A CASE OF NEUROPSYCHOLOGICAL SEQUELAE OF CARBON MONOXIDE POISONING TREATED WITH HYPERBARIC OXYGEN THERAPY

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ABSTRACT

Carbon Monoxide is one of the most common causes of inhalation poisoning worldwide and can result in significant morbidity due to persistent neuropathology and cognitive sequelae. The manifestation of carbon monoxide poisoning are non specific and severity of symptoms range from mild such as headache, confusion, lack of energy to severe, such as coma, respiratory depression, cardiac dysfunction and even death. Hyperbaric oxygen therapy has been used for decades in the treatment of acute and delayed effects of carbon monoxide poisoning. Hyperbaric Oxygen is the only treatment for acute CO poisoning where it competes with CarboxyHemoglobin preventing the resultant damage and in delayed neuropsychological sequelae. This case report is of a young lady from rural Haryana presented after 48 hours of CO Poisoning with acute confusion, partial retrograde and complete anterograde memory loss. Following 15 days of hyperbaric oxygen she showed near complete recovery and her Mini Mental Scale Examination (MMSE) score which was 12/30 on the first day went up to 29/30. This case demonstrates that HBO therapy is efficacious in recovery of delayed neuropsychiatric sequelae of Carbon Monoxide poisoning.

Key Words: Carbon monoxide, hyperbaric oxygen therapy, cognitive sequelae.

INTRODUCTION

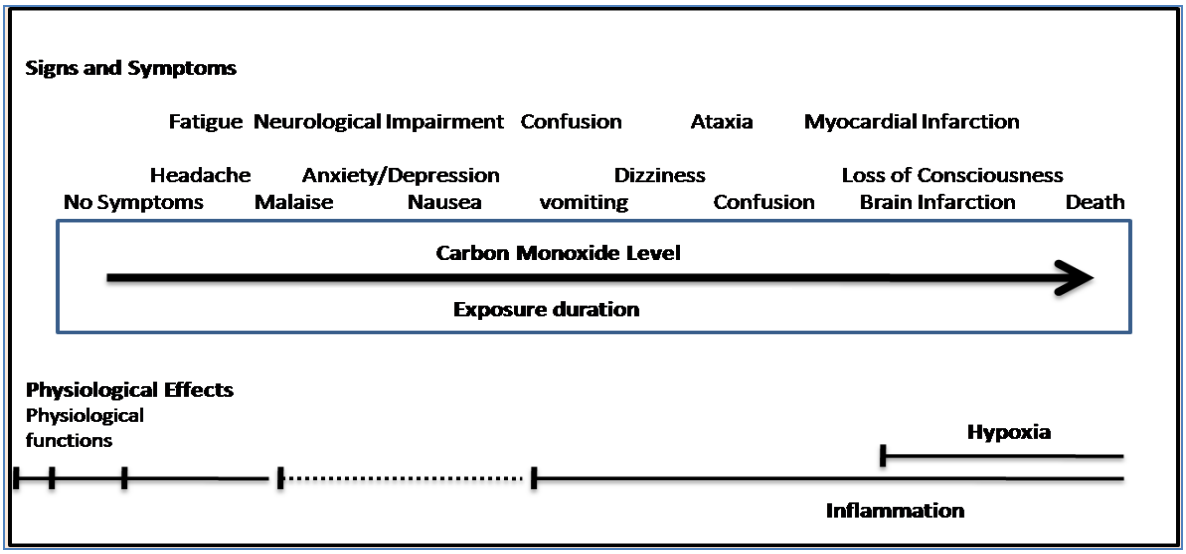
Carbon monoxide (CO) is a colourless, odourless, tasteless, non irritating, but significantly toxic gas. CO is one of the leading causes of accidental poisonings worldwide and also in India both in rural and urban areas. However there is improper reporting of morbidity and mortality attributable to suspected CO poisoning. The sources of exogenous carbon monoxide that causes poisoning include motor vehicle fumes, poorly functioning heating systems (gas heaters, ovens, stoves),improper use of coal or wood stoves and inhaled smoke [1,2,3]. It often results in residual and persistent neuropathologic and cognitive sequel[1, 2, 3].Carbon monoxide's affinity for haemoglobin is more than 200 times that of oxygen resulting in the formation of carboxyhemoglobin causing hypoxia and shifting the oxyhemoglobin dissociation curve to the left [2,3,4] .The acute injuries caused by CO are due to a hypoxic stress mediated by an elevated carboxyhemoglobin (COHb) level[3,4,5,6].In acute phase, the level of carboxyhemoglobin is directly proportional to the severity of poisoning and the long term effects (Table1). Experts agree that it is difficult to estimate the incidence of CO poisoning cases, because the symptoms resemble many other common ailments [1.2].High index of suspicion, and a careful history taking, helps in making the diagnosis (Figure 1).

Table 1: Symptoms associated with a given concentration of COHb

Level of COHb (%)	Symptoms
0-10	No symptoms
15	Mild headache
25	Nausea and serious headache
30	Symptoms intensify
45	Unconsciousness
>50	Death

The delayed neuropsychological sequelae are a result of inflammatory mediated residual injury to the brain and may manifest many years later. Administration of supplemental oxygen is the cornerstone of treatment of CO poisoning. Hyperbaric Oxygen inhalation will hasten disassociation of CO from haemoglobin to occur at rate greater than that achievable by breathing pure oxygen at sea-level pressure and is better than normobaric oxygen [4,6,8].

Figure 1: Spectrum of symptoms of exposure to Carbon Monoxide, on the basis of duration of exposure.



CASE REPORT

A 31 year old lady from Sirsa, Haryana presented with a history of acute confusion and partial retrograde and total anterograde memory loss of approx 48 hours duration following exposure to leakage from a gas heater for about 45 minutes. She was lying unconscious on the floor after 12 hours and on regaining consciousness, she was in a confused state and unable to recognize her family members.

When examined, she was alert but not oriented to person, place or time. Mood was inappropriate; speech was spontaneous with normal rate but of high tone. She denied any perceptual disturbances. The patient displayed impaired short term and long term recall memory. There was no history of any psychiatric illnesses, abnormal behavior, seizures or head trauma.

Assessment of neuropsychological parameters by MMSE gave her a score of 12/30 indicating severe impairment in orientation, attention, anterograde and retrograde memory, calculation, language and

construction (Table 2, 3). She required constant reorientation by family members to place, time and person. The patient showed no ability to recall events spontaneously that had occurred the day before.

Patient underwent MRI Brain scan and EEG which were normal. The level of carboxyhemoglobin was not measured at the rural hospital since this facility was not available and since more than 48 hours has elapsed, it was not considered relevant at this hospital. As a part of medical treatment she was prescribed an anti oxidant, Clonazepam (1mg), Aspirin (75mg) for 10 days. The patient was referred to this centre 48 hours after the event for Hyperbaric Oxygen Therapy for possible resolution of her cognitive symptoms. The patient underwent fifteen, ninety minute treatment with hyperbaric oxygen at 2.4 ATA.

On completion of hyperbaric treatment the patient was alert and oriented to person, place, month, and year her mini mental state examination score was 29/30. Her short term memory was normal (ability to recall three objects at 3 minutes and three of three with prompting). There was also an increased ability to recall recent events. She showed improvement in her recall and remembered the event and was aware of why she was undergoing treatment. There was also a remarkable improvement in her ability to care for herself and was able to perform activities of daily living without assistance. Neuropsychological testing (MMSE Score) obtained two weeks after hyperbaric oxygen treatment disclosed a further improvement with score of 29/30 from score of 12/30 at her initial testing. The patient had improved memory for events, but was unable to reproduce the designs after a delay. An additional set of five hyperbaric treatments for residual cognitive losses were given, however this did not improve her MMSE score further.

Table 2: Assessment of patient using MMSE during different phases of treatment

Assessment	Patient's Score			Maximum Score
	Day 1	Day 10	Day 15	
Orientation to Time	0	5	5	5
Orientation to place	0	5	5	5
Immediate Recall	3	3	3	3
Delayed Verbal Recall	2	3	3	3
Attention	0	2	5	5
Naming	2	2	2	2
Repetition	1	1	1	1
3 Stage Command	3	3	3	3
Reading	1	1	1	1
Writing	0	0	1	1
Copying	0	0	0	1
Total	12	25	29	30

Table 3: Interpretation of MMSE

Score	Interpretation
24-30	No cognitive impairment
18-23	Mild cognitive impairment
0-17	Severe cognitive impairment

DISCUSSION

Carbon monoxide poisoning is resulting in more than 50,000 emergency department visits per year globally. Sources of carbon monoxide include faulty furnaces, inadequate ventilation of heating sources, and exposure to engine exhaust. Spectrum of CO Poisoning symptoms may be wide and variable, most common symptoms are mild headache, nausea, malaise, dizziness, syncope, altered mental state and loss of consciousness [3,4,5,6]. The symptoms of carbon monoxide poisoning occur with many other types of poisonings and infections. The diagnosis is often difficult and hence high level of suspicion is essential [1, 2]. If CO poisoning is suspected, the carboxyhemoglobin level is measured if available and determines the severity of the poisoning[7, 8, 9.] as explained in Table 1.

No specific therapy for neuropsychological sequelae after carbon monoxide poisoning is well established, but clinical experience has shown that patients with sequelae should be treated with hyperbaric oxygen therapy [10]. Substantial improvement in memory, concentration, attention, and activities of daily living were seen in this patient when treated with hyperbaric oxygen. Several animal studies show that treatment with hyperbaric oxygen is effective in reducing carbon monoxide associated brain injury and suggests that hyperbaric oxygen acts possibly by inhibiting cellular processes such as oxidative injury and brain lipid peroxidation[11,12,13,14]. Weaver et al[3] reported that treatment of patients with acute symptomatic CO poisoning with HBOT sessions appeared to reduce rate of cognitive sequelae and support the use of HBO. It has been reported that HBOT has provided a prominent improvement in the early and late effects of CO poisoning and improvement is more effective when applied in acute phase. Hyperbaric-oxygen therapy elevates arterial and tissue oxygen tensions, promoting carbon monoxide elimination and also increases adenosine triphosphate production and reduces oxidative stress and inflammation[12, 13]. Thom et al[4] showed in their study that hyperbaric oxygen leads to improvement of neuropsychological consequences similar to response in our patient. HBOT is a life saving procedure in acute carbon monoxide poisoning as it speeds removal of CO from tissues and counters a number of its deleterious effects. Further research is needed to examine the exact mechanism by which hyperbaric oxygen acts. In this case report, the patient continued to have some degree of neurological sequelae despite the treatment given.

CONCLUSION

Acute carbon monoxide poisoning continues to be an important health problem. The spectrum of CO poisoning symptoms may be wide and variable, therefore high index of suspicion, detailed history and examination is required. Hyperbaric oxygen therapy has not only a well defined role in acute but also in the delayed effects of CO poisoning and the same has been demonstrated in this case report.

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