Carbon monoxide poisoning: Same time, same place but different outcomes

Phee-Kheng Cheah, Fatin Salwani Zaharuddin, Nik Hisamuddin Rahman, Muhamad Yaakub Arifin, Mohd Hakimi Abdullah

ABSTRACT

Introduction: There have been numerous reports documenting the differences in outcomes after carbon monoxide poisoning between men and women. There are also several reports mentioning the differences in presentation and outcome of poisoning in adults and children.

Case Series: We report two construction workers who had carbon monoxide poisoning while watching television. They used a petrol generator to power their television during a power outage, and the generator was placed in the same confined room. All windows were closed due to heavy rain and strong winds outside. Both patients were found motionless the next morning in front of the television. The two patients aged 16 and 22 years old presented to emergency department with almost similar complaints but had a very differing hospital course. The 16-year-old had status epilepticus needing intubation and ICU admission while the other was well throughout his stay. Both patients underwent two courses of hyperbaric oxygen therapy using the Royal Malaysian Navy treatment table 18-60-30. The 16-year-old was placed in the multi-place hyperbaric chamber while still intubated. Both patients were discharged without any neurological deficits.

Conclusion: The clinical course of patients with carbon monoxide poisoning may differ due to factors other than gas concentration and duration of exposure.

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Introduction: There have been numerous reports documenting the differences in outcomes after carbon monoxide poisoning between men and women. There are also several reports mentioning the differences in presentation and outcome of poisoning in adults and children. **Case Series:** We report two construction workers who had carbon monoxide poisoning while watching television. They used a petrol generator to power their television during a power outage, and the generator was placed in the same confined room. All windows were closed due to heavy rain and strong winds outside. Both patients were found motionless the next morning in front of the television. The two patients aged 16 and 22 years old presented to emergency department with almost similar complaints but had a very differing hospital course. The 16-year-old had status epilepticus needing intubation and ICU

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Keywords: Carbon Monoxide Poisoning, Hyperbaric Oxygen Therapy, Status Epilepticus

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INTRODUCTION

Carbon monoxide poisoning has been dubbed as a silent killer as its presence goes undetected by human senses. It is a colorless, odorless, tasteless and a non-irritant gas. It produces variable extremes of symptoms and complications. Patients may be totally asymptomatic or present with only mild symptoms such as dizziness, headache and flu-like symptoms to extremes of severe cardiopulmonary failure, nervous system dysfunction and death [1].

Severity of carbon monoxide poisoning is related to the amount of carbon monoxide inspired [2]. However, patients exposed to the same amount of carbon monoxide may have different clinical manifestations. Few factors

have been studied as to what determines the severity of poisoning and the parameters contributing to these differences. The relation of these factors to the severity of poisoning is yet to be clearly proven although some explanations had been offered. We report two young healthy adult males, accidentally exposed to carbon monoxide at the same location with equal amounts of exposure. Surprisingly, they developed neurological complications of differing severity and had a very different clinical course in hospital. Both patients were young and well with no preexisting medical conditions that could have explained the differences.

Principles in managing carbon monoxide poisoning involves dissociating the carbon monoxide bond from hemoglobin which is 230 times stronger to split compared to the oxygen-hemoglobin bond. Hyperbaric oxygen therapy (HBOT) has been long recognized as the best modality to deliver super-saturated oxygen to hypoxic tissues and disable the carbon monoxide-hemoglobin bond [3].

CASE SERIES

Two construction workers, aged 16 years and 22 years, were found unconscious by their relatives. Both had been sleeping in a confined room with a petrol generator placed inside the room. The generator was running from 10.00 pm until the next morning due to a power failure. They presumably fell asleep or were rendered unconscious by the carbon monoxide while watching television, which was powered by the generator. All windows were closed, as it was raining heavily with strong winds. Both patients were previously well with no medical illness and neither had exhibited any features of depression, suicidal or parasuicidal tendencies. Neither was known to have abused illicit drugs. Both patients were brought immediately to the emergency department (ED) by their relatives who found them.

Patient A is a 16-year-old boy had Glasgow Coma Scale (GCS) of 10/15 (E3V2M5) on arrival to ED. His blood pressure was 120/71 mmHg and pulse rate 126 bpm. Initial oxygen saturation was 90%, rising to 100% after oxygen was given via non-rebreather mask. Cardiovascular and respiratory examinations were normal. Neurological examination revealed upper motor neuron lesion findings with no signs of meningism. Otoloscopic examination was unremarkable. Blood investigations showed mild leukocytosis with normal level of hemoglobin, platelet, glucose and electrolytes. Creatinine kinase was elevated; 2590 mmol/L. Serum lactate level was 4 mmol/L. Initial blood gas on oxygen showed pH 7.25, pO_2 90 mmHg, pCO_2 33 mmHg and bicarbonate 13.2 mmol/L. Serial electrocardiogram showed sinus tachycardia. Chest film and computed tomography (CT) scan of brain revealed normal findings. After other possible causes of altered mental status had been ruled out, a diagnosis of carbon monoxide poisoning

with neurological complication was made. In casualty, this patient developed status epilepticus requiring emergency intubation. The patient needed inotropic support post intubation. The post intubation chest X-ray showed haziness in the right lower zone, hence patient was also treated for aspiration pneumonia. The patient underwent two sessions of hyperbaric oxygen therapy (HBOT) using the Royal Malaysian Navy treatment table 18-60-30 in the multi-place hyperbaric chamber while still intubated. No complications were observed during the session. Subsequently, he was monitored in the Intensive Care Unit. On day-3, inotropic support could be weaned off. He was extubated and regained full GCS with normal neurological assessment on day-5. He was discharged on day-8.

Patient B is a 22-year-old male also arrived with GCS of 10/15 (E3V2M5). Oxygen saturation was 86–88% on room air, which improved to 100% with oxygen via a non-rebreather mask. His blood pressure was 115/62 mmHg and pulse rate 105 beats/min. Cardiovascular and respiratory examination were normal. His neurological assessment also indicated an upper motor neuron lesion. No evidence of meningism was found. Blood investigations showed mild leukocytosis with otherwise normal hemoglobin, platelet, glucose, electrolytes and creatinine levels. Similar to patient A, there was an elevated level of creatinine kinase with a reading of 2162 mmol/L. Serum lactate was 3 mmol/L. Initial blood gas shows, pH 7.30, pO_2 105 mmHg, pCO_2 27 mmHg and bicarbonate 16.1 mmol/L. Serial measurements of electrocardiogram did not show any ischemic changes and chest radiograph was normal. Computed tomography scan of brain was also normal. This patient developed generalized tonic-clonic seizure in casualty but aborted spontaneously after 2 minutes. After the seizure, he continued breathing spontaneously without needing any ventilatory support as compared to patient A. The patient underwent two sessions of HBOT using the Royal Navy treatment table 18-60-30. Reassessment after the first session of HBOT showed full recovery of GCS with normal neurological assessment. He was discharged on day-2 after admission.

Subsequent follow-up at day-30 via phone revealed that both patients returned to normal daily activities with no residual neurological deficits.

DISCUSSION

Methods of defining severity of carbon monoxide poisoning are still being debated. It is generally agreed that it depends on the inspired concentration of carbon monoxide, the length of exposure towards the poisonous gas, and the general health status of the individual being exposed [2]. From the history, we can safely conclude that both patients were presumably exposed to carbon monoxide for an equal amount of time as they were in the same confined room. Therefore, we can also postulate that both had inspired an equal amount of carbon

monoxide during that duration. Both are young, fit, male construction workers with no known medical illnesses.

Despite those similarities and the presumed same carbon monoxide exposure, they developed neurological complications with different extremes of severity. Age is one factor that influences outcome. It has been reported that children are more susceptible to high level of toxicity as they have a higher metabolic rate as compared to adults [4]. Although one of our patients were in the pediatric age group, they had an age difference of only 6 years. They showed a marked difference in their disease process with patient A needing intubation and ICU care. However, their final neurological outcome was similar. Did the difference in age become the deciding factor in the divergent outcome in these two cases?

Impaired consciousness level has been related to severity of carbon monoxide poisoning. Grieb et al. concluded in their study that there was an inverse correlation between initial GCS and severity of carbon monoxide toxicity [4]. Both patients presented with initial GCS of 10/15 but their clinical courses differ in severity. Elevated leucocyte counts and C-reactive proteins were found to be associated with severe carbon monoxide toxicity [5]. Both patients had elevation of white cell counts in their blood investigations, reflecting only severity of their carbon monoxide level but not the difference in clinical outcome. Lactate elevation results from anaerobic glycolysis due to inadequate oxygen supply. Reports concluded that blood lactate level might also be a useful indicator in predicting severity of carbon monoxide poisoning [6]. Both patients had a raised level of lactate with metabolic acidosis.

Measuring carboxyhemoglobin level is not yet available in our current setting. Many reports however have concluded that levels of carboxyhemoglobin have little or variable correlation with symptoms or severity of carbon monoxide poisoning [5]. A recent study only recorded 61.9% positive toxicology screening results in intubated carbon monoxide poisoned patients [7]. In a setting like ours which carboxyhemoglobin levels may not be readily available, we suggest a high index of suspicion for this common form of poisoning. Clinical history and physical examination is of utmost importance in making this crucial diagnosis. Certain laboratory parameters such as respiratory alkalosis are suggestive and should be able to increase suspicion of carbon monoxide poisoning [8].

Hyperbaric oxygen therapy remains the gold standard for the treatment of carbon monoxide poisoning. In the military hospital in Malaysia the treatment table used is the Royal Malaysian Navy Treatment Table 18-60-30 whereby the patient is pressurized to an atmospheric pressure equivalent to 18 m depth in a hyperbaric chamber within a few minutes. Once the depth of 18 m is reached, a cycle of 25 minutes of 100% oxygen and 5 minutes of air-break will be delivered and repeated once before the patient is surfaced from 18 m to 0 m while breathing 100% oxygen. The air-break in between the oxygen delivery is to prevent the effect of oxygen toxicity.

Although many case reports has documented the differences in carbon monoxide toxicity in mass group poisoning, the factors identified could not explain the difference in the clinical course of our patients. From our patients, we agree that impaired consciousness, elevated leucocyte and high lactate levels reflect the severity of carbon monoxide toxicity and may predict the final neurological outcome. However, questions still remain as to why the clinical courses are different and how these factors correlate with each other to produce a different outcome. Perhaps further understanding of this differing clinical course will shed more light on measuring the severity in carbon monoxide poisoning.

CONCLUSION

The clinical course of patients with carbon monoxide poisoning may differ due to factors other than gas concentration and duration of exposure. Management of these patients especially when there are multiple victims involving pediatric age group should be tailored to individual presentation and clinical course.

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Author Contributions

Phee-Kheng Cheah – Conception and design, Drafting the article, critical revision of the article, Final approval of the version to be published

Fatin Salwani Zaharuddin – Acquisition of data, Drafting the article, Final approval of the version to be published

Nik Hisamuddin Rahman – Conception and design, Critical revision of the article, Final approval of the version to be published

Muhamad Yaakub Arifin – Acquisition of data, Drafting the article, Final approval of the version to be published

Mohd Hakimi Abdullah – Acquisition of data, Critical revision of the article, Final approval of the version to be published

Guarantor

The corresponding author is the guarantor of submission.

Conflict of Interest

Authors declare no conflict of interest.

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