the myocardial injury group.

Electrocardiograms

Electrocardiograms showed all patients to be in sinus rhythm. In the myocardial injury group, ST segment depression occurred in 11 of 17 (64.7%) patients; this resolved within three days of admission in seven patients, became less severe in one patient and persisted until the day of discharge in one patient. The remaining two patients left the hospital before a follow-up electrocardiogram was performed. In patients with other changes in the electrocardiogram on admission (atypical ST-segment elevation, intraventricular conduction disorders), the recording did not change during hospitalization. Detailed information about electrocardiogram changes in the myocardial injury group patients is presented in Table 3. In the non-myocardial injury group, 10 of 29 (34.4%) patients had normal electrocardiograms. ST segment depression in the electrocardiogram occurred in 7 of 29 (24.1%) patients; in six patients the changes resolved within three days of admission, but in one patient they persisted until the day of discharge. Non-specific ST elevation occurred in seven of 29 patients, and persisted to discharge in six patients. Other changes in this group included transient intraventricular conduction disorder (rSr′ in lead V2) (one patient), persistent intraventricular conduction disorder (three patients), and transient high T waves in lead V4 (one patient). The QT interval duration was within normal limits in all patients included in the study.

Echocardiography

The myocardial injury group showed a significantly higher ($P=0.008$) mean (±SD) global longitudinal strain (-20.1 ± 1.8%) compared to the non-myocardial injury group (-22.1 ± 2.4%). However, there was no significant difference ($P=0.06$) in the mean (±SD) left ventricular ejection fraction, assessed by the Simpson biplane method, between the myocardial injury group (59.8±5.4%) and the non-myocardial injury group (62.9 ± 5.5%). Echocardiographic findings are presented in Table 4.

Discussion

Our study found that in patients without coexisting chronic diseases, acute carbon monoxide poisoning led to myocardial injury in 17 of 46 (36.9%) patients. This finding is consistent with previous observations in patients with acute carbon monoxide poisoning [4]. Our results suggest that patients with moderate to severe carbon monoxide poisoning who have myocardial injury may show relatively minor changes in echocardiography. Furthermore, normal left ventricular systolic function does not exclude the possibility of left ventricular myocardial damage.

The patients in this study were younger than in previous studies (median age 28 years, [IQR: 22–42 years]) and did not have chronic diseases. Echocardiographic examination, including an expanded protocol for assessing left ventricular global longitudinal strain, was conducted in all patients, irrespective of the presence of myocardial injury. In our study, carboxyhaemoglobin levels were not a reliable marker for left ventricular damage, echoing findings from earlier research [11]. Carboxyhaemoglobin as a biomarker has little prognostic value because the elimination of carbon monoxide begins from the moment of interruption of exposure, and is impacted by the administration of supplemental oxygen. In carbon monoxide-poisoned patients, the half-life of carboxyhaemoglobin in a patient breathing room air is approximately 250

Table 3. Clinical characteristics and echocardiography results in the 17 patients in the myocardial injury group.

Patient	Age (years)	Symptoms	High-sensitivity troponin concentration (ng/L)	Heart rate (beats/min)	Electrocardiographic changes	Left ventricular ejection fraction biplane (%)	Left ventricular global longitudinal strain (%)
1	21	Syncope, vomiting	123.5	120	↓ST II, III, aVF, V3–V6	59	−20.2
2	25	Syncope, headache	14.6	111	↓ ST II, III, aVF, V4–V6	57	−19.3
3	23	Palpitations, headache, vomiting	62.5	124	↓ ST II, III, aVF, V4–V6	51	−16.6
4	27	Syncope, headache, dizziness, nausea	14.8	100	↑ ST V4 (1 mm)	66	−22.9
5	18	Headache, dizziness	25.0	120	↓ ST III, aVF	58	−19.3
6	40	Syncope, vomiting, dizziness	75.5	140	Right bundle branch block	52	−18.9
7	42	Dyspnoea, dizziness	16.4	110	↓ ST I, II, III, aVF, V4–V6	58	−17.9
8	35	Weakness, dizziness	15.3	105	↓ ST II, III	59	−21.1
9	31	Syncope	88.4	140	↓ ST V5, V6	53	−19.3
10	34	Syncope	96.5	130	↑ ST aVF, V5, V6	54	−22.6
11	42	Headache, dizziness	38.9	90	↓ ST V4, V5	65	−20.2
12	18	Syncope	22.2	120	↓ ST 1 mm V4–V6	66	−20.7
13	21	Syncope, headache, dizziness	15.4	70	Normal	62	−23.5
14	29	Syncope, nausea	70.3	100	↓ ST 1 mm I, II, III, V4–V6	66	−21.6
15	19	Syncope, defecation	32.4	102	↓ ST 0,5 mm V4, V5	58	−19.2
16	52	Palpitaions, dizziness	33.1	100	Incomplete right bundle branch block	68	−21.4
17	69	Dizziness	14.4	70	↑ ST 2 mm V4, V5	65	−18.0

Table 4. Echocardiographic parameters in patients with carbon monoxide poisoning.

Characteristic	Total ($n=46$)	Myocardial injury group ($n=17$)	Non-myocardial injury group ($n=29$)	P value
Left ventricle in apical four chamber view (mm), mean ± SD	46.2 ± 3.7	43.7 ± 11.4	44.7 ± 9.3	0.96
Right ventricle in apical four chamber view (mm), mean ± SD	36.1 ± 5.0	34.3 ± 10.2	34.8 ± 8.0	0.82
Left ventricle in parasternal long axis view (mm), mean ± SD	47.3 ± 4.3	45.9 ± 12.2	45 ± 9.4	0.12
Right ventricle in parasternal long axis view (mm), mean ± SD	28.4 ± 4.0	27.9 ± 8.1	26.8 ± 6.2	0.13
Left atrium in four chamber view (mm), median (IQR)	32 (27–45)	30.5 (29–35)	32 (30–35)	0.60
Interventricular septum (mm), median (IQR)	8 (6–11.5)	8.25 (7–9)	8 (7.4–9.5)	0.61
Posterior wall (mm), median (IQR)	8 (6–12)	8 (7–9)	8 (7–8.5)	0.34
Aortic root (mm), median (IQR)	30.5 (21–43)	29.5 (27–31)	30.5 (27–33)	0.83
Left atrium area in four chamber view (cm^2), mean ± SD	19 ± 3.4	18.6 ± 5.1	17.9 ± 5.1	0.24
Right atrium area in four chamber view (cm^2), median (IQR)	15.2 (8.2–24)	15.9 (14–18)	15 (13.5–17)	0.08
Left atrium volume, in four chamber view (mL), mean ± SD	59.6 ± 15.4	60.1 ± 18.9	55.3 ± 19.6	0.17
Left atrium volume index (mL/m^2), mean ± SD	32.5 ± 8.5	32.8 ± 10.4	30.1 ± 10.7	0.18
Tricuspid regurgitant peak velocity (m/sec), mean ± SD	2.16 ± 0.4	2.17 ± 0.6	1.94 ± 0.6	0.06
Tricuspid annulus plane systolic excursion (mm), mean ± SD	26.4 ± 3.9	24 ± 7.3	26.2 ± 6.0	0.16
Right ventricular peak systolic wave velocity – S′ (cm/sec), mean ± SD	16.1 ± 2.7	14.2 ± 4.4	16.1 ± 4.1	0.06
Mitral valve peak E wave velocity (m/sec), median (IQR)	0.9 (0.8–1)	0.9 (0.7–1)	0.8 (0.8–1)	0.28
Mitral valve peak A wave velocity (m/sec), median (IQR)	0.6 (0.5–0.7)	0.6 (0.5–0.7)	0.6 (0.5–0.7)	0.16
Mitral valve E/A ratio, median (IQR)	1.5 (1.2–1.7)	1.5 (0.9–1.8)	1.4 (1.2–1.6)	1.0
Mitral valve E wave deceleration time (msec), mean ± SD	208.4 ± 42.1	202.1 ± 66.9	198.3 ± 54.7	0.49
Pulsed-wave tissue Doppler imaging e'_{septal} velocity (cm/sec), mean ± SD	13.7 ± 2.9	13.4 ± 4.5	12.9 ± 3.6	0.36
Pulsed-wave tissue Doppler imaging $e'_{lateral}$ velocity (cm/sec), mean ± SD	18 ± 3.9	17.3 ± 5.9	17.2 ± 4.9	0.64
Pulsed-wave tissue Doppler imaging $e'_{average}$ (cm/sec), mean ± SD	15.8 ± 2.9	15.3 ± 4.8	15 ± 4.0	0.45
$E/e'_{average}$ ratio, median (IQR)	5.6 (4.5–7)	4.5 (4.3–6.9)	5.6 (4.4–7)	0.14
Left ventricular ejection fraction (%), mean ± SD	61.8 ± 5.6	59.8 ± 5.4	62.9 ± 5.5	0.06
Left ventricular global longitudinal strain (%), mean ± SD	−21.3 ± 2.4	-20.1 ± 1.8	-22.1 ± 2.4	0.008

to 320 min, but in patients treated with 100% oxygen at atmospheric pressure is a mean (±SD) of 74 ± 25 min with a range of 26 to 148 min [1].

The most frequent symptom of carbon monoxide poisoning in patients with myocardial injury was loss of consciousness (10 of 17 [58.8%] patients), and none experienced chest pain. Previous research noted that chest pain is present in only about 9% of patients with myocardial damage [9].

Patients with suicidal intoxication typically have longer exposure durations, higher carboxyhaemoglobin levels, and more significant left ventricular damage. This damage is likely related to the duration and concentration of carbon monoxide in inspired air, a catecholamine surge and myocardial stunning [15,16]. In the study of Cha and colleagues [9], suicidal poisoning was observed in 77.4% of patients, averaging 60 years old (range 21–86 years), with a long average exposure time of 9.5 h. In this group, carbon monoxide-induced cardiomyopathy was noted in 74.4% of patients.

In our research, the only echocardiographic parameter that differed significantly between groups based on left ventricular damage was the left ventricular global longitudinal strain. The left ventricular ejection fraction and right ventricular peak systolic wave velocity (S′) did not show statistically significant differences, although there was a trend toward lower values in the myocardial injury group. Moreover, patients with myocardial injury tended to have a higher ($P=0.06$) mean tricuspid regurgitant peak velocity (2.17 m/sec) compared to the non-myocardial injury group (1.94 m/sec). These observations might relate to more common occurrences of tachycardia and hyperkinesia in young patients with myocardial injury and acute carbon monoxide poisoning. No patients in the study group exhibited tako-tsubo-like cardiomyopathy or significant

global left ventricular dysfunction; the lowest ejection fraction in patients with myocardial injury was 51%, compared to 54% in the group without myocardial injury. In the study of Cha and colleagues [9], 74.4% of patients with carbon monoxide poisoning and troponin I concentrations >0.046 ng/mL demonstrated abnormalities in global or regional (tako-tsubo-like) left ventricle dysfunction. However, patients in that study were older than our patients, with only 32.6% being cases of unintentional poisoning and 39.5% having at least hypertension [9].

Jung and colleagues [7] reported that carbon monoxide-induced cardiomyopathy occurs in approximately 3% of all patients. However, echocardiography in that study was performed only on patients with increased troponin concentrations or creatine kinase-MB activities, electrocardiogram changes, dysrhythmias, or continuous low blood pressure (<90 mmHg systolic) [7]. In the study by Rose and colleagues [1], the Heart Brain 346-7 score was proposed for assessing inpatient and 1-year mortality, considering three factors: age, altered mental status, and cardiac complications. Cardiac complications were defined as cardiac arrest, shock, dysrhythmias, myocardial infarction mentioned in clinical chart reviews, increased serum troponin concentration >100 ng/L, and shock requiring vasopressors or inotropes. According to this score, patients under 37 years old without cardiac complications have a good inpatient and 1-year mortality [1].

A significant strength of our study is that echocardiography was performed on all patients admitted to the hospital due to carbon monoxide poisoning who met entry criteria, who did not self-discharge before the procedure could be conducted and in whom adequate imaging could be obtained.

In a study by Saracoglu and colleagues [8], 72 consecutive patients with carbon monoxide poisoning (also with hypertension, coronary artery disease, diabetes, chronic obstructive pulmonary disease) were analyzed and divided into two groups based on left ventricular ejection fraction: those with left ventricular ejection fraction of less than 55% and those with left ventricular ejection fraction of 55% or higher. Left ventricular global longitudinal strain ≥−19.1% was found as an independent predictor of cardiotoxicity with a sensitivity of 70.3% and specificity of 100% (area under the curve 0.840; 95% CI: 0.735–0.916; $P<0.001$). In our study, only four patients in the myocardial injury group and two patients in the non-myocardial injury group had left ventricular ejection fraction under 55%. The rest of the patients exhibited a higher ejection fraction, and left ventricular global longitudinal strain was lower than −19.1%.

Study limitations

This is a single-centre study with a relatively small sample size, which primarily involved young patients who were unintentionally poisoned, predominantly due to malfunctioning heating systems. The neurological evaluation of patients in our study was conducted using various protocols. Hence, these data were not incorporated into our research. We only analyzed changes in patients within a few days of carbon monoxide poisoning as, due to the COVID-19 pandemic, we were unable to conduct follow-up in all patients. However, we are conducting further observations in our patients, obtaining longer-term cardiac imaging and biochemical tests, and data on cardiovascular morbidity and mortality.

Conclusions

In patients who experience moderate to severe carbon monoxide poisoning and who do not have concurrent chronic diseases, echocardiography usually reveals only minor abnormalities, predominantly identified by the analysis of global longitudinal strain.

Disclosure statement

No potential conflict of interest was reported by the authors.

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