REPORTE DE CASOS

Acute carbon monoxide poisoning with coexisting cerebral and myocardial infarction: The brain-heart connection and challenges of management in resource-limited settings

Intoxicación aguda por monóxido de carbono con infarto cerebral y de miocardio coexistente: la conexión cerebro-corazón y los desafíos del manejo en entornos con recursos limitados

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Abstract. Carbon monoxide poisoning pose significant public health consequence and management can be challenging in resource-poor countries. This is a commentary of a 40 year-old farmer with a five day history of loss of consciousness following overnight inhalation of carbon monoxide. Neuroimaging and electrophysiologic studies showed bilateral internal capsule and myocardial infarction. He had unsuccessful treatment with normobaric oxygen therapy in the absence of hyperbaric oxygen. This case depicts the brain-heart connection in carbon monoxide poisoning and highlights the challenges of management in a resource poor setting.

Keywords: Carbon monoxide; Poisoning; Acute; Infarction.

Resumen. La intoxicación por monóxido de carbono representa un significativo problema para la salud pública de cualquier país y su manejo puede resultar en un gran desafío en los países emergentes, con escasez de recursos para la atención de este tipo de pacientes. Se presenta el caso de un agricultor de 40 años de edad, con antecedentes de cinco días previos de pérdida de la conciencia, posterior a la inhalación de monóxido de carbono. Las evaluaciones realizadas: tomografía axial computada de cerebro y electrocardiograma, mostraron la presencia concomitante de infarto cerebral, con compromiso de ambas cápsulas internas e Infarto de miocardio, respectivamente. Se inició tratamiento con oxígeno normobárico, falleciendo el paciente dentro de las 24 horas posteriores a su admisión. Este caso representa la conexión en la afectación de corazón y cerebro, en intoxicaciones por monóxido de carbono y sirve para destacar los desafíos de su manejo en entornos con limitaciones de recursos.

Palabras clave: Monóxido de carbono; Envenenamiento; Accidente cerebrovascular; Infarto de miocardio

Introduction

Carbon monoxide (CO) poisoning is commonly under diagnosed, under treated and results in preventable morbidity and mortality in resource-limited settings. (Raub *et al*. 2000). The precise number of individuals who have suffered from CO intoxication is not known. The health effects associated with exposure to CO range from the more subtle cardiovascular and neurobehavioral effects at low concentrations to unconsciousness and death after acute or chronic exposure to higher concentrations of CO. The morbidity and mortality resulting from the latter exposures are described briefly to complete the picture of CO exposure in present-day society. The symptoms, signs and prognosis of acute CO poisoning correlate poorly with the level of carboxyhemoglobin (COHb) (Afolayan *et al*. 2014).

There are copious commentaries of isolated neurologic and cardiovascular complications of CO (Ernst and Zibrak 1998; Prockop 2007). Reports of coexisting cerebral and myocardial infarction are rare and have not been reported in our environment. We report a case of carbon monoxide poisoning with coexisting cerebral and myocardial infarction in a young healthy adult male without risk factors to illustrate the fatality and challenges of management in resource-poor settings.

Case description

A 40 year old farmer with a five-day history of loss of consciousness following overnight inhalation of exhaust vapour from an indoor power generator. He was last seen conscious twelve hours earlier. He was found unconscious with two other persons who became awake on exposure to standard oxygen therapy. He had two

episodes of non-projectile vomiting. There was no history of head injury, epilepsy, hypertension or diabetes mellitus. He had pyrexia of 39 degree Celsius, and was markedly dehydrated. Glasgow coma score was 4/15 – eye opening=2, verbal response=1, motor response=1. The pupils were equal, 3mm in diameter, and sluggish to light. There was global hypotonia and hyporeflexia with bilateral extensor plantar response. Cardiovascular examination is significant only for tachycardia (144 bpm), and blood pressure of 150/80 mmHg. The respiratory rate was 56 cpm with widespread crepitations.

An initial consideration of acute CO poisoning with cerebral and non-cardiogenic pulmonary o edema was entertained and subsequently commenced on normobaric oxygen, intravenous 20% mannitol, 0.9% saline, frusemide and broad-spectrum antibiotics.

Complete blood count showed haematocrit of 53.2% (haemoglobin=17.0 g/dl), neutrophillic leukocytosis of 12.5 X 10^3 cells/µl (granulocytes 92.3%, lymphocytes 4.6%, monocytes 3.1%), elevated mean corpuscular volume (MCV) 104 fl/red cell, and thrombocytopaenia of 71 X 103 cells/µl. Other red cell indices were normal. Blood chemistry revealed hypernatraemia (149 mmol/l), hypokalaemia (2.5 mmol/l) and acidosis (bicarbonate=15 mmol/l). Random blood glucose was 116 mg/dl and urinalysis showed urobilinogenuria. Brain computer tomography scan done six hours into admission revealed bilateral internal capsule haemorrhagic infarct (*figure 1*). Electrocardiography demonstrated

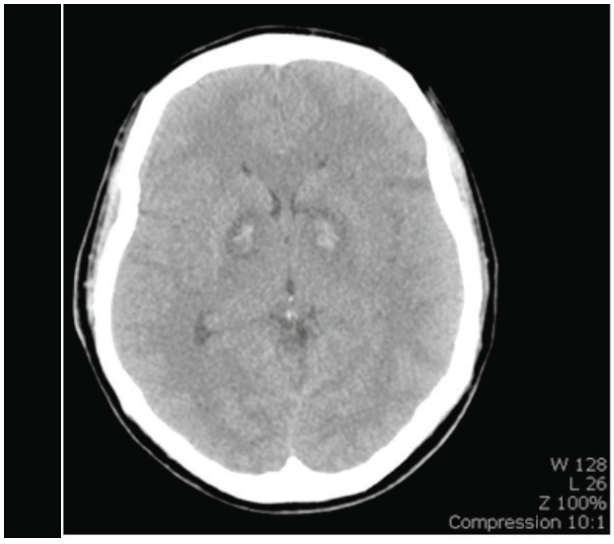

Figure 1. Computer tomography scan showing bilateral internal capsule haemorrhagic infarct.

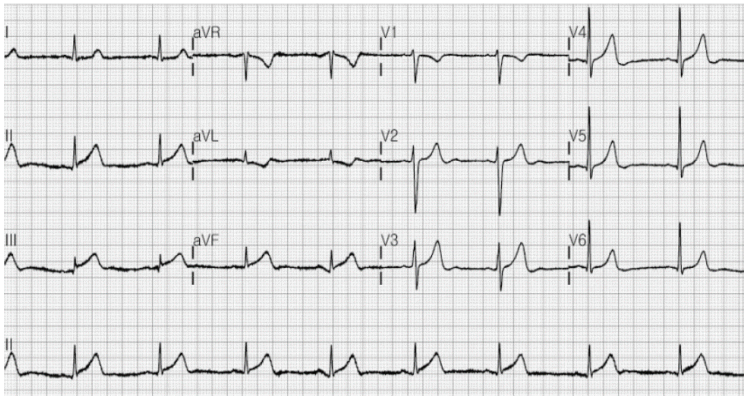

Figure 2. Electrocardiogram showing ST elevation in leads II, III, and aVF.

sinus tachycardia, left axis deviation and ST segment elevation in inferior leads (*figure 2*). The diagnosis was then modified to CO poisoning with coexisting cerebral and myocardial infarction. In spite of the subsequent introduction of aspirin, patient condition deteriorated rapidly and finally succumbed to illness on the second day of admission.

Discussion

The diagnosis of CO poisoning was based on historical and epidemiological factors in this case. Existing data suggest that unintentional poisoning from electrical generators is common in developing countries (Raub *et al*. 2000, Weaver 2009). Sequelae may be immediate or delayed occurring 2–21 days after exposure (Ernst and Zibrak 1998). The patient developed complications attributable to CO poisoning within five days of exposure. For example, vomiting and systolic hypertension may be explained by the cerebral oedema (Cushing's reflex), while the tachypnoea is possibly a compensatory response to acidosis from carbon dioxide retention. Tachycardia, pyrexia and pulmonary oedema could relate to the myocardial ischaemia and direct toxicity of CO. Haemoconcentration due to dehydration, may explain the observed hypernatremia and relative polycythemia. The hypokalaemia could not be readily explained but diuretic-induced loss could not be ruled out. Leukocytosis and fever without a septic focus excluded the consideration of sepsis. The low platelets may result from direct anti-platelet effect of CO.

Cerebral infarction is a rare and delayed presentation of CO poisoning and can occur in

subjects with or without risk factors (Finelli and Di Mario 2004). Conversely, the cardiovascular consequences of CO poisoning are frequent and limited to isolated case reports of electrocardiographic changes, myocardial dysfunction, and infarction (Chang *et al*. 1999). Evidence of the brain-heart connection is restricted to coexisting cerebral oedema and myocardial infarction (Choi 2001, Satran *et al*. 2005). This case is of interest because it describes the coexistence of bilateral internal capsule infarction and ST segment elevation myocardial infarction in the same patient. Available reports suggest that in CO poisoning, the basal ganglia, particularly the globus pallidus is the preferential site of infarction, and non ST elevation myocardial infarction is the commonest myocardial injury (Satran *et al*. 2005, Prockop 2007).

Injury in CO poisoning is mediated via a combination of direct CO toxicity, carboxyl haemoglobin-induced hypoxia, and inflammation (Goldbaum *et al*. 1976). CO binds 200 to 250 times more avidly to haemoglobin than oxygen, and decrease oxygen saturation in a dose-dependent fashion seen as a leftward shift in the oxygen dissociation curve. In addition, 10% - 15% of CO binds to myoglobin, cytochromes C and P450 leading to myocardial depression and impaired cellular respiration and hypoxia. CO directly induces mitochondrial dysfunction, endothelial homeostatic disturbance and tissue oedema. Hypoxia initiate a vicious cycle of free oxygen and nitric oxide radical formation sustained by lipid peroxidation and neutrophil activation which in turn leads to cellular apoptosis and necrosis. CO interaction with platelet heme protein, nitric oxide and myeloperoxidase leads to infarction and oxidative endothelial injury.

Successful management of CO poisoning requires measurement of carboxyl haemoglobin (COHb) levels and institution of hyperbaric oxygen therapy (HBOT) (Juurlink *et al*. 2000). The treatment of CO poisoning is anchored on the rapid restoration of oxygenation to bodily tissues. HBOT shortens the half-life of CO from 4-5 hours to 20 minutes, rapidly reverses CO's binding to haemoglobin and myoglobin, and directly provide oxygen to tissues independent of haemoglobin. COHb levels > 25% and presence of neurologic and/or cardiovascular symptoms are independent indications for HBOT (Ernst and Zibrak 1998, Weaver 2009). Clearly, our patient did not have the benefit of this treatment, which may have influenced the outcome. Other limitations of this report are the absence of cardiac enzyme assay, arterial blood gas analysis and cerebral/coronary angiography, which featured copiously in reports from other climes. Management of carbon monoxide poisoning is therefore challenging in resource-limited settings.

Conclusion

Coexisting cerebral and myocardial infarction in CO poisoning is rare but real and probably depends on the dose and duration of exposure. This case depicts the brain-heart connection in CO poisoning and highlights the challenges of management in resource-poor settings.

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