Mass carbon monoxide poisoning

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Abstract

The largest occurrence of carbon monoxide poisoning in Britain demonstrates the potential for mass accidental poisoning. It emphasises the need for strict public health controls and the importance of good liaison between emergency services to ensure that such events are quickly recognised and that the necessary resources are organised.

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Carbon monoxide poisoning remains a leading cause of poisoning related deaths both in the UK^{1,2} and the United States.³ We describe the largest carbon monoxide poisoning episode reported in British medical literature. This event highlights the potential for mass accidental poisoning.

Case report

An evening memorial service was held in a church hall. The meeting was attended by 60 adults, who all arrived at around 7.00 pm. Everyone was seated in the middle of the hall and no alcohol was drunk. During the service, most of those present experienced symptoms, including lightheadedness, headache and nausea, which they attributed to an emotional response to the service. At 9.15 pm, a 54 year old man suddenly fell off his chair unconscious. An ambulance was called. While waiting for this to arrive, another man complained of chest pain and many others complained of feeling unwell.

The 54 year old man arrived at the accident and emergency (A&E) department at 9.45 pm. He was a non-smoker, fully conscious, but distressed with a resting tachycardia. Suspicion of carbon monoxide poisoning was aroused by the clinical presentation combined with rumours of many people affected. Carbon monoxide poisoning was confirmed at 10.00 pm when his carboxyhaemoglobin concentration was shown to be 35%. Around this time, an ambulance crew bringing a further two patients from the scene reported that there were multiple symptomatic casualties on-site. At 10.15 pm, liaison with police and ambulance services established that a large number of people were affected. A major incident was declared.

Thereafter, a fleet of ambulances equipped with oxygen was dispatched to the scene. At the hospital, extra oxygen cylinders and trolleys were made available. Doctors from a variety of other specialties were recruited to help. An additional medical receiving ward was opened and laboratory services were alerted.

Table 1 Carboxyhaemoglobin (COHb) concentrations of patients who attended the A&E department

COHb range	No
0%–10%	5
11%–20%	12
21%–30%	4
31%–40%	1

Local radio was used to attempt to contact those individuals who had attended the meeting, but who had left and gone home. In total, 22 patients presented to the A&E department. The distribution of measured carboxyhaemoglobin concentrations is shown in table 1. All patients were treated with high flow oxygen and 17 were subsequently admitted to the medical wards. Two additional patients attended the A&E department the next day. Sixteen patients were discharged from hospital within 24 hours, but one, who had been complaining of chest pain, was kept for a myocardial infarction screen, which proved positive.

It later transpired that building work on the hall several months previously had affected its ventilation and also that the central heating boiler had been “repaired” two days before the incident.

All of those admitted were invited to attend a follow up clinic three months after the incident. Eleven patients attended: seven had experienced neuropsychiatric problems, including depression, apathy, irritability, and poor concentration. For four of the patients, these episodes were self limiting, lasting only a few weeks, but three patients described persistent symptoms.

Discussion

Carbon monoxide is produced from incomplete combustion of carbon based fuels. It is a tasteless, odourless, colourless, and non-irritating gas. Production is frequently associated with faulty heating equipment. Carbon monoxide has several pathophysiological effects^{4,5}: firstly, it binds to haemoglobin with an affinity 250 times that of oxygen to form carboxyhaemoglobin, thus displacing oxygen. Secondly, it alters the binding sites on the remaining haem groups and this affects oxygen dissociation, making transfer of oxygen to tissues more difficult.^{5,6} Thirdly, at a cellular level, it binds to cytochrome *a3*, causing partial inactivation with a direct effect on cellular respiration.^{5,7} There is debate regarding the relative importance of each of these mechanisms, but the net result is to cause generalised tissue hypoxia which can present clinically with a variety of symptoms and signs.⁸ These range from non-specific presentations such as headache, fatigue, or gastrointestinal symptoms

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through to altered conscious level and coma.⁵ The severity of the poisoning and the nature of its presentation depends on the inspired concentration of carbon monoxide, the length of exposure, and the pre-existing health of the patient. Correlation between clinical presentation and measured concentrations of carboxyhaemoglobin is poor—the degree of metabolic acidosis may be a better marker of severity.⁹ In non-smokers, the concentration of carboxyhaemoglobin is normally around 1% of total haemoglobin. Cigarette smokers commonly have values between 5% and 10% and urban joggers have been recorded with even higher measurements. Symptoms typically develop at values between 10% and 30%, with measurements above that being progressively more likely to be associated with altered conscious level and confusion. Pre-existing cardiovascular disease makes tissue hypoxia more likely to present clinically, and may result in myocardial infarction. Concentrations around 40% are associated with loss of consciousness and when in excess of 60% convulsions and death may occur.¹⁰ The wide range of carboxyhaemoglobin concentrations measured in this incident reflects both the different times at which patients presented to hospital, as well as the duration of oxygen treatment before carboxyhaemoglobin was measured.

Recognition of carbon monoxide poisoning can be difficult due to various non-specific presentations.^{11 12} Failure to diagnose it may have serious implications not only for patients affected, but also for other members of a family or group who may subsequently be exposed to carbon monoxide poisoning in the same environment. Previous reports of carbon monoxide poisoning in the UK have involved small groups of individuals or families. This report demonstrates the potential for mass casualties from a single incident and highlights the need to ensure adequate health and safety checks on halls and other public meeting places. It also demonstrates the importance of early communication between the emergency services and rapid mobilisation of staff in such an event.

Treatment of carbon monoxide poisoning requires administration of high concentration oxygen, thereby reducing the half life of carboxyhaemoglobin from approximately 240 minutes to less than 90 minutes.¹³ Hyperbaric oxygen further reduces the half life and has an important role in patients with high carboxyhaemoglobin concentrations, loss of consciousness, significant neurological symptoms, cardiac complications, and in pregnancy.^{5 14}

After exposure and despite initial treatment, many patients subsequently experience neuropsychiatric symptoms. Features include lethargy, disorientation, and inability to concentrate, recent memory impairment, and personality changes such as irritability and moodiness. Neuropsychiatric problems may develop insidiously over weeks after carbon

monoxide poisoning.¹⁵ These problems may result from diffuse reversible demyelination caused by the hypoxic insult of initial carbon monoxide poisoning. The follow up of patients after the incident described supports the previously reported high incidence of neuropsychiatric sequelae after carbon monoxide poisoning.^{15 16} Such sequelae are difficult to treat—the best hope lies with their prevention using early high concentration oxygen treatment. Unfortunately, current evidence suggests that there is considerable room for improvement in the management of these patients in the UK.¹⁷

The logistic difficulties of managing an incident of this scale in terms of liaison with emergency services, mobilising medical and nursing staff, ambulances, oxygen, trolleys, ward space, and laboratory support services outweighed the problem associated with the diagnosis, which itself was eased by the number of casualties involved. This contrasts with a previous report which emphasised the difficulty of diagnosis and relative ease of treatment with reference to poisoning episodes involving fewer patients.¹⁸

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