



High-sensitivity Troponin I Assay for Early Detection of Myocardial Injury in Carbon Monoxide Intoxication

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Background: Carbon monoxide (CO) intoxication is the most common gas-related event resulting in emergency room (ER) visits, often leading to myocardial injuries measured through the assessment of troponin levels. Recent efforts have focused on developing high-sensitivity troponin assays for more accurate acute myocardial infarction diagnosis. However, studies using high-sensitivity troponin I (hs-TnI) assays to identify myocardial injury following CO intoxication are lacking. **Aim:** We compared the measurement of circulating troponin I levels using the hs-TnI assay and the contemporary troponin I (cTnI) assay. **Methods:** We prospectively analyzed 50 patients with CO intoxication who were admitted to the ER between September 2012 and October 2013. Clinical, demographic, electrocardiographic (ECG), echocardiographic, and laboratory data were compared. Circulating troponin I levels were measured using hs-TnI and cTnI assays upon arrival in the ER and 24, 48, and 72 h after admission. **Results:** Most patients with moderate or severe CO intoxication had elevated circulating troponin I levels, as detected using the hs-TnI assay. In addition, none of the results above or below the cutoff value for circulating troponin I changed from those found in the ER. In the cTnI assay, elevated circulating troponin I above the cutoff value in the ER was detected in 21 of 36 patients (58%). Participants with elevated troponin I levels, as measured by the hs-TnI assay, were more likely to experience various unfavorable clinical outcomes. **Conclusion:** The hs-TnI assay can detect elevated circulating troponin I levels earlier than the cTnI assay in patients with CO intoxication and myocardial injury.

Key words: High-sensitivity cardiac troponin I, carbon monoxide intoxication, myocardial injury

INTRODUCTION

Carbon monoxide (CO), an odorless and colorless gas, is present at low concentrations in the atmosphere but can be toxic and even fatal at high concentrations. CO has a high affinity for hemoglobin (Hb) and can replace oxygen in the blood following long-term exposure. This leads to reduced oxygen delivery to tissues resulting in hypoxia.¹ The heart and brain are the primary organs that are impacted, resulting in the manifestation of related symptoms. The impact of CO

on the cardiovascular system includes arrhythmia, ventricular dysfunction, heart failure, sudden death, and myocardial infarction.^{2,3} Circulating troponin assays are widely used to detect myocardial ischemia and myocardial infarction.⁴ Serum troponin levels are not only related to the extent of myocardial injury but also predict the prognosis of myocardial infarction.⁵ Contemporary troponin I (cTnI) assays can only

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How to cite this article: Tsai TN, Shen CH, Lin GM, Liu PY, Liu MY, Huang LY. High-sensitivity troponin I assay for early detection of myocardial injury in carbon monoxide intoxication. J Med Sci 2024;44:173-8.

Received: July 03, 2023; Revised: October 24, 2023;
Accepted: October 24, 2023; Published: March 06, 2024
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detect myocardial injury more than 4 h after it has occurred. High-sensitivity troponin I (hs-TnI) assays have been developed to overcome this limitation. hs-TnI assays are also 10 times more sensitive than cTnI assays.⁶ Therefore, hs-TnI assays have the potential to be a tool for the early detection of myocardial injury or myocardial infarction, to change the current diagnostic method, and to improve the management of affected patients.

cTnI assays are widely used to detect myocardial injury during CO intoxication.⁷⁻⁹ However, studies using hs-TnI assays in patients with CO toxicity are lacking. In the present study, we investigated the daily serum levels of troponin I in patients with moderate and severe CO intoxication using hs-TnI and cTnI assays.

MATERIALS AND METHODS

Participant selection

The present prospective study enrolled patients with moderate or severe CO intoxication who visited the Emergency Department of the Tri-Service General Hospital (TSGH) between December 2012 and October 2013. TSGH is a medical center in Taiwan that provides 24-h hyperbaric oxygen therapy. Patients were classified as having acute CO intoxication if their history suggested acute exposure to CO and their arterial serum *carboxyhemoglobin* level was >15%. The exclusion criteria were as follows: age <20 years old, pregnancy, history of coronary artery disease, or suspected coronary artery disease. Fifty patients were included in the study. The study was conducted according to the principles of the Declaration of Helsinki and was approved by the Institutional Review and Ethics Board of the Tri-Service General Hospital, Taipei, Taiwan (2-101-05-052). All participants provided informed consent before the examinations. The procedures were performed according to the institutional guidelines.

The following information was collected from all the enrolled patients: age, sex, initial Glasgow Coma Scale (GCS) score, COHb level, arterial blood gas data, and laboratory data. Laboratory data included serum levels of white blood cells, Hb, platelets, sodium (Na), potassium (K), creatinine, creatine kinase (CK), and cTnI. The cTnI assay was performed using a Beckman Coulter Access immunoassay analyzer, and the lower limit of detection was 0.5 ng/mL.

Collection, storage, and assay with high-sensitivity troponin I

Blood samples were collected on arrival at the emergency room (ER) and 24 and 48 h after arrival. Blood was processed by two-step centrifugation, and the supernatant

was transferred to a *ribonuclease*-free Eppendorf tube. The samples were stored at -80°C until the assessments were performed. The hs-TnI assay was performed using an Elecsys 2010 autoanalyzer (Roche Diagnostics) with an ARCHITECT STAT hs-TnI immunoassay; the lower limit of detection was 0.3 ng/mL.

Electrocardiogram and echocardiography

Electrocardiogram (ECG) and echocardiography were performed upon arrival at the ER and again at 24, 48, and 72 h after arrival. Two cardiologists analyzed the ECG data. Ischemic changes on the ECG were defined as new ST-segment elevation (>1 mm), depression (>0.5 mm), or T-wave inversion in two consecutive leads. Echocardiography was performed using the Philips IE33 system (Phillips Medical Systems, Andover, MA, USA), and the images obtained included parasternal long axis, short axis, and two-chamber, four-chamber, and five-chamber views. Color Doppler imaging was performed at a horizontal sweep velocity of 100 mm/s to exclude valvular dysfunction. Ejection fraction (EF) was automatically calculated using the software. To improve reproducibility, data were analyzed by another observer.

Statistical analysis

Participants were categorized into two groups according to the predicted cutoff value for the cTnI and hs-TnI assay in the ER, i.e. 0.5 ng/mL and 0.3 ng/mL. The differences in clinical characteristics between the two groups were evaluated using the *t*-test for continuous variables (e.g. age, COHb, and ejection fraction). Statistical significance was set at $P < 0.05$, and all *P* values were two-sided. The SPSS (IBM Corp. Released 2012. IBM SPSS Statistics for Windows, Version 21.0. Armonk, NY: IBM Corp) statistical software was used for data processing and modeling.

RESULTS

Clinical characteristics of the participants

Fifty patients diagnosed with CO intoxication between December 2012 and October 2013 were enrolled. The average age of participants was 39.9 years, and 56% (28/50) were men. The mean GCS score upon arrival at the ER was 6.68. Endotracheal tube insertion with ventilator support was required in 72% of participants because of acute respiratory failure or for airway protection [Table 1]. The average COHb level in arterial gas was 32.6%. Sinus tachycardia was present in 82% of participants and 38% had ischemic changes upon arrival at the ER. None of the patients showed new ischemic changes at the 48-h follow-up ECG. The average EF of

participants was 39.3%, and 58% had a depressed ejection fraction (EF <40%) on arrival at the ER. The average CK serum level on arrival was 1681.44 U/L. The mean values of the troponin I levels on arrival at the ER by cTnI and hs-TnI assays were 1.06 and 2.13 ng/mL, respectively.

The character of circulating troponins I analyzed by high-sensitivity troponin I and contemporary troponins I assay

Changes in troponin I levels were measured using both hs-TnI and cTnI assays. Blood was collected when the patients arrived at the ER and 24, 48, and 72 h after admission. The hs-TnI assay identified 33 (66%) patients above the cutoff value for serum troponin I, while the cTnI assay identified

36 (72%) patients. Participants with hs-TnI-measured troponin levels above the cutoff value had higher COHb levels, lower GCS scores, and depressed EF. They were also more likely to have undergone endotracheal tube insertion and had ischemic changes on ECG performed at the ER [Table 2].

The hs-TnI assay detected peak serum troponin I levels in 45 participants (90%) in the ER and 5 participants (10%) at 24 h after admission. Using the cTnI assay, eight participants (16%) had a peak serum level in the ER, with only one having a level above the cutoff value. Of the remaining participants, 29, 11, and 2 participants had peak serum levels at 24, 48, and 72 h after admission, respectively. Interestingly, all serum levels of troponin I measured by the hs-TnI assay showed consistent results, i.e. either elevated or not elevated levels, for 72 h. On the other hand, using the cTnI assay, 15 participants (30%) who had a level below the cutoff value in the ER had a level above the cutoff value within 72 h [Table 3].

DISCUSSION

Cardiovascular injury is a major consequence of CO poisoning. Cardiovascular involvement may present as myocardial ischemia, stunning, arrhythmia, heart failure, myocardial infarction, or sudden death.^{2,3} The detection of myocardial injury after CO exposure using ECG lacks sensitivity. Various biomarkers have been suggested to detect

Table 1. The characteristic of patients with CO intoxication

Characteristic	Results
Mean age (yo)	39.9
Male/Female	28/22
Average of COHb (%)	32.6%
Number of ECG with ischemia change	19 (38%)
Average of Ejection Fraction(%)	39.3%
Number of patient receive intubation with ventilator support	36 (72%)
Average of GCS on ER	6.68
COHb: carboxyhemoglobin, ECG: electrocardiogram, GCS: Glasgow Coma Scale, ER: Emergency room	

Table 2. General parameters of patients with carbon monoxide intoxication assessed using the troponin I assay method

	Contemporary Troponin I Assay		High-Sensitivity Troponin I Assay	
	Above predict cutting value	Below predict cutting value	Above predict cutting value	Below predict cutting value
Number of patients	36	14	33	17
Male/Female	20/16	8/6	19/14	9/8
Average of age (yo)	39.7	40.5	39.2	41
Average of COHb (%)	34.2	31.8	34.5	31.7
Ischemia change on ECG	17	2	17	2
Average of EF on ER (%)	37.8	43.1	37.8	43
Endotracheal tube insertion	28	8	25	11
GCS on ER	6.1	8.1	6	7.9
Average value on ER (ng/ml)	1.26	0.10	3.19	0.11
Average Peak value (ng/ml)	4.19	0.17	4	0.1
Number of patients below predict cutting value on ER	15	14	0	17
The peak of troponin I present on				
ER	1	13	29	16
24hours o f admission	21	1	4	1
48 hours o f admission	12	0	0	0
72 hours o f admission	2	0	0	0
DNS	10	2	10	2

Table 3. Predictors of elevated circulating troponin I levels assessed using high-sensitivity troponin I assay

	High-Sensitivity Cardiac Troponin I Assay		P
	Above predict cutting value	Below predict cutting value	
Number of patients	33	17	
Age (yo)	38.2±11.6	43.2±14.6	0.230
COHb (%)	34.5±16.4	31.7±15.9	0.552
EF on ER (%)	37.8±12.4	42.4±11.1	0.189
GCS on ER section	6.0±3.8	7.9±5.1	0.197
White Blood Cells (/ul)	19171±7187	12398±3614	0.001*
Hemoglobin (mg/dl)	14.8±1.9	14.5±2.2	0.654
Platelet (/dl)	279716±89422	265353±69455	0.535
Sodium (mmol/L)	139.2±2.7	138.6±2.9	0.511
Potassium (mmol/L)	3.69±0.85	3.66±0.52	0.862
PH	7.32±0.14	7.40±0.06	0.007*
HCO ₃ ⁻ (mmol/L)	18.0±5.7	20.6±4.1	0.070
Creatinine (mg/dL)	1.37±0.51	1.17±0.61	0.252
Creatine kinase (U/L)	1820±4492	1413±3128	0.711

COHb: carboxyhemoglobin, ECG: electrocardiogram, EF: ejection fraction, GCS: Glasgow Coma Scale, ER: Emergency room.

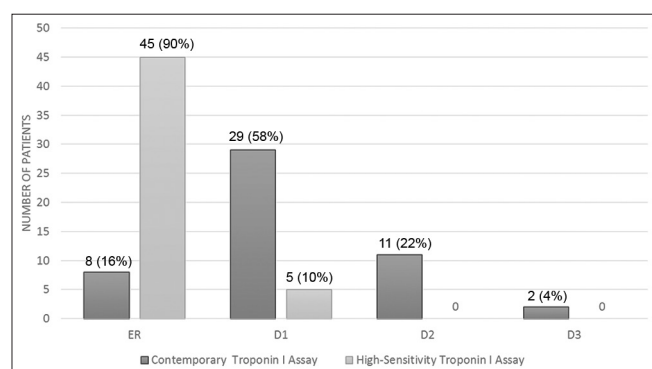


Figure 1: Number and percentage of patients presenting peak troponin levels as assessed using the contemporary troponin I and high-sensitivity troponin I assays during the follow-up period

myocardial injury in the ER.⁸⁻¹⁰ Troponin I, a cardiac protein, is released into circulation when myocardial necrosis occurs. Various conditions, including CO intoxication, can cause an increase in circulating troponin I, impacting the long-term prognosis of affected individuals.¹¹⁻¹⁴ CO intoxication can cause myocardial damage that results in the elevation of circulating troponin I levels, especially in cases of severe and moderate intoxication.^{7,15,16} Elevated troponin levels are associated with increased short- and long-term mortality rates in patients with CO intoxication.¹⁷ The cTnI assay can detect damage 4 h after myocardial infarction. The newer hs-TnI assay shortens the detection time to 2 h.⁶ The use of hs-TnI assays in ERs has increased worldwide, owing to their high sensitivity and ability to detect myocardial injury early. Here, we were able to achieve early detection of myocardial injury by utilizing

the hs-TnI assay. Circulating troponin I levels detected with this method had consistent results, i.e. were either elevated or not, over the period of 72 h. However, when using the cTnI assay, 15 participants (30%) had levels below the cutoff value in the ER, but elevation above the cutoff value within 72 h. This suggests that the hs-TnI assay is more sensitive than the cTnI assay.

Serial detection of circulating troponin levels has been used to determine the progression of myocardial injury in myocardial infarction. A decrease in circulating troponin levels indicates a reduction in myocardial injury. The early detection of peak serum troponin levels can help in decision-making in clinical practice. Our data showed that for most patients, the peak level of troponin I was detected sooner by the hs-TnI assay than by the cTnI assay [Figure 1] or CK (data not shown). This suggests that the hs-TnI assay can detect the peak level of troponin I earlier than the cTnI or CK assays used in previous myocardial infarction studies.^{18,19} A study from Cha *et al.*²⁰ showed that 20% of patients had myocardial injury on hospital arrival. The average time for circulating troponin I to reach its peak level was 11 h after ER admission.²⁰ In our study, we found that the majority of patients (45 out of 50, 90%) exhibited a peak level of hs-TnI in the ER, while the remaining five patients reached their peak level within 24 h of admission. Using the cTnI assay, most patients (44%) had circulating troponin I within 24 h of admission. This may be because we collected samples every 24 h and our cases were more critical with generally higher levels of troponin I (0.9 vs. 4.18 ng/mL). Interestingly, three patients had a troponin I level above the cutoff value

when assessed by the cTnI assay, but not when measured by the hs-TnI assay. These patients had levels below the cutoff value, as detected by the hs-TnI assay, within the follow-up period. The circulating troponin I was found to be mildly elevated (0.55, 0.69, and 0.93 ng/mL) above the cutoff value when measured by the cTnI assay 24 h after admission, but not in ER. This could also be attributed to the elevated peak levels of hs-TnI, which were not reached upon arrival at the ER, but gradually decreased to normal levels within 24 h. Therefore, a large-scale study is required to clarify this issue.

A previous study reported that myocardial injury occurred in approximately one-third of patients with severe CO intoxication and was related to the duration of CO exposure and hospital mortality.²¹ The severity of myocardial injury is related to the duration and amount of CO exposure.²² Satran *et al.* showed that 35% of patients with moderate-to-severe CO poisoning had CK-MB or elevated troponin I.²³ In our study, elevated circulating troponin I was detected in 66% of participants by the hs-TnI assay and in 72% of participants by the cTnI assay. The higher levels in our study may be due to our participants being more critically ill (72% underwent endotracheal tube insertion) and in need of admission for further management.

Huysal *et al.* demonstrated that circulating troponin T, measured using a high-sensitivity troponin T assay, was not correlated with COHb blood levels. In our study, we also found that circulating troponin I levels did not correlate with blood COHb levels, which is consistent with the results of previous studies that measured troponin I levels using the cTnI assay.²⁴ Myocardial injury caused by CO toxicity may be triggered by inflammation.¹ Baydin *et al.* found that the white blood cell count was significantly increased upon myocardial injury.²⁵ In the present study, we also found an elevated white blood cell count related to elevated troponin I levels, as detected by the hs-TnI assay [Table 3].

Delayed neuropsychiatric sequelae (DNS) are serious neurological impairments associated with CO intoxication. Cha *et al.* reported a statistical difference in troponin I levels between non-DNS and DNS patients.²⁶ In our study, we found that participants with a circulating troponin I level above the cutoff value, as measured by the hs-TnI assay, were more likely to present with DNS sequelae than those below the cutoff value (30% vs. 11.8%). However, there was no statistically significant difference between the two groups, which may be due to the small number of participants included in this study.

CONCLUSION

In the present study, we demonstrated that the hs-TnI assay could detect myocardial injury earlier than the cTnI assay

in patients with moderate or severe CO intoxication. Most patients assessed using the hs-TnI assay presented with a peak serum troponin I level within 24 h of the event. Participants with a circulating troponin I level >0.3 ng/mL, as measured by the hs-TnI assay, had more DNS events and higher white blood cell counts than those with a level <0.3 ng/mL.

Study limitations

The relatively small number of participants is the main limitation of the present study. Therefore, a long-term study with a larger sample size is warranted. Moreover, patients with coronary artery disease should be excluded from future examinations including myocardial scintigraphy or coronary computed tomography angiography, which may provide more accurate information on the impact of CO poisoning on the cardiovascular system. In addition, the current data were generated using the ARCHITECT Stat hs-TnI and cTnI (Roche Diagnostics) assays and may not be applicable to other high-sensitivity assays.

Acknowledgments

We thank Dr. Chung Kan Peng, Dr. Chun Hsien Wu, Dr. Kun-Lun Huang and Dr. Cheng-Chung Cheng for clinical care of the enrolled patients.

Data availability statement

The datasets utilized in this study are not publicly available; however, they can be obtained from the corresponding author upon a reasonable request.

Financial support and sponsorship

This work was supported in part by the Tri-Service General Hospital (TSGH-C104-029; TSGH-C106-005-007-S04), Taoyuan Armed Forces General Hospital (804-C104-163), and Teh-Tzer Study Group for Human Medical Research Foundation (B1051005).

Conflicts of interest

There are no conflicts of interest.

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