Original Article

Carbon Monoxide Poisoning: A Five-year Review at Tan Tock Seng Hospital, Singapore

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Abstract

Introduction: Carbon monoxide poisoning (COP) is one of the leading causes of death from poisoning worldwide. There is no published study of COP in Singapore so far. Materials and Methods: All patients admitted with the diagnosis of COP to Tan Tock Seng Hospital (TTSH) over 5 years from 1999 to 2003 were retrospectively reviewed. The diagnosis was based on a history of potential exposure to carbon monoxide (CO) and elevated levels of carboxyhaemoglobin (COHb). The causes, demographic data, clinical presentations, management and complications were analysed. Results: There were 12 patients with COP. Their average age was 38.9 (± 11.8) years, with a male-to-female ratio of 3:1. Accidental COP (58.3%) was more common than intentional COP (41.7%). The most common cause of accidental COP was smoke inhalation from a faulty vehicle. Gas stove was the most preferred source for intentional poisoning. Presenting features were headache (83.3%), confusion (83.3%), coma (12.7%) and agitation (8.3%). The mean COHb level on admission was 35.9% (± 13.6). All were treated with 100% oxygen. All the patients achieved normal levels of COHb within 24 hours of admission. Two (16.7%) required intubation for airway protection as they were comatose on arrival, of which 1 presented with very high level of COHb (48.1%) and was the only patient to be treated with hyperbaric oxygen. Acute complications were globus pallidus infarction (16.6%), acute respiratory distress syndrome (8.3%) and myocardial ischaemia (8.3%). Most of the patients (91.7%) were discharged well from the hospital. One patient developed parkinsonism after a follow-up of 2 years. There were no deaths. Conclusion: COP is relatively uncommon in Singapore. It has a low rate of short- and long-term complications.

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Key words: Carboxyhaemoglobin, Complications, Gas stove, Oxygen, Vehicle

Introduction

Carbon monoxide (CO) is an odourless, tasteless and toxic gas and is the most abundant pollutant in the lower atmosphere. It is produced largely in industrial processes, internal combustion engines, malfunctioning home appliances and as a result of the incomplete combustion of wood and tobacco products.

Carbon monoxide poisoning (COP) was first described by Claude Bernard in 1865. It could result from either accidental or intentional causes. However, the number of intentional COP cases is about 10 times higher than accidental ones.[1] The incidence of accidental COP appears to be declining due to the implementation of improved preventive measures for transport and heating devices.[2] Accidental COP is seen most commonly during the winter months in countries with cold climates, when heating systems are used and windows are kept closed.[2] Smoke inhalation from the motor vehicle exhaust is the most common cause of accidental COP death.[1,2] In addition, inhalation of methylene chloride (a component of paint strippers) can also cause COP as it is metabolised in the liver to form a clinically significant amount of CO.[1,3]

To date, there is no published study of COP in Singapore. We, therefore, carried out a retrospective review of patients admitted to Tan Tock Seng Hospital (TTSH), Singapore with diagnosis of COP over a period of 5 years, from 1999 to 2003.

Materials and Methods

All patients discharged with the diagnosis of COP from TTSH from 1999 to 2003 were identified by the medical record office of the hospital. We carried out a retrospective

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analysis of causes, demographic data, clinical presentations, management and complications of COP. The diagnoses were based on a history of exposure to sources of CO and elevated levels of carboxyhaemoglobin (COHb).

Results

There were only 12 patients admitted with the diagnosis of COP during the study period. Their average age (± SD) was 38.9 (± 11.8) years, with an age range of 21 to 56 years. The male-to-female ratio was 3:1. There were 10 ethnic Chinese, and 1 ethnic Indian and 1 ethnic Malay.

Accidental COP (58.3%) was more common than intentional COP (41.7%). The most common cause of accidental COP was smoke inhalation from a faulty vehicle (n = 4), while the gas stove (n = 5) was the most preferred source for intentional poisoning. Accidents from house fire (n = 3), which included one incident each of television fire, barbecue fire and residential fire, accounted for the remaining cases of COP (Table 1).

All patients were admitted through the emergency medicine department (EMD) of TTSH. Presenting features were headache (83.3%), confusion (83.3%), dizziness (58.3%), coma (12.7%) and agitation (8.3%) (Fig. 1). Monitoring of vital signs at EMD showed that all the patients had pulse rate, blood pressure and respiration rate within normal range. Arterial blood gas analyses revealed 5 patients with mild metabolic acidosis [pH 7.28 to 7.34; standard bicarbonate (SBC), 17 to 19 mmol/L; pCO_2, 35 to 38 mm Hg]. The mean (SD) COHb level on admission was 35.9% (± 13.6).

All the patients in our study were treated with 100% oxygen (O_2). Two were intubated at EMD for airway protection, as they were comatose on arrival. The first patient was extubated after 10 hours of ventilation, while the second patient was weaned off ventilatory support only after 72 hours. The latter also presented with the highest level of COHb (48.1%) in our study and was the only patient who was treated with hyperbaric oxygen. All the patients achieved normal levels of COHb within 24 hours of admission.

Acute complications were globus pallidus infarction (16.6%), acute respiratory distress syndrome (ARDS) (8.3%) and myocardial ischaemia (8.3%). The diagnosis of globus pallidus infarction was based on computed tomography (CT), which was performed for 2 patients who were comatose on presentation. ARDS in 1 patient, as evidenced by the findings of bilateral pulmonary infiltrates and PaO_2/FiO_2 <200 mm Hg,[4], was thought to be due to either smoke inhalation or aspiration. Myocardial ischaemia was confirmed by new onset T wave inversion in anterolateral leads on routine electrocardiogram, transient elevation of cardiac enzymes and reversible wall motion abnormality on echocardiography.

The median (range) length of stay in the general ward was 2 (1 to 7) days and the high dependency unit was 1 (1 to 3) day. The total hospital stay was 3.5 (3 to 8) days. The 2 patients who were intubated also stayed in the intensive care unit, 1 for around 12 hours and the other for 3 days. All the patients were evaluated by psychiatrists during the course of hospitalisation. While 2 of them were found to have a history of depression, 1 was diagnosed to have new onset depression.

Most of the patients (91.7%) were discharged well from the hospital. Only 1 patient had on discharge symptoms of generalised slowness, lethargy and low mood. There were no deaths recorded in our study. All the patients were asked to return for review after 2 months at the specialist outpatient clinics. However, only 4 of them turned up. The defaulters were contacted via telephone and found to be healthy with no complications after 2 months of discharge. Patients were informed of the possible long-term complications of COP and they were advised to seek medical attention if such symptoms developed. Of the 4 patients who turned up for review, 1 continued to have slowly worsening slowness and lethargy and was diagnosed to have developed parkinsonism after a follow-up of 2 years in a neurology clinic. The other 3, on follow-up with a psychiatrist's clinic for treatment of depression, did not develop any long-term complications after completing 6 months of follow-up.

Table 1. Causes of Carbon Monoxide Poisoning

Our study
• Accident from vehicular fumes (n = 4)
• Accident from house fire (n = 3), e.g. television fire, barbecue fire, residential fire
• Intentional poisoning from gas stove (n = 5)
Global
• Poorly functioning heating systems
• Improperly vented fuel-burning devices like charcoal grills and gas cooking stoves
• Motor vehicles operating in poorly ventilated areas like parking garages and ice rinks
• Inhalation of methylene chloride

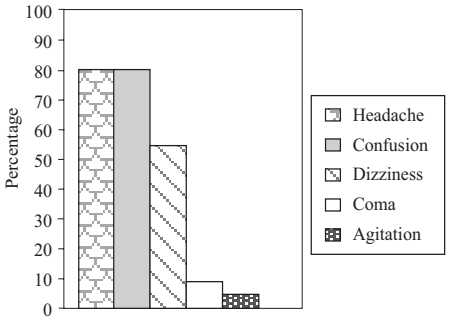

Fig. 1. Presenting features of carbon monoxide poisoning.

Discussion

TTSH is the second largest acute care hospital in Singapore. A total of 584,480 patients, averaging more than 100,000 patients per year, were seen in the EMD of the hospital from 1999 to 2003.[5] The total number of patients admitted to hospital over the same period was 234,540, averaging just under 50,000 admissions per year (Table 2).[5] Despite this huge turnover of patients, we found that only 12 (0.005%) cases of COP were admitted to the hospital.

We did not find any age predilection for COP. The youngest person who had COP was 21 years old and the oldest was 56 years of age. Our study showed that COP was 3 times more common in male patients. Male patients accounted for the majority of cases of intentional causes (88%) while accidental poisoning was equally common in both sexes. We also found that the risk of accidental COP was highest in car drivers, while no particular occupational group was prone to intentional COP poisoning. The racial distribution corresponded to the ethnic composition of Singapore,[6] with Chinese accounting for the majority of cases, followed by Malays and Indians.

In our study, the most common symptoms seen were headache, confusion and dizziness. COP is known to have very variable presenting features.[1] The most common misdiagnosis is a "flu-like" syndrome.[7] Notwithstanding the variability and non-specificity in clinical presentations, the severity of observed symptoms correlates roughly with the observed level of COHb.[7,8] Usually, there are no symptoms with COHb levels below 10%. Headache, nausea, dizziness and confusion may develop with levels of above 20%. Finally, with levels more than 40%, the patient may develop coma and seizures, and death may ensue when the levels go beyond 60%.[7-9] However, it is worth mentioning that there is evidence that such a fixed relation between COHb levels and clinical features does not always occur.[10] CO poisons cytochromes in the mitochondria leading to tissue hypoxia. By treating COP with oxygen therapy, PaO_2 in the intravascular compartment increases earlier than the tissue PaO_2. Consequently, tissue hypoxia is not reversed immediately. Therefore, although COHb levels in blood normalised, the tissues have not yet cleared the CO and can still sustain hypoxic injury. In our study, although the 2 patients who developed complications had relatively high COHb levels (30.8% and 43%), the 1 with the highest level of COHb (48.1%) developed no complications.[11]

Table 2. Annual Patient Load at Tan Tock Seng Hospital[5]

	1999	2000	2001	2002	2003*
Attendance at EMD	107,173	116,125	124,951	131,127	105,104
Admissions to hospital	46,215	49,356	50,050	51,484	37,435

EMD: emergency medicine department

* The lower number of admissions in the year 2003 was a result of the severe acute respiratory syndrome (SARS) outbreak in Singapore from March 2003 to May 2003. TTSH was the designated national SARS hospital for the isolation and management of SARS patients.

After apparent recovery from acute effects of COP, persistent or delayed neurological effects have also been reported. A syndrome of delayed neurologic sequelae known as delayed neuropsychiatric syndrome (DNS) has been reported in up to 40% of patients with significant poisoning.[1,8] It is characterised by variable degrees of cognitive deficits, personality changes, movement disorders and focal neurologic deficits. It usually occurs within 20 days of COP but may occur anytime from 3 to 240 days after COP, and the resulting deficits may last for a year or longer. The majority of DNS cases are associated with unconsciousness and advanced age, although the syndrome correlates poorly with COHb levels.[1,11,12] The incidence of DNS tends to increase with length of unconsciousness but it may occur even when the mental state is clear during the acute episode.[13]

One should have a high index of suspicion when making a diagnosis of COP. The diagnosis is based on a compatible history and elevated COHb levels by co-oximeter. A history of potential exposure is the most reliable indicator of COP.[9] Pulse oximetry does not differentiate COHb from oxyhaemoglobin and therefore cannot screen for CO exposure.[1,14] Venous sample may be used to measure COHb levels as arterial and venous COHb levels show good correlation.[15] However, it must be borne in mind that COHb levels may be low or even undetectable because of the time elapsed between exposure and drawing of blood sample for determination of COHb or because of the initial oxygen therapy.[7,9,16] In cases of residential fire, the patient should be evaluated for burns, the presence of other gas inhalation and concomitant cyanide toxicity.[8,9,16] Lactate has been used as a marker for severe poisoning.[7,16] In all cases of severe poisoning, an electrocardiogram and, if needed, serial cardiac enzymes, to rule out myocardial ischaemia and cardiac dysrhythmias, should also be obtained. Chest radiograph is required to look for any evidence of non-cardiogenic pulmonary oedema. Patients are also recommended to undergo psychometric testing to screen for cognitive dysfunction.[1,9] CT or magnetic resonance imaging may show characteristic low density lesions in the globus pallidus as well as concomitant white matter lesions.[1,17]

The mainstay of treatment of COP includes supplemental O_2, airway management and the stabilisation of cardiovascular status. Administration of 100% O_2 has been widely accepted as the clinical standard of the treatment of COP.[1,8-10] However, it is debatable whether this O_2 should be given under ambient pressure [normobaric O_2 (NBO)] or under increased pressure [hyperbaric O_2 therapy

(HBOT)]. The half-life of CO while breathing air is around 320 minutes; while on 100% O_2 it is 80 minutes and at 3 atmospheres absolute (ATA) it is reduced to 23 minutes.[1,7] HBOT consists of the administration of 100% O_2 within a pressurised chamber resulting in a very significant increase of dissolved O_2 in the body. The various benefits of HBOT include rapid elimination of COHb, maintenance of adequate cerebral O_2 delivery, elimination of CO from tissue haem proteins, decrease in cerebral oedema, leukocyte adherence and oxidative stress.[11] There have been 6 randomised controlled trials comparing HBOT with NBO in the treatment of COP.[16,18-20] While 4 of these trials showed benefit for HBOT, the other 2 failed to do so. The main criticism of the latter 2 studies included poor study designs, inadequate oxygen doses and weak outcome measures.[11]

The reasons for consideration of HBOT in our patient were multiple risk factors for bad outcome, i.e., deep coma (Glasgow Coma Scale 5) on arrival at the EMD, a very high level of COHb, and CT findings of bilateral globus pallidus infarction.

The Undersea and Hyperbaric Medical Society recommends HBOT for all patients with clinical evidence of severe COP regardless of COHb levels, such as those with history of loss of consciousness, neurological signs, cardiovascular dysfunction and severe metabolic acidosis.[9] Despite some controversies, HBOT is recommended for pregnant patients with COP as it has potential benefits for the mother and the fetus.[21] The most common harmful effect of HBOT is painful barotrauma affecting the ears and sinuses. Less common risks are O_2 toxicity, seizures, pulmonary oedema, decompression sickness, pneumothorax and fire hazards.[16] It is also not suitable for claustrophobics.

COP has very variable prognoses. Death rates in severely intoxicated patients may be as high as 30%.[9] As many as 40% of patients may be left with persistent neurocognitive deficits.[1,12] Elderly patients, those with history of unconsciousness, or cardiovascular disease, metabolic acidosis and structural defects on neuroimaging have poorer prognoses.[9]

Conclusion

COP has diverse clinical features and hence its diagnosis is often missed. The diagnosis primarily rests on suggestive history and elevated COHb levels. 100% O_2 given under ambient pressure remains the most accepted first-line treatment. The role of HBOT is still controversial, although there is evidence that it may be beneficial in severely intoxicated patients. COP continues to be a global health problem with significant morbidity and mortality. There has been no nationwide study of COP in Singapore. Our study, conducted at the second largest acute care hospital in Singapore, may suggest that COP is uncommon in Singapore. This may be due to relatively low vehicular pollution and the non-use of heating devices.

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