A near death event following a barbecue

Introduction

This article describes the case and sequelae of a young man accidently poisoned by carbon monoxide from a barbecue placed in his camper van. This seemingly innocent action, when the barbecue was apparently extinct and cool enough to pick up with his bare hands, resulted in him being discovered unconscious.

Recognized as the most common cause of fatal poisoning worldwide (Hopkins and Woon, 2006), many people remain unaware of the dangers of carbon monoxide.

Discussion

Death rates (England and Wales) from carbon monoxide poisoning continue to fall. A total of 1042 people died in 1995 which had fallen to 236 deaths in 2009 (Penneck, 2011). Nevertheless, it remains the commonest cause of fatal poisoning in the UK (NHS Choices, 2010).

Carbon monoxide results from the incomplete combustion of various fuels, including wood, coal and charcoal but also liquid fuels (petrol), oils, propane and natural gas; it is tasteless and odourless. Combining with haemoglobin at around a 240-fold higher affinity than oxygen, it forms carboxyhaemoglobin. Exposure to carbon monoxide causes hypoxia, cell damage and death. Blood levels range from 'normal' values in non-smokers of <1.5% (1.5–5% or higher in smokers), through to severe poisoning at levels of 20–25%. Concentrations of around 40% are associated with loss of consciousness (Meredith and Vale, 1988). Fatal levels, of around 60%, occur in situations ranging from accidental, occupational or recreational exposure to deliberate self-poisoning (Meredith and Vale, 1988).

Dr Marion J Marshall *is General Practitioner and Occupational Health Physician, Brunel Medical Centre, Brunel University, Uxbridge, Middlesex, and* **Dr Simon W Dubrey** *is Consultant Cardiologist in the Department of Cardiology, Hillingdon Hospital, Uxbridge, Middlesex UB8 3NN*

Correspondence to: Dr SW Dubrey

The circumstances of discovery of this patient, unconscious in a closed vehicle with the barbecue as a source, immediately suggested carbon monoxide poisoning. Symptoms of carbon monoxide poisoning can be non-specific, including headache, fatigue, confusion, breathlessness, nausea and dizziness. These symptoms are related to the effects of hypoxia on the brain, nervous system and heart. Cardiotoxicity can present with arrhythmias, chest pain, pulmonary oedema, heart failure and myocardial infarction.

Several reviews describe delayed encephalopathy and cognitive dysfunction after acute carbon monoxide exposure (Choi and Cheon, 1999), with estimates that as many as 50% of individuals will have some neurological or cognitive sequelae (Hopkins and Woon, 2006). Symptoms can include memory impairment, attention disorders, motor (movement disorders include parkinsonism and chorea type effects), visual impairment and slow mental processing. These features seem likely to result from a process of diffuse reversible demyelination.

The physiological mechanisms at the cellular level are complex and not fully elucidated, but hyperbaric oxygen helps reduce carbon monoxide-induced neuronal apoptosis. This benefit is likely mediated through preservation of mitochondrial membrane integrity (Neuman and Thom, 2008; Thom, 2009). In this patient, the delay until development of sensory neurological features was around 6 weeks but reports indicate this can be anything from 2 to 50 weeks (Choi and Cheon, 1999). The development of such sequelae cannot be predicted on the basis of the clinical history, blood carbon monoxide levels (Thom et al, 1995) or features on computed tomographic brain scans (Choi and Cheon, 1999).

Magnetic resonance scanning of the brain after carbon monoxide poisoning has revealed abnormalities in many areas (O'Donnell et al, 2000), including white matter hyper-intensity abnormalities and basal ganglia lesions. Many are still evident 6 months after the poisoning event (Parkinson et al, 2002).

Although hyperbaric oxygen is widely used (*Table 1*) for acute carbon monoxide

Case Report

A young man was found unconscious in his camper van while attending a motorcar race meeting. Following a social event he had placed a barbecue stove into his vehicle. The metal casement of the barbecue was cool enough to lift with his bare hands. He retired to bed and was discovered at 13.30 hours the following day.

He was intubated and transferred by helicopter air-ambulance to hospital. Blood gas analysis revealed a carboxyhaemoglobin level of 21.6% (normal range <1.5%). A chest radiograph and significantly elevated C-reactive protein (215 mg/litre) also indicated aspiration pneumonia. Acute renal compromise was evident from a raised creatinine level of 141 mmol/litre, which responded to rehydration. The patient was referred to a regional centre for hyperbaric oxygen therapy. The aspiration pneumonia was treated with an appropriate course of antibiotics. A total of 7 days mechanical ventilation was required. He was discharged home on day 14 with little obvious neurological deficit and Glasgow Coma Scale score of 15/15.

At discharge, the patient found multi-tasking slightly more onerous than before, which was particularly evident when driving a car. He returned to work 4 weeks after discharge from hospital, with only minor issues around mental concentration. At 6 weeks after his exposure, he developed discomfort and hyperaesthesia of both hands with a mottled appearance to the palms. These sensory features responded almost immediately to the use of the calcium-channel blocker nifedipine; this was maintained and then weaned off and stopped after 6 weeks of treatment, as his symptoms resolved.

Subsequent referral for full neuropsychological assessments, 3 months after discharge, revealed no evidence of a significant drop from estimates of his previous levels of functioning. Ear, nose and throat specialist follow up is in place to monitor the bilateral grommets placed during hyperbaric oxygen therapy. Audiology tests have been normal.

Six months after his accidental poisoning he is back motor racing and performing well at this event.

Table 1. Institutions that offer 'category 1' medical hyperbaric oxygen facilities

Name of chamber	Location of unit
London Hyperbaric Medicine	Whipps Cross University Trust NHS Trust, London
Hyperbaric Medicine Unit	Aberdeen Royal Hospitals NHS Trust, Aberdeen
Chichester Hyperbaric Unit	St Richard's Hospital, Chichester, W. Sussex
East of England Hyperbaric Unit	James Paget Hospital, Great Yarmouth
Hyperbaric Unit	St Peter Port, Guernsey
North of England Medical Hyperbaric Unit	BUPA Hospital, Lowfield Road, Anlaby, Hull
Atlantic Enterprise UK Ltd	Poole Hyperbaric Centre, Poole, Dorset
The Hyperbaric Medical Centre (DDRC)	Tamar Science Park, Derriford, Plymouth
London Diving Centre	Hospital of St John and St Elizabeth, London
North West Emergency Recompression Unit	Murrayfield Hospital, Thingwall, Wirral

Full details of these category 1 (category 1 = facilities capable of receiving patients in any diagnostic category who may require life support either immediately, or during hyperbaric oxygen therapy) units and other hyperbaric chambers, address, contact phone numbers and facility categories (categories 2–4, depending on the level of care required by the patient) can be found on the British Hyperbaric Association website (www.ukdiving.co.uk). This list is fluctuant, with units and categories changing; readers are advised to contact the resources cited to obtain the latest information

poisoning, the evidence for any benefit over normobaric oxygen in preventing neurological deficit remains contentious (Thom et al, 1995; Buckley et al, 2005). However, depressed consciousness, neurological deficit, cardiac dysfunction and pregnancy with high carbon monoxide levels remain current primary criteria to initiate hyperbaric therapy.

Conclusions

Recognized as the most common cause of fatal poisoning worldwide (Hopkins and Woon, 2006), many people remain unaware of the dangers from carbon monoxide. On the lower end of the toxicity scale, a 'winter (heating season) headache' can indicate carbon monoxide exposure. At another extreme, circumstances seemingly benign – using portable generators, heaters in vehicles, camping stoves and barbecues in tents, garages, sheds or other enclosed areas – can result in serious injury or death. BJHM

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LEARNING POINTS

- Carbon monoxide poisoning is the commonest cause of fatal poisoning worldwide.
- Barbecues, in common with all other combustion devices, have the potential to produce carbon monoxide poisoning.
- Suspect carbon monoxide poisoning in patients found with impaired consciousness in confined environments and potential exposure to gaseous compounds (carbon monoxide).
- Delayed neurological sequelae are a recognized feature of recovery from carbon monoxide poisoning.