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Late-onset sick sinus syndrome after carbon monoxide poisoning

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ABSTRACT

Carbon monoxide (CO) is a known, potent poisonous gas that causes hypoxaemia because of its high affinity for haemoglobin. It also induces inflammatory responses that cause tissue injury, particularly to the nervous and cardiovascular systems. Here we present a case of late-onset sick sinus syndrome (SSS) after CO poisoning. Arrhythmia during the acute phase has been recorded in the literature, but to the best of our knowledge, this is the first report of late-onset SSS. Late-onset neuropathy after CO poisoning is well known, and it seems that a similar mechanism develops in cardiac conduction after CO poisoning. This report highlights the importance of follow-up for arrhythmia after CO poisoning.

Key words: sick sinus syndrome, carbon monoxide poisoning, arrhythmia, neurologic injury, cardiac injury

INTRODUCTION

Carbon monoxide (CO) intoxication is one of the most common types of poisoning worldwide. However, the underlying mechanism of injury and treatment methods remains unclear. CO is a colourless, odourless, tasteless gas produced by incomplete combustion of carbonaceous material. CO forms carboxyhaemoglobin (COHb), which decreases oxygen transport. Depending on the COHb level and exposure time, patients have various non-specific symptoms, ranging from headache, myalgia and dizziness to severe conditions, such as unconsciousness and eventually death. (1) Lack of clinical specificity often leads to CO poisoning being overlooked or misdiagnosed. In addition to these acute-phase symptoms, neuropathy and psychiatric disorders are well known. (1) Neuropsychological sequelae occur at 6 weeks after poisoning in 46% patients. (2) However, we have not paid attention to arrhythmias after CO poisoning as much. Here we report a case of late-onset sick sinus syndrome (SSS) after CO poisoning.

CASE PRESENTATION

An 87-year-old Japanese woman was found unconscious in a room with a briquette heater and was brought to our hospital. She had been exposed to CO for at least 12 h. She was under treatment for high blood pressure and Hashimoto disease, without arrhythmia. Medical examination findings obtained 4 months earlier, including electrocardiogram (ECG) results, revealed no abnormalities. On admission, her initial Glasgow Coma Scale score was 6/15 (E1V1M4), her pupils were 3/3 mm and her pupillary reflex was slow/slow (right/left). Her blood pressure was 162/69 mmHg, but other vital signs were non-specific. Physical examination revealed no abnormalities. The results of laboratory studies, including routine haemogram, electrolyte level, blood sugar levels and thyroidal function, were in the normal range. The troponin I level was 0.008 ng/mL, creatinine kinase level was 135 IU/L, and ECG showed normal cardiac function (figure 1). Myocardial damage was not suspected. Blood gas tests showed pH 7.394, PO2 level of 194.0 mmHg, PCO2 level of 43.4 mmHg, HCO3 level of 25.9 mmHg, base excess level of 1.2 mmol/L and COHb level of 16.0%. The actual COHb level may have been higher than the measured level because she already had been treated with an oxygen reservoir mask (10 L/min) for 30 min during time spent in the ambulance. Brain computed tomography revealed no cerebral infarction or haemorrhagic lesions. We diagnosed her with CO poisoning, and hyperbaric oxygen therapy (HBO) was initiated and continued for 5 days. Her level of consciousness gradually improved, and she was shifted from the intensive care unit on day 5. On day 13, she developed bradycardia, with a heart rate between 30 and 40 beats/min; however, she did not have any symptoms. Sinus arrest occurred for 5 s at midnight on the following day (figure 2). The next day, a prolonged RR interval (maximum, 4.08 s) was detected by Holter electrocardiography. Echocardiography showed no dilatation, and the ejection fraction was 77.1%. She was diagnosed with SSS, and a pacemaker was implanted. The pacemaker was set at 60 beats/min and no spontaneous rhythm was noted thereafter. She did not develop other late-onset neurologic injuries, such as dementia, parkinsonism or changes in the globus pallidus on magnetic resonance imaging.

DISCUSSION

In this case, the patient experienced late-onset SSS after CO poisoning. Before hospitalisation and even after hospitalisation, the patient had a normal sinus rhythm, but she gradually developed bradycardia, which progressed to SSS over the course of approximately 2 weeks.

CO functions as a neurotransmitter in physiologic amounts. It modulates inflammation, (3) apoptosis (4) and cell proliferation (5) and upregulates mitochondrial biogenesis. (6) CO has 200 times higher affinity for haemoglobin than oxygen and forms COHb, which decreases oxygen transport at a high level. It also causes a leftward shift of the oxyhaemoglobin dis-

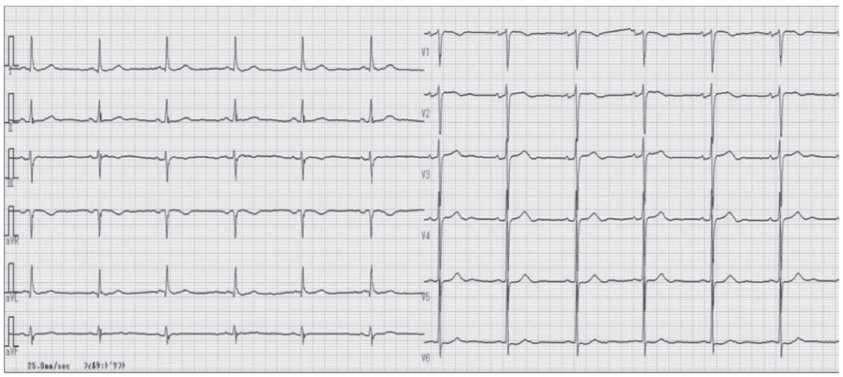

Figure 1. ECG result on admission showed normal sinus rhythm

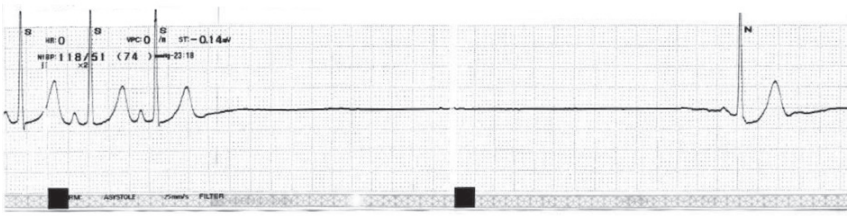

Figure 2. On day 14, sinus arrest occurred for 5 seconds.

sociation curve and promotes oxygen deficiency. In addition, CO binds to cytochrome c oxidase and interrupts cellular respiration, resulting in the production of reactive species, which leads to necrosis and apoptosis. Apart from that, inflammatory mechanisms have been discussed. These mechanisms, including unidentified ones, damage nearly all the organs and tissues, particularly in the central nervous and cardiovascular systems. (1) The cardiac conduction system is part of the nervous system; the mechanism of injury to this system may be similar to that of late-onset injury to the central nervous system. Considering the similarity between these mechanisms, neuropathy and arrhythmia should be correlated. However, the reason underlying the development of only SSS and no brain injury in our case remains unclear. Neurological injuries have not yet been elucidated, and there is also a report that COHb values correlate poorly with neurologic injuries. The pathogenesis and risk factors for delayed neurologic injuries remain unknown. (7)

Myocardial ischaemia and injury may occur in the acute phase, and ECG and cardiac enzyme tests are recommended. (1,8) In a study on patients with moderate to severe CO poisoning, 37% had elevated cardiac enzymes and/or diagnostic ECG changes. (9) However, there were no abnormalities in our case. A cohort study demonstrated a possible correlation between CO poisoning and the risk of developing arrhythmia on long-term observation. Arrhythmia was the most common cause of death after the acute phase, with paroxysmal tachycardia as the most frequent (24%), followed by ventricular fibrillation or flutter (15%) and paroxysmal supraventricular tachycardia (7%). (10)

HBO is an effective therapy for CO poisoning. (1) However, during HBO, electronic devices, such as monitors, cannot be used because of the risk of fire. In the event of lethal arrhythmias during HBO administration, because the pressure inside the capsule is high, we cannot immediately attend to the patient; this lag in treatment due to waiting for the pressure to decrease may lead to patient's death in some cases. The patient's cardiac function should be checked before administering HBO.

This case showed the possibility of late-onset lethal arrhythmia in patients with CO poisoning. Reports of the long-term outcome in patients with CO poisoning are limited, but CO poisoning is associated with increased mortality and arrhythmia. (10) Thus, follow-up observation is necessary.

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