Cardiovasc Toxicol (2017) 17:326–334
DOI 10.1007/s12012-016-9389-4

Effects of Acute Carbon Monoxide Poisoning on ECG and Echocardiographic Parameters in Children

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Published online: 24 October 2016

Abstract The aim of the current study is to investigate the effects of CO (carbon monoxide) on the cardiovascular system via electrocardiographic, echocardiographic and biochemical findings in children. This prospective study included 22 children with CO poisoning and 24 healthy children as a control group. The CO-intoxicated children were evaluated via electrocardiography and echocardiography 1 h after admission to the emergency department and daily until their discharge from the hospital. Blood gasses, complete blood account, troponin I and creatinine kinase-MB(CK-MB) were assessed daily. Tpeak–end (p:0.001), QTc durations (p:0.02), Tpeak–end dispersion (p:0.001) and Tpeak–end/QT ratio (p:0.001) of CO-intoxicated patients were significantly higher than those in the control group. Mitral E duration (p:0.001), mitral E/A ratio (p:0.001) and left ventricle contractile fraction (p:0.023) at admission were significantly lower, and left ventricle myocardial performance index was higher (p:0.001) in the CO poisoning group. Troponin I and CK-MB levels were higher at admission in 6 (27 %) and 4 (18 %) patients, respectively. The heart is the most critical organ in pediatric CO poisoning. These children present subclinical systolic and diastolic left ventricle dysfunction even in mild cases. Although, in children with acute CO-intoxication ventricular repolarization is impaired, it seems to be reversible like other findings.

Keywords Carbon monoxide · Cardiomyopathy · Cardiac biomarkers · Echocardiography · Electrocardiography · Dysrhythmia · Poisoning · Ventricular repolarization

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Introduction

Carbon monoxide (CO) poisoning is the most common cause of poisoning-related deaths and complications [1, 2]. Although coal is frequently used as a source of energy in developing countries such as Turkey, actual incidence of CO poisoning is unknown. Myocardial injury is a well-known major complication of CO poisoning. It can be mediated by carboxyhemoglobin (CO-Hb)-induced hypoxia, direct damage to myocardial cellular respiration and to coronary arteries. This results in certain electrical, morphological and functional alterations of the heart. In several studies on myocardial injury-related CO poisoning, changes in electrocardiography (ECG), myocardial dysfunction in echocardiography and a case of myocardial infarction and rupture in adult patients have been reported [3–12]. However, in pediatric patients, there are only few

comprehensive studies related to the cardiac effects of CO intoxication [13].

Nonspecific cardiac repolarization changes and arrhythmias are seen on ECG in patients with CO poisoning. Sudden deaths due to CO poisoning are assumed to occur depending on arrhythmias originating from the ventricles [7, 10, 11, 14]. The T wave in ECG is indicative of ventricular repolarization. It has recently been reported that trans-myocardial repolarization parameters, which include Tpeak–Tend interval (time from the peak to the end of the T wave [Tp-e]), Tp-e dispersion, QT interval, corrected QT interval (QTc), QTc dispersion and the Tp-e/QT ratio, are associated with an increased risk of cardiac arrhythmia [14–16].

In recent years, Doppler echocardiography in combination with tissue Doppler imaging has become a well-accepted, practical, safe and noninvasive method for diagnosing left and right ventricular systolic and diastolic function in the clinical setting [17]. Several clinical studies have revealed that even mild CO poisoning has acute unfavorable effects on left and right ventricular function in adults [5, 6, 8].

The aim of the study is to investigate the effects of CO exposure on trans-myocardial repolarization parameters in the asymptomatic children and possible relationships between these parameters and myocardial injury including echocardiographic features, cardiac biomarkers and CO-Hb levels. Furthermore, in this study, considering the previous emphasis on the effects of CO poisoning on the cardiovascular system in adults, we have hypothesized that even mild CO poisoning may lead to impairment of global ventricular function in children.

Material and Method

Study Populations

This prospective, cross-sectional, controlled, double-blind study included 22 consecutive pediatric patients who were diagnosed with CO poisoning from February 2014 to March 2015 in the emergency department of the hospital. The control group was consisted of 24 healthy children. The diagnosis of CO poisoning was based on medical history with a CO-Hb level of >5 %, and all patients were hospitalized.

Complete physical examination including blood pressure measuring was performed in all patients. 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 kinase-MB(CK-MB) fraction, arterial blood gas 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 CK-MB(Abbott Laboratories, Diagnostics Division, Abbott Park, IL, USA) levels of <24 U/L were considered as normal. Carboxyhemoglobin levels were measured with anOSM3 Hemoximeter (Radiometer, Copenhagen, Denmark). Blood gases including Co-Hb level, troponin I and CK-MB were taken daily until discharged. 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.

Exclusion criteria consisted of history of known cardiovascular disease (congenital heart disease, rheumatic carditis, hemodynamically significant valve regurgitation, heart failure and rhythm disorders), other chronic diseases, use of antiarrhythmic drugs and electrolyte abnormalities.

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. The written informed consent was obtained from the parents of each subject.

All data including demographic, clinical, biochemical, electrocardiographic, echocardiographic and previous medical history were obtained and recorded.

Electrocardiography

Electrocardiography was performed immediately after admission to the emergency department and repeated daily each 24 h until discharge. A Nihon Kohden ECG 1250 Cardiofax S (2009, Tokyo, Japan) device at a standard velocity and amplitude was used. Electrocardiography images had 600-dpi resolution, and measurements were taken on the computer by 2 experts who were blinded to the status of each participant. ST elevation-ST depression in aVR lead, T-wave inversion and QT interval were measured. Based on these measurements, Tp-e, Tp-e dispersion and Tp-e/QT ratio were calculated. The QT interval was defined as the distance from the start of QRS to the end of the T wave in all derivations, and averages were recorded for each group. Tpeak–Tend was measured via the tangent method in precordial leads [14, 18]. A tangential line was drawn where the downward curve of the T wave intersected the isoelectric line. The Tp-e duration was calculated by measuring the distance between the 2 points in the isoelectric line (Fig. 1). The difference between the maximum and the minimum Tp-e in the precordial leads was the Tp-e dispersion.

Echocardiography

A transthoracic echocardiographic investigation (Vivid 7, GE, Horten, Norway) under electrocardiographic monitoring was performed on all patients, using a 3- to 5-MHz

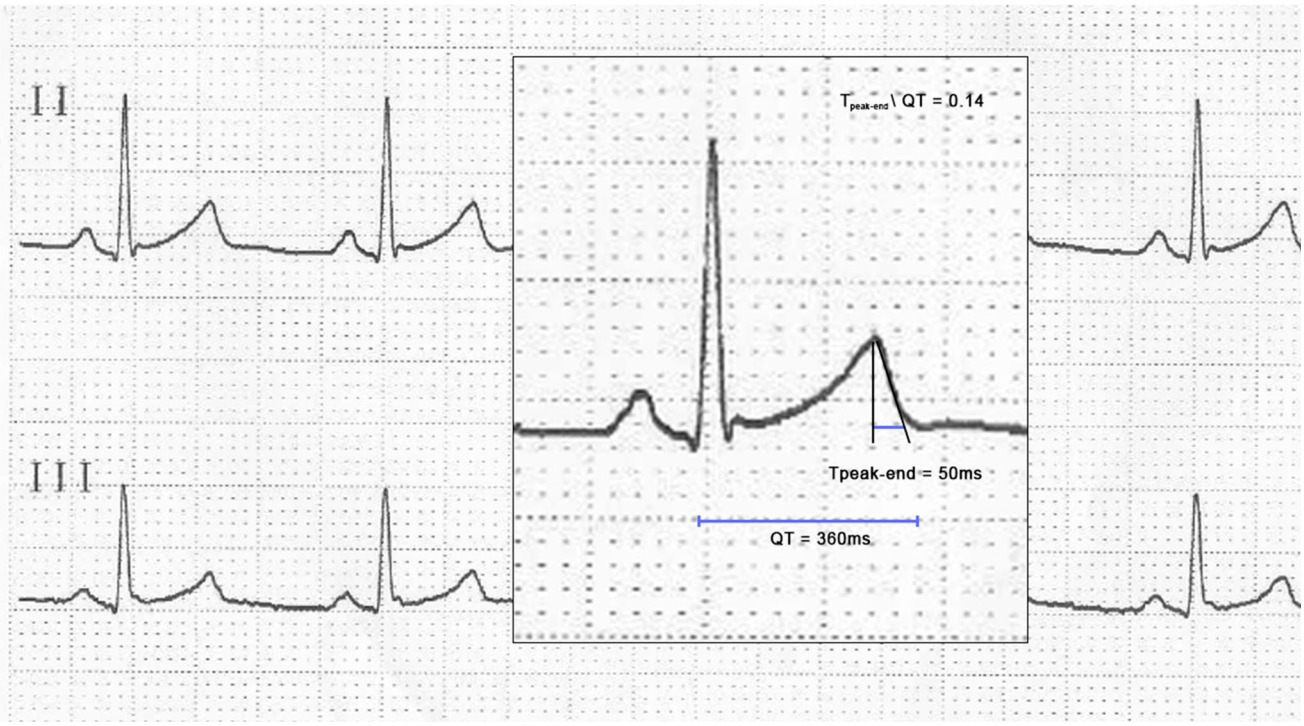

Fig. 1 Measurements of Tpeak–end by the tangent method in precordial leads

transducer. Within 30 min of admission to the emergency department, 2D M-mode, pulsed wave Doppler echocardiography was performed on each subject in the left lateral decubitus position. The pulsed Doppler sample volume was placed at the mitral leaflet tips. Early diastolic peak flow velocity (E), late diastolic peak flow velocity (A), E/A ratio and E wave deceleration time (DT) was measured by using trans-mitral Doppler imaging.

Isovolumetric relaxation time of the left ventricle (IVRT) was defined as the time interval from the cessation of LV outflow to the onset of mitral inflow. The isovolumetric contraction time of the left ventricle (IVCT) was measured from the cessation of mitral inflow to the onset of LV outflow. The myocardial performance index (MPI) of the left ventricle (LV) was calculated by the formula (IVCT + IVRT)/VET [8, 18, 19]. All diastolic parameters were measured in three consecutive cardiac cycles and were averaged. Mitral annular plane systolic excursion (MAPSE), septal annular plane systolic excursion (SAPSE) and tricuspid annular plane systolic excursion (TAPSE) were measured as described before [19]. Left ventricular ejection fraction (EF), MAPSE, SAPSE and TAPSE values were used to demonstrate left–right ventricular contractile (systolic) function. Trans-mitral E wave velocity, A wave velocity, E/A ratios, IVRT, IVCT and ET values were used to demonstrate left ventricular relaxation (diastolic) function, and left ventricle myocardial performance index (LVMPI) was used for left ventricle systolic and diastolic function [8, 19, 20]. Echocardiographic examination of all patients was performed 30 min prior to admission and 1 h after discharge (median hospital stay 3.4 days; min–max: 3–5 days).

Statistical Analysis

Data entry was performed by using the Statistical Package for Social Sciences (SPSS) version 16.0 software package. All variables were first tested for normality of their distribution with Shapiro–Wilk test. Normally distributed variables are presented as mean ± SD, and the non-normally distributed variables are presented as median (25–75th percentiles). To compare the variables across groups, Mann–Whitney U test was used for non-normally distributed variables and two-sample t test was used for normally distributed variable. Spearman correlation test was used for the correlation analysis between two variables. Level of significance was set as $p < 0.05$.

Results

A total of 22 patients (7.8 ± 3.8, 12 females), 24 healthy subjects (8.3 ± 4.3, 12 females) were included in this study. Mean duration of CO exposure was 208 ± 90 min,

and mean hospitalization was 3.4 days (min–max:3–5 days).

Electrocardiographic Results

The Tpeak–end and QTc durations of CO-intoxicated patients were significantly longer than normal subjects (Table 1). At admission, Tpeak–end dispersion value and T peak–end/QT ratio in the intoxicated group was significantly higher than control group (Table 1). It was detected that Tpeak–end, QTc durations, Tpeak–end dispersion value and Tpeak–end/QT ratio were significantly lower after CO poisoning compared to 3.4 days later (during the discharge) (Table 2). The QTc value was detected to be above 440 msn only in 5 CO-intoxicated patients. The QTc durations normalized in all patients before the discharge. No ST changes and arrhythmias were observed in the CO-intoxicated subjects.

Echocardiographic Results

At admission, mitral E duration and mitral E/A ratio showing left ventricle relaxation parameters were significantly lower in intoxicated patients compared to the control subjects (Table 1). Left ventricle (LV) isovolumetric relaxation time (IVRT) duration showing relaxations parameters of CO-intoxicated patients at admission was significantly longer than controls (Table 1). In the CO-intoxicated group, left ventricle myocardial performance index (LVMPI) was significantly higher and left ventricle fractional shortening (LVFS) was significantly lower (Table 1). It was revealed that there is statistically significance in terms of both left ventricle diastolic function parameters (mitral E, mitral IVRT, mitral E/A ratio) and systolic function parameters (LVFS and LVMPI) in CO-intoxicated subjects between admission and discharge (Table 2). Only 2 patients had a left ventricle

Table 1 Demographic, electrocardiographic and echocardiographic result in intoxicated and control groups

	Intoxicated ($n = 22$)	Control ($n = 24$)	p
Demographic characteristics			
Age (year)	7.8 ± 3.8	8.3 ± 4.3	0.782
Gender (female, *n* %)	12/22, (54.5 %)	12/24, (50 %)	0.780
Weight (kg)	23.8 ± 9.7	24 ± 9.4	0.975
Systolic blood pressure (mmHg)	98.3 ± 9.4	102.4 ± 7.7	0.123
Diastolic blood pressure(mmHg)	62.9 ± 9.9	60.7 ± 6.3	0.398
Electrocardiographic characteristics			
Tpeak–end (msn)	65.5 ± 8.6	48.4 ± 8.6	**<0.001**
Tpeak–end variability (msn)	23.9 ± 4.1	15.6 ± 2.7	**<0.001**
Tpeak–end/QT	0.238 ± 0.028	0.167 ± 0.020	**<0.001**
QTc (msn)	0.45 ± 0.12	0.39 ± 0.01	**0.02**
Echocardiographic characteristics			
LVEDD (mm)	34.7 ± 5.8	36.0 ± 5.8	0.452
LVESD (mm)	21.4 ± 3.3	21.3 ± 3.6	0.917
EF (%)	66.1 ± 5.7	69 ± 2.7	0.051
FS (%)	35.2 ± 4.3	39.1 ± 6.5	**0.023**
Mitral E (m/sn)	1.29 ± 0.22	1.72 ± 0.38	**0.001**
Mitral A (m/sn)	0.99 ± 0.37	1.04 ± 0.21	0.410
Mitral E/A (m/sn)	1.19 ± 0.18	1.57 ± 0.32	**<0.001**
LV IVRT	62.1 ± 7.2	52.8 ± 6.4	**<0.001**
LVMPI	0.41 ± 0.05	0.33 ± 0.04	**<0.001**
MAPSE (mm)	10.6 ± 2.6	11.4 ± 1.9	0.709
SAPSE (mm)	11.7 ± 2.4	13.4 ± 1.9	0.582
TAPSE (mm)	23.0 ± 3.8	24.5 ± 2.6	0.831

Bold values indicate statistically significant

Tpeak–end distance from the peak to the end of the T wave on the isoelectric line, *Tpeak–end dispersion* difference between the maximum and minimum Tpeak–end distance on the isoelectric line, *EF* ejection fraction, *FS* fractional shortening, *LVEDD* left ventricle end-diastolic diameter, *LVESD* left ventricle end-systolic diameter, *IVRT* isovolumetric relaxation time, *LVMPI* left ventricular myocardial performance index, *MAPSE* myocardial annular plane systolic excursion, *SAPSE* septal annular plane systolic excursion, *TAPSE* tricuspid annular plane systolic excursion

Table 2 Laboratory, electrocardiographic and echocardiographic changes on admission and discharge in the intoxicated group

	Admission	Discharge	*p*
Laboratory			
Hemoglobin (g/dl)	12 ± 0.87	11.8 ± 0.62	0.274
Leukocyte	10.50 ± 2.71	10.14 ± 2.40	0.249
Thrombocyte	370.45 ± 15.4	363.96 ± 16.1	0.709
Carboxyhemoglobin (%)	18.6 ± 9.2	0.54 ± 0.14	**<0.001**
Bicarbonate (mEq/ml)	19.0 ± 1.3	24.6 ± 1.0	**<0.001**
Troponin (nq/ml)	4.0(1.0–15.7)	1.0(1.0–1.0)	**0.05**
CK-MB (U/L)	2.2 (1.6–3.1)	0.5 (0.6–1.0)	**0.04**
Electrocardiographic			
Tpeak–end (msn)	65.5 ± 8.6	49.6 ± 8.2	**<0.001**
Tpeak–end dispersion (msn)	23.9 ± 4.1	16.3 ± 2.8	**<0.001**
Tpeak–end/QT	0.24 ± 0.028	0.18 ± 0.02	**<0.001**
QTc (msn)	0.45 ± 0.12	0.38 ± 0.01	**0.04**
Echocardiographic			
LVEDD (mm)	34.7 ± 5.8	33.7 ± 5.7	0.452
LVESD (mm)	21.4 ± 3.3	20.0 ± 3.8	0.124
EF (%)	66.1 ± 5.7	70.3 ± 2.9	**0.001**
FS (%)	35.2 ± 4.3	38.8 ± 2.6	**0.001**
Mitral E (m/sn)	1.29 ± 0.22	1.72 ± 0.26	**0.001**
Mitral A (m/sn)	0.99 ± 0.37	1.01 ± 0.28	0.410
Mitral E/A (m/sn)	1.19 ± 0.18	1.66 ± 0.22	**0.001**
LV IVRT	62.1 ± 7.2	50.6 ± 6.9	**<0.001**
LVMPI	0.41 ± 0.05	0.35 ± 0.03	**0.001**
MAPSE (mm)	10.6 ± 2.6	11.1 ± 2.2	0.709
SAPSE (mm)	11.7 ± 2.4	13.1 ± 1.7	0.582
TAPSE (mm)	23.0 ± 3.8	24.4 ± 3.1	0.831

Bold values indicate statistically significant

CK-MB creatinine kinase-MB, *Tpeak–end* distance from the peak to the end of the T wave on the isoelectric line, *Tpeak–end dispersion* difference between the maximum and minimum Tpeak–end distance on the isoelectric line, *EF* ejection fraction, *FS* fractional shortening, *LVEDD* left ventricle end-diastolic diameter, *LVESD* left ventricle end-systolic diameter, *IVRT* isovolumetric relaxation time, *LVMPI* left ventricular myocardial performance index, *MAPSE* myocardial annular plane systolic excursion, *SAPSE* septal annular plane systolic excursion, *TAPSE* tricuspid annular plane systolic excursion

ejection fraction (LVEF) < 60 % in M-mode echocardiography at admission but became normal in 24 h (Fig. 2).

Laboratory and Demographic Features

There was no statistical difference between the groups regarding the systolic–diastolic blood pressure, age and gender. In the intoxicated group, the CO-Hb, bicarbonate, troponin I and CK-MB levels normalized at discharge (mean 3.4 days). The decrease in CO-Hb, troponin I, CK-MB levels and the increase in bicarbonate levels were statistically significant. Troponin I and CK-MB levels were higher at admission in six (27 %) and four (18 %) intoxicated patients, respectively, and both cardiac markers were observed to be normal after 24 h.

Correlation Analysis

In terms of laboratory findings, CO-Hb had a positive correlation with troponin ($r = 0.612$) and CKMB ($r = 0.735$) and negative correlation with bicarbonate ($r = -0.802$). In regard to ECG findings, a positive correlation was observed between CO-Hb levels and Tpe ($r = 0.545$), T dispersion ($r = 0.579$) and Tpe/QTc ($r = 0.475$) values. On the basis of echocardiographic findings, EF ($r = -0.492$), FS ($r = -0.455$), mitral E ($r = -0.452$) and mitral E/A ($r = -0.559$) demonstrated a negative correlation with CO-Hb levels.

On the other side, EF had a negative correlation with troponin ($r = -0.697$), CKMB ($r = -0.464$) and LVMPI ($r = -0.471$). Positive correlations were observed between Tpe/QT ratio and troponin ($r = 0.582$) and CKMB

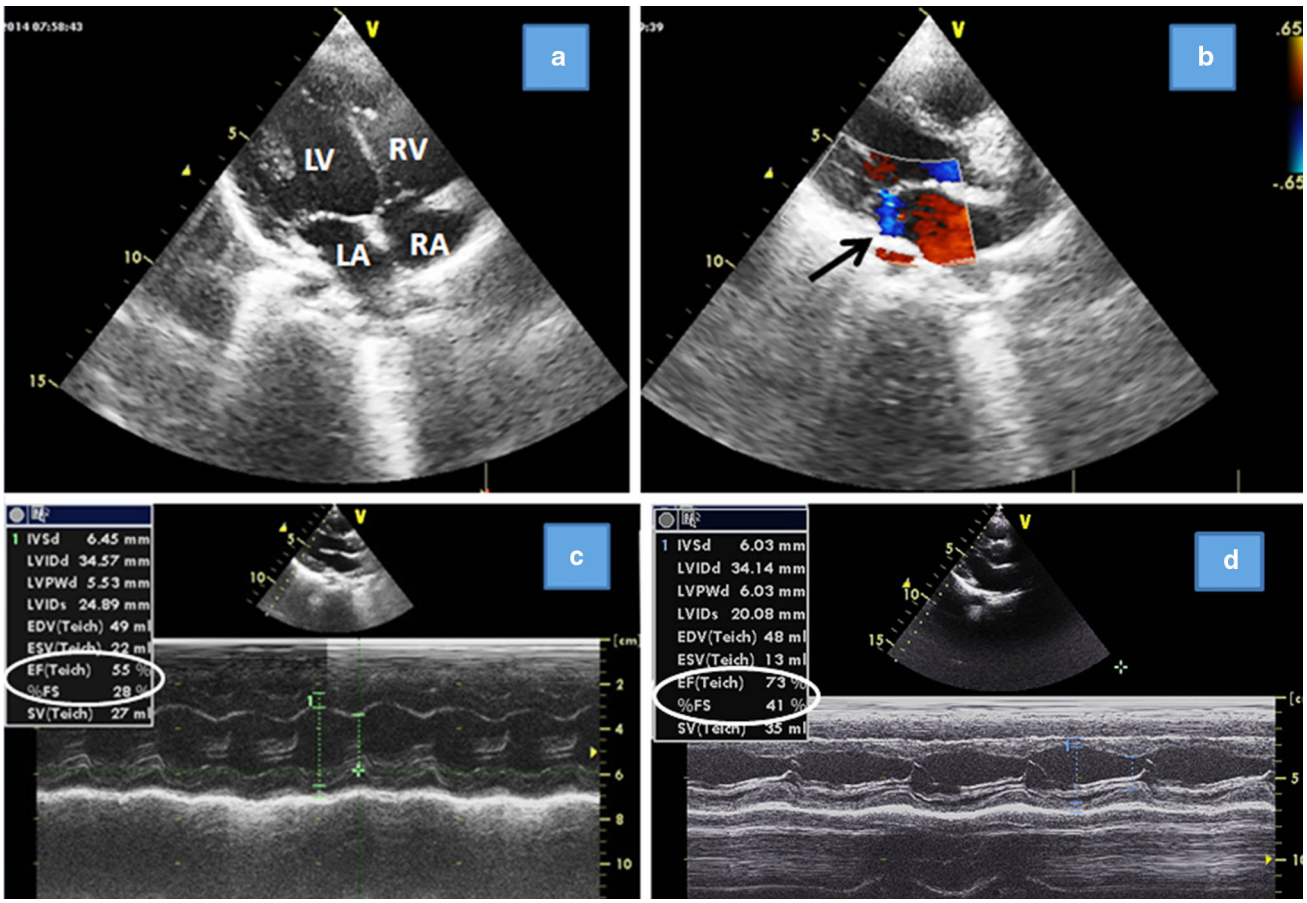

Fig. 2 **a** Enlargement left ventricle in apical four chamber; **b** mild mitral regurgitation jet in parasternal long view on Doppler echocardiography (*black arrow*); **c** decrease left ventricle systolic function with low EF (ejection fraction) and FS (fractional shortening) on M-mode echocardiography at admission; **d** normal left ventricle systolic functions on M-mode echocardiography after three days in same patient. *LV* left ventricle, *RV* right ventricle, *LA* left atrium, *RA* right atrium

($r = 0.496$), whereas there was negative correlation in mitral E/A ratio ($r = -0.515$) (Table 3).

Discussion

The current study demonstrated the negative effects on echocardiography and ECG of CO poisoning with an increase in cardiac enzymes even in asymptomatic children. It also showed that the ventricular repolarization was impaired even without dysrhythmia. However, in several days all of the cardiac effects improved with the normalization of CO-Hb. This is an initial study to show the cardiac effects of CO poisoning with ECG, echocardiography and biochemical markers in a pediatric setting.

Carbon monoxide poisoning can cause reversible or irreversible myocardial injury through the direct toxic effects of myocardial hypoxia and CO. Carbonmonoxide binds to myoglobin and decreases the availability of oxygen, which can cause arrhythmia and cardiac dysfunction [1–3, 21]. The negative effects of CO on cardiovascular system have a wide range, including tachycardia, hypotension, dysrhythmia, ischemia, infarct, cardiac arrest and in some cases myocardial rupture [1–16].

The QTc in an ECG displays the heterogeneous myocardial repolarization. A prolonged QTc indicates an impaired myocardial refractory. A prolonged QTc can cause a wide range of arrhythmias, including ventricular fibrillation, torsades de pointes and polymorphic ventricular tachycardia. It was statically shown that CO intoxication increases the QTc interval in adults similar to the current study [10, 11, 21].

Tpeak–end interval, Tpeak–end dispersion and Tpeak-end/QTc ratio which are novel trans-myocardial repolarization parameters defining trans-myocardial heterogeneity [15, 16, 18]. Moreover, some studies report that the Tp-e interval, Tp-e dispersion, and Tp-e/QT ratio are superior to the QT interval and QT dispersion in predicting ventricular arrhythmias [22]. In the study conducted in adults with acute CO poisoning, it was found that the Tp-e interval was

Table 3 Correlation between carboxyhemoglobin(CoHgb) and other characteristics

	CoHgb	Troponin	CKMB	$HCO3^-$	EF	FS	Mitral E	Mitral E/A	LVMPI	Tp-e	T p-e dispers.	Tp-e/QTc
CoHgb	–	*r*:0.612	*r*:0.735	*r*:−0.802	*r*:-0.492	*r*:−0.455	*r*:−0.452	*r*:−.0559	NS	r:0.545	r:0.579	*r*:0.475
		p:0.002	*p*:0.001	*p*:0.001	*p*:0.020	*p*:0.033	*p*:0.034	*p*:0.007		*p*:0.009	*p*:0.005	*p*:0.025
Troponin	*r*:0.612	–	NS	*r*:−0.718	*r*:−0.697	*r*:−0.680	*r*:−0.524	NS	NS	NS	*r*:0.431	*r*:0.582
	p:0.002			*p*:0.001	*p*:0.001	*p*:0.001	*p*:0.012				*p*:0.045	*p*:0.005
CKMB	*r*:0.735	NS	–	NS	*r*:−0.464	*r*:−0.439	NS	*r*:−0.527	NS	NS	*r*:0.438	*r*:0.496
	p:0.001				*p*:0.030	*p*:0.042		*p*:0.012			*p*:0.041	*p*:0.019
$HCO3^-$	*r*:−0.802	*r*:−0.718	NS	–	NS	NS	*r*:0.582	*r*:0.495	NS	NS	*r*:−0.496	–NS
	p:000.1	*p*:0.001					*p*:0.005	*p*:0.019			*p*:0.019	
EF	*r*:−0.492	*r*:−0.697	*r*:−0.464	NS	–	*r*:0.965	NS	NS	*r*:−0.471	NS	NS	NS
	p:0.020	*p*:0.001	*p*:0.030			*p*:0.001			*p*:0.027			
FS	*r*:−0.455	*r*:−0.680	*r*:−0.439	NS	*r*:0965	–	NS	NS	NS	NS	NS	NS
	p:0.033	*p*:0.001	*p*:0.042		*p*:0.001							
Mitral E	*r*:−0.452	*r*:−0.524	NS	*r*:0.582	NS	NS	–	*r*:0.608	*r*:−0.484	NS	NS	NS
	p:0.034	*p*:0.012		*p*:0.005				*p*:0.003	*p*:0.023			
Mitral E/A	*r*:−.0559	NS	*r*:−0.527	*r*:0.495	NS	NS	*r*:0.608	–	NS	NS	NS	*r*:−0.515
	p:0.007		*p*:0.012	*p*:0.019			*p*:0.003					*p*:0.014
LVMPI	NS	NS	NS	NS	*r*:−0.471	NS	*r*:−0.484	NS	–	NS	NS	− NS
					p:0.027		*p*:0.023					
Tp-e	*r*:0.545	NS	NS	NS	NS	NS	NS	NS	NS	–	NS	NS
	p:0.009											
Tp-e dispers.	*r*:0.579	*r*:0.431	*r*:0.438	*r*:−0.496	NS	NS	NS	NS	NS	NS	–	NS
	p:0.005	*p*:0.045	*p*:0.041	*p*:0.019								
Tp-e/QTc	*r*:0.475	*r*:0.582	*r*:0.496	NS	NS	NS	NS	*r*:−0.515	NS	NS	NS	–
	p:0.025	*p*:0.005	*p*:0.019					*p*:0.014				

r values were defined as follows for the correlation analysis; *r*:0.00–0.20 (very weak), 0.21–0.40: weak, 0.41–0.60 (moderate), 0.61–0.80 (strong), 0.81–1.00 (very strong)

Co-Hb carboxyhemoglobin, *CK-MB* creatinine kinase-MB, *$HCO3^-$* bicarbonate, *LVMPI* left ventricle myocardial performance index, *Tpeak–end* distance from the peak to the end of the T wave on the isoelectric line, *Tpeak–end dispersion* difference between the maximum and minimum Tpeak–end distance on the isoelectric line

significantly longer [14]. In another adult study, similarly, a significant increase in Tp-e dispersion and Tp-e/QTc ratio has been reported [7]. In our study, QTc interval, Tp-e interval, Tp-e dispersion and Tp-e/QTc ratio were significantly increased in the CO-intoxicated children similar to adults (Table 1). Moreover, a positive correlation was observed between CO-Hb levels and Tp-e interval, Tp-e dispersion and Tp-e/QTc ratio. Additionally, troponin I and CKMB were shown to have a positive correlation with Tp-e dispersion and Tp-e/QTc ratio (Table 3).

The MPI, as an index of global myocardial performance, can be obtained as an easy and reliable marker which is independent from heart rate and blood pressure [19, 20]. It has been reported that higher MPI levels might be associated with adverse cardiac events like myocardial infarction [23]. Our results revealed that even mild CO poisoning has acute negative effects on left ventricular systolic (lower FS), diastolic (lower mitral E and mitral E/A ratio) and global (higher LVMPI) functions. Çiftçi, et al. [8] found no statistically significant difference in terms of LVMPI between the groups in a study conducted in 20 adult patients with CO poisoning. However, the present study has shown statistical differences by means of LVMPI measurements in the patient group itself for a-week duration between the pre- and post-periods of admission. This finding indicates that the acute effect of CO poisoning on left ventricle function is reversible and improves in a short while without hyperbaric oxygen treatment. In contrary to the study of Ciftçi et al. [8], our study has shown high LVMPI measurements in patients and also there is statistically significance in terms of LVMPI measurements in patients during admission and discharge. This condition can be explained by the increased sensibility of myocardial tissue to damage due to CO inhalation in children as compared to adults. The morbidity and mortality of viral or toxic myocarditis in the newborn and early infants are higher than adults. One of the attributed causes is increased fragility due to myositis at this age group [24]. The mechanism which causes this state is expressed as increased basal metabolism with less blood volume and high tissue oxygen demand in children.

In our study, the intoxicated group has shown a negative correlation between EF, FS, mitral E, mitral E/A and CO-Hb. Additionally, two intoxicated children developed acute left ventricle dilated cardiomyopathy characterized with low EF-FS and the cardiomyopathy resolved within four days.

Study Limitations

The limitation of this study was the relatively small number of patients with CO poisoning. Due to the fact, a follow-up investigation with a larger sample size is required. Long-term follow-up studies supported with advanced diagnostic and screening methods such as myocardial scintigraphy and cardiac MRI may possibly give detailed information about the cardiovascular side effects of CO poisoning.

Conclusion

We conclude that ventricular repolarization impairment has appeared before any electrocardiographic dysrhythmia in mild acute CO poisoning in children. These changes seem to be completely reversible. Besides, left ventricular contractile and relaxation impairment has also been found in these patients. Acute CO intoxication temporarily increases troponin I and CKMB levels, and cardiac enzyme levels are correlated with echocardiographic and electrocardiographic abnormalities. Further studies are required to evaluate the importance of these new electrocardiographic and echocardiographic parameters in children with CO poisoning.

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