

Increased Troponin-I As An Indicator of Myocardium Injury in A Patient with Severe Carbon Monoxide Poisoning — A Case Report

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Abstract

Myocardial injury can be identified by elevated serum level of troponin, a regulatory protein of thin actin filaments of the cardiac muscle. In patients with inconclusive electrocardiogram (ECG) finding (i.e., no ST-segment elevation), a positive troponin I (Tn I) is 100% and troponin T (Tn T) is 75% predictive of acute myocardial infarction. In daily practice, physicians would not routinely check serum Tn I level in patients suffering from carbon monoxide (CO) intoxication if patients do not complain of chest discomfort or when ECG examination shows no significant ST-segment changes. We report a 20-year-old previously healthy soldier with CO intoxication. Acute myocardial infarction was diagnosed by elevated serum Tn I that was further supported by typical serial cardiac enzyme changes, and severe systolic hypokinesia of left ventricle in echocardiogram. This case suggests the importance of measuring serum Tn I level to early disclose myocardium injury in patients with CO intoxication. (J Intern Med Taiwan 2008; 19: 355-359)

Key Words : Troponin-I, Carbon monoxide intoxication, Acute myocardial infarction

Introduction

Myocardial injury can be identified by elevated serum troponin level. The level of serum cardiac troponin is reported to be more sensitive than creatine kinase MB (CK-MB) for the detection of acute myocardial infarction (AMI) in adults, though some false positive cases of measuring cardiac troponin may occur in patients with chronic renal failure. There are two troponin isoforms, i.e., troponin T (Tn T) and troponin I (Tn I). Both were reported to have similar sensitivity and specificity for the diagnosis of AMI. In patients with inconclusive electrocardiogram (ECG) finding (i.e., no ST-segment elevation), a positive Tn I was 100% and Tn T was 75% predictive of AMI. On the other hand, a Tn T level of less than 1.0 ng/ml at 12 hours after onset of chest pain may effectively exclude the possibility of AMI^{9,11,23}.

In daily practice, physicians would not routinely check serum Tn I level in patients suffering from carbon monoxide (CO) intoxication if patients do not complain of chest discomfort or when ECG examination shows no significant ST-segment changes. Here, we report a 20-year-old previously healthy soldier who suffered from CO intoxication. Acute myocardial infarction was diagnosed by elevated serum Tn I and further supported by typical serial cardiac enzyme changes, and hypokinesia of left ventricle in echocardiogram.

Case Report

A 20-year-old previously healthy soldier was sent to the Emergency Department (ED) of Saint Paul's Hospital due to disturbed consciousness. The witness reported that he was seized with incomplete combustion of coal. CO intoxication was impressed by the ED physician at once.

On arrival at the ED, the blood pressure was 106/66 mmHg, the heart rate was 127/min, the respiratory rate was 20/min, and the body temperature was 38.2 °C. He presented with spontaneous breathing and

the oxygen (O₂) saturation was 99.9% under 100% oxygen non-rebreathing mask. The Glasgow coma scale was 7 (E1 V2 M4). The pupils were isocoric with normal light reflex.

Laboratory examination at ED showed that the patient had normal hemogram except high white blood cell count (WBC, 35,440/ μ L). WBC differential count showed left side shifting with 87.5% being segment form of WBC. The serum glucose level (94 gm/dl) and alcohol concentration (5.7 mg/dl) were normal. Blood chemistry examination revealed slightly abnormal renal (Creatinine = 2.4 mg/dl) and liver (AST = 99 U/L) functions. The ED physician was alert enough to check the serum Tn I level, and it yielded 5.549 ng/ml (normal, <0.4 ng/ml at our laboratory). The CK-MB was 50.2 U/L (normal, < 10.4 U/L). Arterial blood gas analysis showed a pH of 7.429 with a PCO₂ = 29.4 mmHg, PO₂ = 485.9 mmHg, HCO₃ = 19.4 mmol/L, and base excess = -5.3 mmol/L. Total hemoglobin levels (THb) was 16.2 (normal, 13.5-17.5) gm/dl, the percentage of CO-bound hemoglobin was 26.2% (normal, 0.5 - 1.5% for nonsmoker and 4 - 9% for smoker), MET-Hb was 0.3% (normal, 1.4-1.5%), and O₂-HB was 73.4% (normal range for arterial blood, 94-100%).

Chest AP radiograph revealed normal heart size and configuration without definite active lung lesion. Computed tomography (CT) of the brain without enhancement showed essentially negative finding. CT of the abdomen with and without enhancement demonstrated 1) minimal infiltration in right low lung and no definite pleural effusion, 2) no remarkable finding of the visceral organs, and 3) swelling and hypodensity of right gluteus maximus and left gluteus minimus muscles, suggestive of secondary to ischemic change. Emergent echocardiogram revealed severe LV systolic hypokinesia, trivial MR, no right ventricular enlargement, no paradoxical septal motion, and no tricuspid regurgitation.

Tentative diagnosis of acute myocardial infarction was made based on the troponin data and

Table 1. Cardiac enzymes evaluation showed typical changes of MI

Time	7/11	7/11	7/11	7/12	7/12
Item	13:11	17:40	18:43	06:05	11:35
CPK (30-130U/L)		14062	13811	24330	34682
CK-MB (<10.4ng/mL)	50.2	91.5	202	342	571
Troponin-I (0.4ng/mL)	5.549	14.005	15.135		

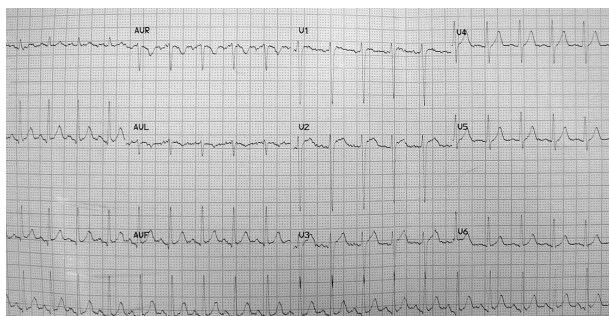


Fig. 1. Electrocardiogram at the ED showed sinus tachycardia without STT change.

echocardiogram findings. We assumed that complete coronary occlusion was unlikely to occur in such young patient. Therefore, medications, such as aspirin, heparin, and thrombolytic agents, were not prescribed. Subsequent ECG examination at 12 hours after the initial examination showed no significant ST-T changes (Fig. 1). Serial cardiac enzymes evaluation showed typical changes of MI (Table 1). Coronary angiogram was not performed due to critical condition of the patient. The patient was transferred to another hospital for hyperbaric oxygen therapy. Unfortunately, the patient expired at the next day after hospitalization.

Discussion

Suicide attempt has increased in recent decades, and it poses 7th rank in the top ten causes of death in Taiwan. Incomplete combustion of coal is one of the most common way people undertook to commit suicide. Therefore, physicians are about to face more and more patients with CO intoxication. The most obvious presentation after CO intoxication is consciousness disturbance that was familiar to most physicians and frequently reported in the literature. However,

less attention was paid to myocardial injury after CO intoxication in the previous literature.

Cardiac troponins I and T are known to start rising within 3-4 hours after myocardial infarction and remain raised for 4-10 days because of a gradual degeneration of myofibrils with release of the troponin complex^{7,22}. Micha M. et al. (2006) reported that elevation of serum cardiac troponin level was due to transient loss in membrane integrity with subsequent troponin leakage or microvascular thrombotic injury¹⁷. The controversies concerning whether irreversible myocardial damage or reversible myocardial depression caused troponin release in patients with sepsis remain unsettled.²⁴ The fact that elevated troponin level has been found in patients with unstable angina suggests that troponin leakage due to ischemia or other stimuli is possible even if no myocardial necrosis develops¹³. Thus, raised cardiac troponins alone will never allow us to make a clinical diagnosis. The most important issue is that cardiac troponins contain prognostic information for most of these conditions. Previous studies have confirmed that cardiac dysfunction and grave outcome are implicated by elevated troponin level^{2,4,16,17}.

Early marked increase (about 15 folds of normal value) of serum Tn I level in the reported case indicated that myocardial injury developed soon after carbon monoxide exposure. Severe global left ventricle systolic hypokinesis disclosed in echocardiogram gave support to extensive myocardial dysfunction that reached cellular level. Previous studies reported that patients with CO intoxication presented with ST segment and T wave change at ECG examination¹⁵. ECG of the reported case revealed no ST-T change.

Chest pain was not possible to be a complaint due to consciousness disturbance of patients with CO intoxication. All these reasons may lead the physician to neglect the existence of myocardial infarction. However, serial typical changes of cardiac enzymes implicated the diagnosis of myocardium infarction. The results of this case report remind us to keep in mind that there is a possibility of myocardium infarction in patients with CO intoxication. We thus suggest that serum Tn I level should be measured in unconscious patients with CO intoxication.

Studies investigating the pathophysiology of CO intoxication suggest the mechanism underlying CO intoxication may be the disruption of intracellular respiratory reaction by carbon monoxide binding to the mitochondrial cytochrome oxidase a3^{6,12,19}. Theoretically, patients with CO intoxication will suffer from impaired oxygen delivery due to high affinity of CO with hemoglobin. The impairment of oxygen delivery in the capillary level will result in subsequent ischemic insult to the myocardium¹. Thus, myocardial infarction may not result from coronary artery occlusion in patients with CO intoxication. This point is supported by what reported by Lee *et al*¹⁵. They reported a 42-year-old woman suffering from CO intoxication in combination with ST-segment changes in ECG traces, while the coronary artery angiogram was normal. They hypothesized that impaired oxygen delivery and disturbed intracellular mitochondrial metabolism were responsible for the ischemic insult associated with CO exposure, and suggested hyperbaric oxygen therapy (HBOT) may be beneficial to the patient.

The mechanism and pathophysiology of severe CO intoxication induced myocardial damage is distinct from acute coronary syndrome resulting from coronary atherosclerosis. The treatment strategy about myocardium infarction after CO exposure is thus different from those for coronary atherosclerosis. In subject with severe CO intoxication induced AMI, the mainstay of management lies on the use of

hyperbaric oxygen therapy (HBOT) and oxygen by non-rebreathing mask if HBOT is not available¹⁴. Some studies reported that carboxyhemoglobin can lead to patchy myocardial necrosis in human and laboratory animals^{3,10,18,20}. Steven *et al.* (1974) reported that previously existed coronary atherosclerosis may increase the possibility of angina pectoris and even produce myocardial infarction after CO exposure²¹. The pre-existing disparity between oxygen supply and oxygen demand in patients with coronary atherosclerosis will limit the ability of coronary flow to increase and thus further interfere the compensatory response to carbon monoxide exposure. Treatment of underlying coronary atherosclerosis may thus be helpful in patients with known history of coronary atherosclerosis and CO intoxication. Furthermore, hematological studies demonstrated increased thrombotic tendency secondary to platelet stickiness and polycythemia in patients with CO intoxication. These studies indicated that thrombosis of vessels after CO exposure may be another possible cause leading to acute myocardial infarction^{5,8}.

In conclusion, this case report suggests the importance of measuring serum Tn I level to disclose myocardium injury in patients with CO intoxication. Elevated Tn I level indicating myocardium injury may be detected soon after CO intoxication. Elevated cardiac troponins T and I implicate grave prognosis that suggest the patient should be managed aggressively. Therefore, we strongly recommend that Tn I should be routinely measured in patients suffering from CO intoxication to early detect the presence of AMI. However, well-designed prospective, randomized, controlled clinical trials are needed to give evidence of checking Tn I on CO exposure.

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嚴重一氧化碳中毒患者檢測肌鈣蛋白病例報告

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摘要

肌鈣蛋白 (Troponins) 是診斷急性心肌梗塞的重要標記。I 型肌鈣蛋白 (Tn I) 是存在於心臟肌肉細胞內之調節蛋白質，因此當心肌細胞受損時，血清中肌鈣蛋白數值便會升高。實證醫學顯示，雖病人心電圖未呈現 STT 節段上昇，若檢測血清 I 型肌鈣蛋白升高時，在診斷急性心肌梗塞之正確率達 100%，而檢測血清 T 型肌鈣蛋白升高，則診斷正確率達 75%。醫師平時針對一氧化碳中毒患者之醫療處置，若患者沒有胸部不適，或心電圖 STT 節段的無異常變化，並不會常規檢測血清 I 型肌鈣蛋白。本文為探討一名 20 歲原身體健康狀況良好的軍人，因懷疑一氧化碳中毒，檢測血清中 I 型肌鈣蛋白上昇，而診斷為急性心肌梗塞。特提出報告，藉能拋磚引玉，有更大規模研究來確立嚴重一氧化碳中毒病患，檢驗肌鈣蛋白的重要性。