

Can Tp-e/QTc Ratio and Blood Lactate Levels Serve as an Earlier Indicator Than Troponin for Detecting Cardiac Ischemia in Patients with Carbon Monoxide Poisoning in the emergency Department?

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Abstract

Carbon monoxide (CO) poisoning is a clinical condition with serious cardiotoxic effects. This study reports two cases of CO poisoning to evaluate whether Tp-e/QTc ratio and blood lactate levels can serve as earlier indicators of cardiac ischemia than troponin levels. Two male patients, aged 37 and 47, who presented to the emergency department due to CO exposure, were analyzed. Their electrocardiographic (ECG) parameters, troponin levels, and lactate values were compared. Additionally, the clinical course of both patients was assessed.

In the first case (37 years old), ST depression in leads V1-V6 was detected on ECG, and troponin levels showed a progressive increase. Coronary angiography revealed no significant narrowing of the epicardial coronary arteries. The patient's QTc interval was 481 ms at admission, Tp-e duration was 79 ms, and Tp-e/QTc ratio was 0.16. Upon discharge, these values returned to normal. The second case (47 years old) had normal ECG and laboratory findings and was discharged without complications. Furthermore, in the first case, lactate levels were significantly higher at admission and remained elevated for a prolonged period.

Minimal QTc prolongation and a significant increase in lactate levels following CO poisoning may serve as early indicators of cardiac ischemia. Despite normal troponin levels, the first case required further cardiac evaluation. These findings suggest that monitoring QTc and lactate levels may help in the early detection of cardiac involvement in CO poisoning. However, further research is required to establish the prognostic value of these markers.

Keywords: Carbon monoxide poisoning, Cardiac ischemia, Tp-e/QTc ratio, Blood lactate level, Electrocardiography.

Introduction

Carbon monoxide (CO) poisoning can occur due to various sources, including fuels used for heating and cooking, industrial chemical production, poorly ventilated environments with motor vehicle emissions, leaks in home heating systems, and intentional exposure. Regardless of the source, CO exerts toxic effects on the human body, primarily due to tissue hypoxia, affecting the cardiovascular and neurological systems. Numerous studies in the literature have investigated the etiology, frequency, and prognostic factors of CO poisoning¹⁻⁴. In CO poisoning, diagnosis is primarily based on a history of exposure, the presence of nonspecific symptoms, and blood carboxyhemoglobin (COHb) levels. However, there are a limited number of studies focusing on predicting prognosis and organ damage in emergency settings⁵⁻⁷. In this article, we present two cases in which we compare data suggesting that the cardiotoxic effects of CO poisoning may be detected earlier using specific parameters rather than troponin levels.

Case Presentations

Two patients presented to the emergency department approximately two hours after exposure to fumes from a coal-burning heating stove, complaining of headache, fatigue, nausea, vomiting, dizziness, and shortness of breath.

Case 1

A 37-year-old male patient admitted at the emergency department in stable condition with no impairment in consciousness. On physical examination, blood pressure, 138/88 mmHg; heart rate, 112 beats/min; body temperature, 36.4°C; and respiratory rate, 34/min. After presentation to the emergency department, 100% oxygen therapy was started at 15 L/min using a reservoir mask without respiration and intravenous access was provided. The patient did not report any active complaints other than feeling generally unwell. The patient had no medical history and tobacco use, prior surgical procedures, or regular medication use. On physical examination, breath sounds were normal, and there

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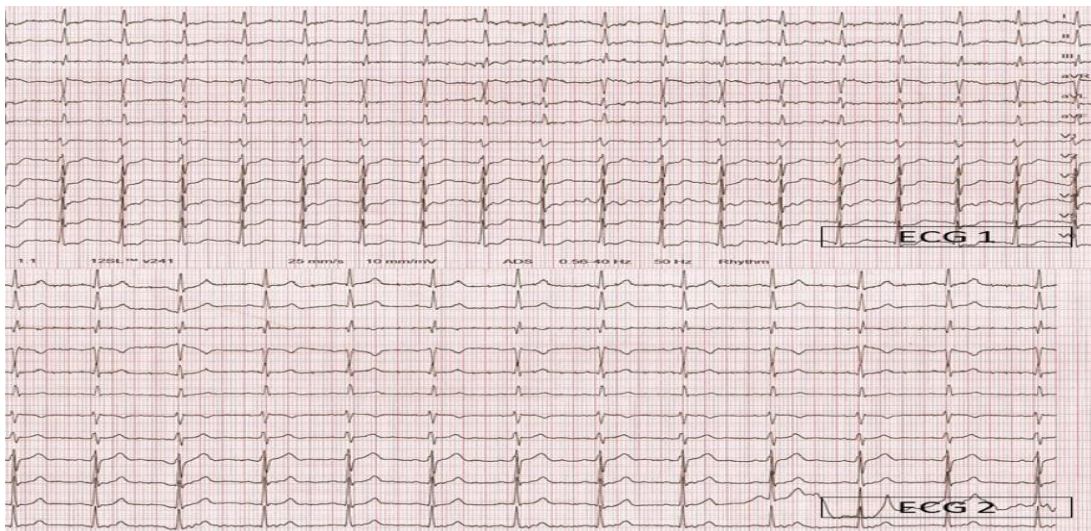


Figure 1: ECG 1-2

were no signs of dyspnea, neurological deficits, or chest pain. However, his electrocardiogram (ECG) revealed ST depression in leads V1-V6. Transthoracic echocardiography (TTE) showed no pathological findings, and chest radiography was unremarkable. At hospital admission, ECG findings (Figure 1, ECG 1) revealed a QTc of 481 ms, Tp-e of 79 ms, and a Tp-e/QTc ratio of 0.16. Upon discharge (Figure 1, ECG 2), QTc decreased to 456 ms, Tp-e was 80 ms, and the Tp-e/QTc ratio was 0.17. Additionally, lactate levels at admission were significantly higher in Case 1 compared to Case 2, and despite normobaric oxygen therapy, Case 1 exhibited prolonged lactate elevation relative to Case 2.

Due to the presence of ST depression in leads V1-V6 on initial ECG and progressive elevation of troponin levels, Case 1 was admitted to the coronary intensive care unit. As troponin levels continued to rise, the patient underwent coronary angiography (CAG), which revealed normal coronary arteries. Following 48 hours of observation, the patient was discharged after normalization of troponin

levels and the absence of additional complications related to CO poisoning.

Case 2

A 47-year-old male patient, also in stable condition with no impairment in consciousness, presented with the following physical examination findings: blood pressure, 127/77 mmHg; heart rate, 98 beats/min; body temperature, 36.7°C; and respiratory rate, 32/min. He was immediately started on 100% oxygen therapy at 15 L/min via a non-rebreathing reservoir mask, and intravenous access was established. Like to Case 1, he had no medical history. He did not use tobacco, had no prior surgical history, and was not on any regular medication. His physical examination revealed normal breath sounds without signs of dyspnea, neurological deficits, or chest pain. His ECG and chest X-ray findings were within normal limits. Laboratory test results for both patients are presented in **Table 1**. Given he absence of active

Table 1: Patients' laboratory values

Patient	Laboratory values	1*	2**	3***	At discharge	Mean	Median	St. Deviation	Rate of Change	Normal values
Case 1	pH	7.26	7.35	7.41	7.36	7.34	7.35	0.06	0.008	7.35-7.45
	PO ₂ mmHg	129	272	160	84	161.2	144.5	80.1	-3.75	83-108
	PCO ₂ mmHg	27.5	38.4	31.5	39.4	34.2	34.9	5.6	0.99	32-48
	SO ₂ %	98.2	99.4	99.2	96.3	98.2	98.7	1.4	-0.15	95-99
	Lac mg/dL	79	22	13	10	31	17.5	32.4	-5.7	5.0-14.0
	MetHb %	1.6	2.2	1.8	1.8	1.85	1.8	0.2	0.01	0-1.5
	O ₂ Hb %	67.4	67.4	96.3	96.5	81.9	81.8	16.7	2.4	94-98
	COHb %	29.8	11.9	1.3	0.9	10.9	6.6	13.5	-2.4	0.5-1.5
	cHCO ₃ ST	14.5	20.6	22	22.4	19.8	21.3	3.6	0.65	-
	TROPONIN ng/L	300.5	777.4	1129	494	675.2	635.7	360.3	16.12	0-19.8
Case 2	pH	7.336	7.39	7.368	7.39	7.36	7.37	0.28	0.001	7.35-7.45
	PO ₂ mmHg	208	247	128	81.1	166.2	168.5	75.0	-2.64	83-108
	PCO ₂ mmHg	40.5	40	44	43.6	42.0	42.0	2.06	0.064	32-48
	SO ₂ %	99.2	99.6	98.7	95.5	98.2	98.9	1.86	-0.077	95-99
	Lac mg/dL	27	9	7	7	12.5	8.0	9.7	-0.41	5.0-14.0
	MetHb %	2.1	1.5	2	1.7	1.8	1.8	0.27	-0.008	0-1.5
	O ₂ Hb %	69.9	96.1	95.7	92.7	88.6	94.2	12.5	0.47	94-98
	COHb %	27.2	2	1	1	7.8	1.5	12.94	-0.54	0.5-1.5
	cHCO ₃ ST	23.1	24.2	24.2	25.7	24.3	24.2	1.06	0.54	-
	TROPONIN ng/L	10.7	34	65.9	91.6	50.5	49.9	35.5	1.68	0-19.8

*Admission to the hospital, **2nd Hour, ***6th Hour

complaints, normal laboratory values, and stable clinical findings, Case 2 was discharged 12 hours after admission.

Discussion

Pathological cardiac changes observed during CO poisoning are often associated with prognosis⁸. Electrocardiographic changes such as ST and T-wave abnormalities, QT prolongation, and arrhythmias are frequently reported in CO poisoning cases⁸. While a significant correlation between the cardiac effects of CO and T peak-to-T end (Tp-e) dispersion and Tp-e/QTc ratio has been documented, there is limited research on whether these parameters serve as early warning markers for cardiac ischemia in CO poisoning⁹.

The slightly elevated QTc values observed in Case 1 align with existing literature suggesting that minimal QTc prolongation may serve as an early marker of cardiac involvement in CO poisoning⁹. Previous studies indicate that lactate levels rise before troponin in CO poisoning, suggesting that lactate may serve as an early biomarker for cardiac ischemia. Furthermore, lactate levels have been associated with prognosis prediction in CO poisoning cases^{6,10}.

In the present report, two cases with no significant differences in medical history were evaluated, yet Case 1 demonstrated a progressive increase in troponin levels compared to Case 2, raising concerns for acute coronary syndrome (ACS) and prompting the emergency department team to perform further investigation. Case 1 was monitored in the coronary intensive care unit (ICU), and subsequent coronary angiography (CAG) confirmed patent coronary arteries, thereby verifying cardiac involvement secondary to CO toxicity. Following comprehensive evaluation, treatment, and monitoring, the patient was discharged in good health. A retrospective comparison of the two cases was conducted to identify potential differences. To minimize measurement errors, ECG intervals (QT, QRS, and Tp-e) were manually measured using calipers and magnification. The QT interval was measured from the beginning of the QRS complex to the end of the T wave, and heart rate-corrected QT (QTc) was calculated using Bazett's formula. Tp-e interval was defined as the duration from the peak to the end of the T wave. Our findings support existing literature demonstrating the prognostic significance of elevated lactate in CO poisoning-related cardiac ischemia and hospital stay duration^{6,10}.

Limitations

One of the main limitations of this study is the lack of long-term follow-up for both cases, preventing further assessment of myocardial ischemia using imaging modalities such as myocardial scintigraphy. It is well established that age is a risk factor for poor prognosis in CO poisoning, partly due to the increased prevalence of comorbid conditions with

aging. However, in our cases, the younger patient exhibited a more severe clinical course, suggesting that unidentified physiological factors may contribute to individual susceptibility to CO poisoning. These factors represent the primary limitations of the presented cases.

Conclusion

Both patients were exposed to CO in the same environment for the same duration. Despite no significant differences in baseline characteristics, one patient required advanced evaluation, treatment, and ICU admission. This case highlights the potential role of minimal deviations in QTc from normal values as a clinically relevant marker of coronary ischemia in CO poisoning.

Additionally, the persistently elevated lactate levels in Case 1, which demonstrated a weaker response to oxygen therapy, suggest that lactate measurements alongside QTc monitoring may provide valuable insights into the early cardiac effects of CO poisoning. These findings indicate that the combined assessment of these two parameters may improve early detection of cardiac involvement in CO poisoning. However, further research is required to establish rapid and reliable biomarkers that can be used at the time of emergency department admission to predict prognosis and reduce observation times in CO poisoning cases.

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