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LATE SEQUELAE OF CARBON MONOXIDE POISONING 2 CASE REPORTS

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Key Words

Carbon monoxide, hyperbaric oxygen, sequelae, treatment.

Introduction

Neuropsychiatric manifestations of acute carbon monoxide (CO) poisoning may include non-focal changes in mental state, seizures, amnesia, apraxia, agnosia, Parkinsonism, cortical blindness, incontinence and peripheral neuropathy. A lucid period of up to twenty one days may occur followed by the delayed sequelae of CO poisoning which may include aphasia, apathy, disorientation, psychosis, gait disturbances, faecal and urinary incontinence and bradykinesia. Cognitive and neurological deficits may also be present, as can personality changes with impulsiveness, violence, verbal aggressiveness and mood changes.[1]

This syndrome has a reported incidence of 3% to 40%,[2] with a set of risk factors having been identified within the group of affected patients.[3] The neuropsychological deficits associated with CO poisoning are highly variable despite exposure to similar levels of CO poisoning.[4] The white matter of the frontal lobe is involved but the pathological mechanism leading to demyelisation, petechiae, oedema and necrosis is poorly defined. Depressed cardiovascular function induced by CO, and a limited cerebral blood flow, may be major factors leading to neurologic cellular damage from CO poisoning.[1]

Case histories

The Hyperbaric Unit at Fremantle Hospital actively treats CO poisoning with about 30 cases per annum being referred from Perth and more remote regions. The unit recently treated two cases with apparent late sequelae with resulting clinical improvement.

CASE ONE

A 61 year old female patient, who attempted suicide by connecting the exhaust pipe of her car to the cabin, was found by her neighbour at about 0950 with the car engine still running. The Glasgow Coma Scale (GCS) at the site was reported to be 9/15. In the Emergency Department of a peripheral hospital the patient was noted to have deteriorated with hypotension (55/28 mm Hg). She had an oxygen

saturation of 77% on air and an acidosis with a bicarbonate of 12.1 mmol/l (normal 22-32 mmol/l). Her ECG showed sinus tachycardia and carboxyhaemoglobin estimation revealed a level of 41.9% four hours after being removed from the vehicle. Transfer to Fremantle Hospital was accomplished with the patient intubated and ventilated.

In the Intensive Care Unit at Fremantle Hospital the patient was unrousable despite withholding all sedation. Initial treatment was performed using an 18 m for sixty minutes with a thirty five minute decompression (18:60:35) hyperbaric oxygen treatment (HBO_2T) table while the patient remained sedated and ventilated. Sedation was withdrawn overnight and extubation performed the following morning with improved and stable observations. The patient received two further daily treatments using the same treatment table. She was noted to have an improved mental state during and between visits to the chamber. Without sedation the patient responded to commands by eye opening only and was restless and disorientated during the second day. A fourth treatment using the RN 61 table was administered on day four after which it was noted that she was able to conduct a rational conversation and read printed material. The patient was handed over to the care of the medical team who discharged her seven days after admission. Appropriate psychiatric follow up had been arranged.

Eighteen days after her discharge the patient was readmitted with a marked deterioration in psychological, cognitive and neurological function after having been found at the side of the road in the driver's seat of her car and clothed only in a bath towel. Signs and symptoms included urinary and faecal incontinence, inability to perform routine activities of daily living, loss of short term memory, disorientation in time, place and person, ataxic gait, and inability to perform a Mini Mental State Examination (MMSE). Neuropsychological testing was difficult and attempted on two occasions with the latter revealing exceptional weakness in her Verbal Test Score and inability to negotiate Performance tests. Short and long term recall for verbal and non-verbal material was zero and the patient was unable to distinguish between her left and right hand.

The patient remained on a medical ward for four weeks at the end of which she was assessed to be suitable for nursing home care due to an inability to care for her self. A subsequent referral of the patient for hyperbaric treatment resulted in a series of 14 daily hyperbaric treatments using table 14:90:24 being administered with steady recovery almost on a daily basis. The patient regained urinary and faecal continence and independence in activities of daily living (ADLs). Post-treatment neuropsychological testing revealed an ability to cooperate with full scale IQ in the average range. Memory function assessment for verbal material was normal whilst non-verbal was moderately impaired.

The patient was discharged to live independently, although an assessment before hyperbaric treatment indicated permanent placement in hostel accommodation. At follow up interview the patient was functioning at a normal level living independently and requiring only the support of her local general practitioner.

CASE TWO

A 45 year old male had apparently been exposed to 30 minutes of car exhaust gas four days before assessment. Upon failing to follow through on his suicide attempt the patient had consumed beer and Prothiaden (dothiepin hydrochloride) without acute sequelae.

He presented to his General Practitioner with various symptoms and general malaise the day after the incident but was reassured there was nothing seriously wrong. No carboxyhaemoglobin estimation was performed at presentation. His condition deteriorated over several days following the poisoning with poor balance, headache, poor short-term memory and slow mentation noted by his wife. Initial assessment by the hyperbaric team four days later revealed a Sharpened Romberg of less than 5 seconds on three attempts, a Mini-Mental State Examination (MMSE) score of 17/30 and a GCS of 15/15.

Following a treatment using the 18:60:35 protocol four days after the poisoning the patient's MMSE improved to 23/30 and the Sharpened Romberg time was 40 seconds. The patient was given two further treatments with subsequent improvement arresting the decline in his condition. Treatment had to be curtailed for social reasons and the patient has not returned for review.

Discussion

The use of HBO_2T for carbon monoxide poisoning remains contentious with polarised views about its efficacy. A recent review of the evidence available suggests that a substantial study with adequate controls and patient numbers still needs to be completed.[5] These two cases had variable signs of delayed sequelae of carbon monoxide poisoning, however both responded to hyperbaric oxygen therapy. Clinical deterioration was reversed in both cases preventing nursing home placement in the female patient, who maintains her independence ten months after her treatment. Risk factors for neuropsychological sequelae include older age (50+ yrs), loss of consciousness, COHb > 25% and metabolic acidosis.[3]

Conclusion

In cases demonstrating neuropsychiatric sequelae due to CO poisoning it is worth considering hyperbaric oxygen

therapy to assist in the recovery from a debilitating syndrome.

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