Case Report

Myocardial injury mimicking acute myocardial infarction from methane gas intoxication: a case report and literature review

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Abstract: Methane gas is a colorless and suffocating gas. We described the first case of methane gas intoxication in which the patient showed lethal myocardial injury. A 45-year-old man was presented to our hospital with unconsciousness after an accidental exposure to methane gas for 3 hours. On admission, serum troponin I was mild elevated. The initial electrocardiogram (ECG) showed sinus tachycardia. After 3 days, ECG showed diffuse elevation of the ST segment, and the patient developed refractory ventricular fibrillation (VF) and cardiac pump failure and died. However, coronary artery angiography showed that there was no occlusion. Therefore, it is possible that ischemic changes on the later ECG can predict in-hospital adverse cardiovascular events for methane gas intoxication.

Keywords: Intoxication; methane; myocardial injury

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Introduction

Methane exposure has been well documented in accidents involving workers in coal mines or urban tunnels (1-3). Most of patients die of burn/inhalation injury and asphyxiation. There is a paucity of information about myocardial injury mimicking acute myocardial infarction from methane gas intoxication. This is the first case described here in which electrocardiographical manifestations of myocardial injury is more like acute myocardial infarction.

Case presentation

A 45-year-old man presented to our hospital with unconsciousness for 3 hours. Before collapse, he went into urban tunnels for saving his friend. He had a history of essential hypertension and no other relevant medical history. On admission, he had a body temperature of 36 ℃, heart rate of 100 beats/min, respiratory rate of 20/min, and blood pressure of 124/60 mmHg. The rest of physical examination was otherwise unremarkable except for deep unconsciousness. Blood tests revealed that his white blood cell counts were significantly elevated and serum troponin I was mild elevated. However, his liver and renal parameters were within normal limits. Electrocardiogram (ECG) revealed sinus tachycardia (*Figure 1*). His computed tomography of the lung revealed exudative lesions in right mid-low lobe and left lower lobe. He was admitted to the hospital. The patient was given 125 mL of 20% mannitol every 8 hours to lower intracranial pressure for 7 days. He also received hyperbaric oxygen therapy and supportive care. On hospital day (HD) 1 he was awake and complained of headache and generalized fatigue.

On HD 3 he developed dramatic chest pain and nausea. ECG showed the elevation of ST segment and wide QRS on lead I, II, III, aVL, aVF, and V2-6 (*Figure 2*). However, coronary artery angiography showed that there was no occlusion (*Figure 3*). Serum troponin I was 0.22 ng/mL (normal value: <0.1 ng/mL). The patient was diagnosed as acute methane gas intoxication and myocardial injury. Then, hormone was administered to decrease myocardial injury.

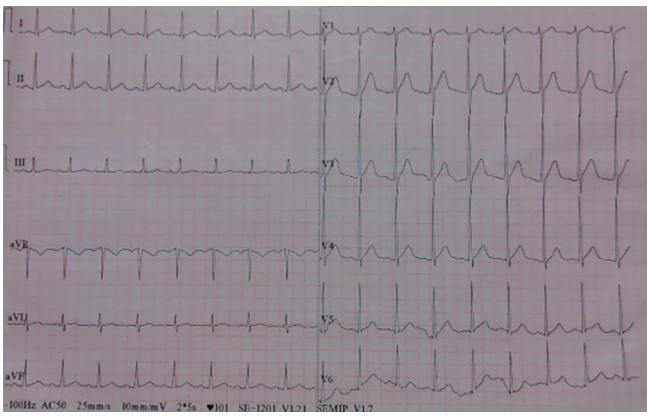

Figure 1 Electrocardiogram showing sinus tachycardia on admission.

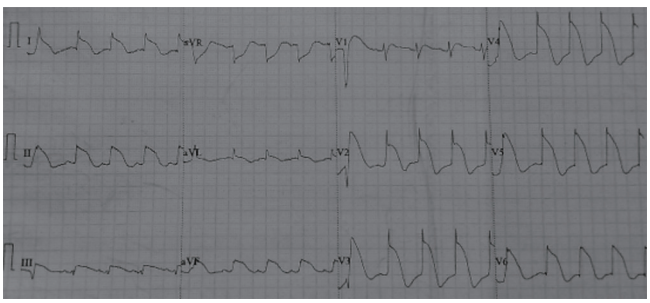

Figure 2 Electrocardiogram showing ST elevation and wide QRS on hospital day 3.

On HD 4 the patient still felt dramatic chest pain. Computed tomographic pulmonary and aortic angiography showed there was no pulmonary embolism (*Figure 4A*) and dissection of aorta (*Figure 4B*). On HD 5 the patient developed refractory ventricular fibrillation (VF). Then, cardiopulmonary resuscitation such as repeated defibrillation, amiodarone and vasoactive drugs was delivered and the patient was resuscitated. However, the patient developed severe cardiac pump failure and died.

Discussion

Methane gas is one of the most commonly suffocating gases and can cause asphyxia, cardiac arrest and death (4). After methane gas intoxication, oxygen from inspired air is displaced by methane gas. The metabolism of cells in the body is affected by acute hypoxemic hypoxia (5). Thus, all organs may be injured, especially for the brain and heart. This is the physiopathological mechanism by which methane gas intoxication leads to death.

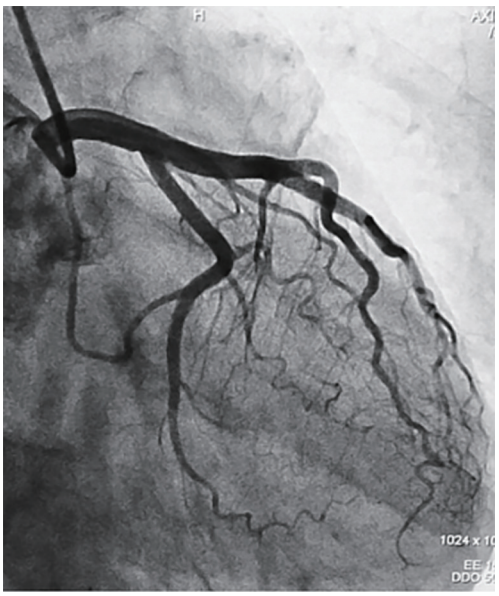

Figure 3 Coronary artery angiography showing no lesion on hospital day 3.

After accidental exposure, the patient became unconscious. Besides, serum troponin level was elevated and ECG revealed sinus tachycardia. All these evidences demonstrated the brain and myocardium in the patient were obviously injured. Later, the patient was awake. However, he had severe chest pain and developed VF. Although serial ECG revealed the manifestation of acute myocardial infarction, coronary artery angiography did not detect any lesion. It indicated that the patient had myocardial ischemia not myocardial infarction. This lent further credence to the hypothesis that microvessels in myocardium were occluded by microthrombus not macrovessels. But, the definite mechanism of action was still unknown. Further autopsy was warranted.

As well as we know, methane gas is known to produce systemic hypoxia. Additionally, any intoxication associated with severe tissue hypoxia can produce ST segment changes due to ischemia (6,7). In this case, diffuse elevation of the ST segment was found later and VF occurred finally. Although the patient was resuscitated, he died of cardiac pump failure. Therefore, it is possible that ischemic changes on the later ECG can predict in-hospital adverse cardiovascular events for methane gas intoxication.

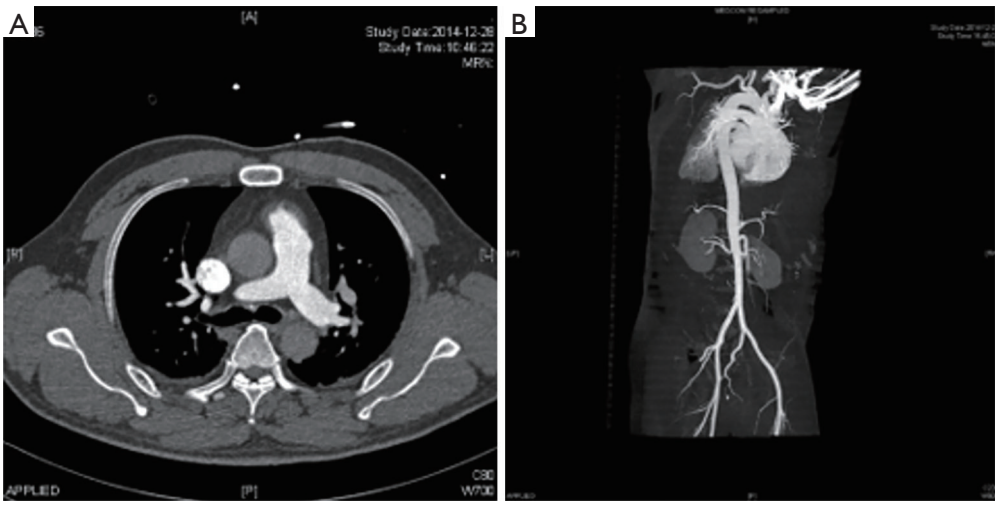

Figure 4 Computed tomographic pulmonary and aortic angiography showing no pulmonary embolism (A) and dissection of aorta (B) on hospital day 4.

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Footnote

Conflicts of Interest: The authors have no conflicts of interest to declare.

Informed Consent: Written informed consent was obtained from family member of the patient for publication of this Case Report and any accompanying images.

References

1. Knoblauch A, Steiner B. Major accidents related to manure: A case series from Switzerland. Int J Occup Environ Health 1999;5:177-86.
2. Nagao M, Takatori T, Oono T, et al. Death due to a methane gas explosion in a tunnel on urban reclaimed land. Am J Forensic Med Pathol 1997;18:135-9.
3. Krzywiecki A, Ziora D, Niepsuj G, et al. Late consequences of respiratory system burns. J Physiol Pharmacol. 2007;58 Suppl 5:319-25.
4. De-Giorgio F, Grassi VM, Vetrugno G, et al. Homicide by methane gas. Forensic Sci Int 2012;221:e1-3.
5. Rossi R, De-Giorgio F, Grassi VM, et al. An unusual suicide: asphyxia by methane gas. Am J Forensic Med Pathol 2013;34:83-5.
6. Yates C, Manini AF. Utility of the electrocardiogram in drug overdose and poisoning: theoretical considerations and clinical implications. Curr Cardiol Rev 2012;8:137-51.
7. Inoue Y, Kumagai K, Tanaka T, et al. Lethal myocardial injury associated with hydrogen sulfide poisoning: report of two cases. Chudoku Kenkyu 2011;24:231-5.

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