CASE REPORT

Normal carboxyhaemoglobin level in carbon monoxide poisoning treated with hyperbaric oxygen therapy

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SUMMARY

Throughout the world both intentional and inadvertent exposure to carbon monoxide (CO) remains an important public health issue. While CO poisoning can be lethal, the morbidity is predominantly due to nervous system injury. A previously healthy 22-year-old woman was found unconscious at home by her sister. Her parents were found dead in the house with a recent history of a dysfunctional furnace. She was presumed to have CO poisoning despite an initial carboxyhaemoglobin level of 2.5%. Patient had both clinical and radiological evidence of neurological damage. However, with multiple sessions of hyperbaric oxygen (HBO) therapy she recovered to a near normal functional status. There is no consensus that exists among treating physicians about the role of hyperbaric oxygen in management of neurological injury. The case described here has significant neurological damage related to CO exposure but improved after HBO therapy.

BACKGROUND

Carbon monoxide (CO) poisoning is responsible for approximately 50 deaths per year in the UK, with the vast majority of cases remaining undiagnosed.¹ Diagnostic criteria for CO poisoning include a history and symptoms suggestive for exposure and an elevated venous carboxyhaemoglobin (COHb) level. The symptoms that may be present are dizziness, nausea, vomiting, confusion, fatigue, chest pain, dyspnoea and loss of consciousness.² Clinical symptoms and signs vary widely depending on the level of exposure and underlying comorbidities. Non-smokers that show a COHb greater than 3% have a confirmed diagnosis of CO poisoning, while smokers do not because carbon monoxide accumulates in tobacco smoke and elevates your baseline HbCO level.³ Due to the elimination half-life of CO, it is possible that patients with CO poisoning may have normal carboxyhaemoglobin levels. Treatment for CO poisoning includes prompt removal from source of exposure, 100% oxygen either by mask or endotracheal tube and if available hyperbaric oxygen (HBO) therapy. The use of HBO therapy in CO poisoning is not universally agreed on, but has been considered in the following clinical scenarios: new neurological deficit or mental status change, evidence of end-organ damage, pregnant woman with a COHb greater than 20%, and any patient with a COHb greater than 25%.⁴ We present the

case of a patient with CO poisoning, who presented with a normal COHb level and who had dramatic neurological improvement with hyperbaric oxygen therapy.

CASE PRESENTATION

A previously healthy 22-year-old woman was found unconscious and covered with vomitus at her home by her younger sister. She was last seen well 2 days ago. Emergency medical services (EMS) also found her parents, who were dead at the scene, along with the temperature of the home being 4°C. EMS detected no carbon monoxide on a portable metre. The patient had an initial Glasgow Coma score of 4, was given oxygen by a non-rebreather mask and transferred to a local hospital. The sister reported that their father was having difficulty with the furnace and had attempted to repair it the previous night. At the local emergency department, she was intubated and the initial evaluation revealed sinus tachycardia, pH 7.29, normal serum lactate, glucose, electrolytes and urinalysis. A blood alcohol and drug screen were both negative. A non-contrast CT of the head showed no abnormality. Her initial COHb level was found to be 2.5%, which was taken at the time of intubation. She was presumed to have CO poisoning, despite the normal COHb level, and was airlifted to the nearest centre for HBO therapy. Neurological examination prior to the start of HBO therapy revealed an obtunded female with equal and reactive pupils at 2 mm, diffusely rigid extremities, greater in the right upper and lower extremity than the left, hyper-reflexia in right upper and lower extremities, sustained clonus of right foot, down-going toes bilaterally and withdrawal to pain in all four extremities. She received three 2 hour sessions of HBO within the first 24 hours of arrival. After the first session, her mental status improved and she was extubated. Physical examination showed aphasia with normal cranial nerves, asymmetric weakness improved from initial examination and prominent right ankle clonus. MRI of the brain revealed multiple areas of restricted diffusion, hyperintensities in T2-weighted images and hypointensities in T1-weighted images in bilateral cortices and subcortical white matter, as well as areas posterior to the globus pallidus, consistent with cortical ischaemic infarctions (figures 1 and 2).

OUTCOME AND FOLLOW-UP

The patient received a total of six HBO sessions with noticeable improvement in her neurological

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Figure 1 MRI of the brain from a patient with carbon monoxide poisoning revealing areas of restricted diffusion consistent with ischaemia in the right hemisphere superiorly, within the right centrum semiovale and central white matter in the axial view of a diffusion weighted image.

examination after each session. On discharge, she had normal cognition with only a mild left-sided hemiparesis and she could walk by herself. A couple of weeks after her hospital admission, the autopsy results of her parents revealed severe CO poisoning with levels of 49% and 35% in her mother and father, respectively.

DISCUSSION

This case highlights the therapeutic benefit of HBO therapy in suspected CO poisoning, even when initial CO levels are within normal limits. The toxic CO gas is colourless, odourless, a non-irritant and easily absorbed through the lungs. The most common sources of poisoning in humans includes exposure to motor vehicle exhaust, poorly functioning heating system (like in the current case) and inhaled smoke. The pathophysiology of CO poisoning-related organ injury is incompletely understood. Because of its higher affinity for haemoglobin, CO hampers the body's ability to carry and deliver oxygen. CO also interferes with the cellular oxidative phosphorylation process by binding to proteins and cytochromes.⁴ The combination of tissue hypoxia, ischaemia reperfusion injury and the subsequent inflammatory cascade is primarily responsible for the end-organ damage.

Clinical manifestations of CO poisoning varies from subtle non-specific symptoms, like a headache, to devastating multi-organ dysfunction, to death. Neurocognitive sequelae, persistent and delayed, are the most feared complication related to CO exposure. Up to 40% of patients can have neurological injury,

Figure 2 MRI of the brain from a patient with carbon monoxide poisoning showing the apparent diffusion coefficient sequence revealing areas of hypointensity in the right hemisphere superiorly within the central white matter consistent with ischaemia.

as manifested in our patient, with focal neurological deficits, personality changes and cognitive abnormality.⁵

History is typically sufficient to make the diagnosis of CO poisoning. There are no pathognomonic symptoms and signs specific to CO poisoning and an elevated COHb level in the blood is diagnostic, but not essential. A normal level of COHb in non-smokers is <3% and in smokers is <15%. Severity of poisoning depends on duration of exposure, concentration of ambient CO and underlying health status of the patient. COHb level does not correlate with the severity, acuity or resolution of neurological signs and symptoms.

The standard approach of treatment includes removal from the source of the gas, general supportive measures and administration of 100% oxygen to hasten the elimination from body. The medical community is sceptical about the role of HBO therapy in CO poisoning. This scepticism stems from biased, conflicted and weak evidence from the published data regarding the therapy. The half-life of CO is approximately 4 to 5 hours while breathing room air. Thus, patients with toxic ranges of CO can have normal COHb levels after three or more half-lives. HBO has been shown to: (1) decrease the half-life of CO to approximately 30 min and (2) decrease oxidative stress, inflammation and preserve tissue ATP.⁶ Because of this effect, HBO can improve delayed neurological sequelae, even with a normal COHb level. Limitations of HBO therapy include cost, availability, risk of transportation in unstable patients and potential side effects, which include barotrauma, seizure and oxygen toxicity. Current recommendations suggest initiating HBO therapy in appropriate clinical scenarios even when COHb levels are not elevated.⁷

This case highlights the therapeutic benefit of HBO therapy in suspected CO poisoning, even when initial COHb levels are within normal limits and reinforces the idea that if your clinical suspicion is high enough, a negative lab test should not sway you

from treating the patient. While early HBO can contribute to a rapid recovery, well-designed multicentre randomised controlled trials supporting its use have not been published.^{8 9} Heterogeneity of the available scientific literature should be kept in mind while making management decisions for individual patients.

Learning points

- ▶ Carbon monoxide poisoning remains elusive with many cases going undiagnosed.
- ▶ Carboxyhaemoglobin is the only indicator of true poisoning but may be normal in some cases.
- ▶ Carboxyhaemoglobin level does not correlate with severity, acuity or resolution of neurological signs and symptoms.
- ▶ Current guidelines recommend initiating hyperbaric oxygen therapy in appropriate clinical scenarios, even when carboxyhaemoglobin level is not elevated.
- ▶ Hyperbaric oxygen therapy may benefit suspected carbon monoxide poisoning in patients with neurological symptoms despite low carboxyhaemoglobin level.

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REFERENCES

- 1 The Carbon Monoxide and Gas Safety Society. Statistics of Death and Injuries. <http://www.co-gassafety.co.uk/information/co-gas-safety-statistics-of-deaths-and-injuries/> (accessed 25 May 2017).
- 2 Kao LW, Nānagas KA. Carbon monoxide poisoning. *Emerg Med Clin North Am* 2004;22:985–1018.
- 3 Ernst A, Zibrak JD. Carbon Monoxide Poisoning. *N Engl J Med Overseas Ed* 1998;339:1603–8.
- 4 Hardy KR, Thom SR. Pathophysiology and treatment of carbon monoxide poisoning. *J Toxicol Clin Toxicol* 1994;32:613–29.
- 5 Choi IS. Delayed neurologic sequelae in carbon monoxide intoxication. *Arch Neurol* 1983;40:433–5.
- 6 Thom SR. Hyperbaric oxygen: its mechanisms and efficacy. *Plast Reconstr Surg* 2011;127(Suppl 1):131S–41.
- 7 Hampson NB, Dunford RG, Kramer CC, *et al*. Selection criteria utilized for hyperbaric oxygen treatment of carbon monoxide poisoning. *J Emerg Med* 1995;13:227–31.
- 8 Huang C-C, Ho C-H, Chen Y-C, *et al*. Hyperbaric oxygen therapy is associated with lower short- and long-term mortality in patients with carbon monoxide poisoning. *Chest* 2017.
- 9 Weaver LK, Hopkins RO, Chan KJ, *et al*. Hyperbaric oxygen for acute carbon monoxide poisoning. *N Engl J Med* 2002;347:1057–67.

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