A Case of Severe Carbon Monoxide Poisoning Due to Gas Geyser with Cerebellar Involvement

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ABSTRACT:
Carbon monoxide is responsible for a large number of accidental domestic poisoning and deaths throughout the world. Diffuse hypoxic brain injury due to carbon monoxide poisoning while using gas geyser is very well known. We report a case of young male patient presented with hypoxic brain injury due to carbon monoxide poisoning while using gas geyser in bathroom which didn't have proper ventilation. There is involvement of bilateral cerebellum, which is very rare in such cases. Patient was treated with hyperbaric oxygen therapy and showed improvement on follow-up after three months.

CASE REPORT

HISTORY : 30 year male science professor Mr. PC was found unconscious in bathroom at one government guest house at Anand city, Gujarat in January 2013. He went to take bath and was found unconscious in the bathroom by guest house staff. He was alone in room and last he was seen in room thirty minutes before he was found unconscious. The door had to be broken to rescue him. Apparently he was taking bath with hot water using gas geyser. He was brought to local hospital in comatose state and was given IV Fluids and primary treatment and then shifted to one corporate hospital for further management. Patient regained consciousness within twelve hours but remained in a state of altered sensorium for next twelve hours. He could not remember any events of the incident. After that patient developed profound antegrade as well as retrograde amnesia. No complaints of seizure or any weakness of any limbs.

EXAMINATION : Patient was conscious. His Mini-mental state examination score was 14/30 and Addenbrookes score was 51/100. Patient was having mild confusion with disorientation to time, place and person and inability to register and recall things. He was having language dysfunction in the form of difficulty in comprehension and anomia. Otherwise no other neurological deficit.

INVESTIGATIONS : All routine investigations including renal and liver function tests were normal. Arterial blood gas analysis was suggestive of acidosis with pCO2 50.1 mmHg. EEG suggestive of diffuse encephalopathy. MRI brain s/o bilateral symmetrical hyper intensity in T2 and FLAIR and restricted diffusion in bilateral basal ganglia and bilateral temporal cortical gyri and bilateral cerebellar hemisphere s/o severe hypoxic encephalopathy.

MANAGEMENT : After two days of the event patient was subjected for hyperbaric oxygen (HBO) therapy for 7 days in appropriate doses. Patient was kept on neurocognitive, neurobehavioral therapy and speech therapy.

FOLLOW-UP : On follow-up after three months patient had some improvement in language and memory function however still he was not able to do his job. His Addenbrookes score was improved to 76/100.

DISCUSSION
Carbon monoxide (CO) is responsible for a large number of accidental domestic poisoning and deaths throughout the world¹. CO is a colourless and odourless toxic gas produced as a by-product of incomplete combustion of carbon-based fuels and substances. The neurologic sequelae are the most frequent form of morbidity². The pathophysiologic mechanisms of CO toxicity can be divided into hypoxic and cellular theories³. The affinity of CO for heme protein is approximately 250 times that of oxygen, and the formation of carboxyhemoglobin reduces the oxygen-carrying capacity of blood, causing tissue hypoxia⁴. CO inhibits the mitochondrial electron transport enzyme system and activates polymorphonuclear leukocytes, which undergo diapedesis and cause brain lipid peroxidation, leading to the delayed effects of CO poisoning. The clinical presentations and imaging features of CO poisoning are diverse.

Acute and intense CO poisoning can lead directly to diffuse hypoxic–ischemic encephalopathy predominantly involving the gray matter there is a predilection for the temporal lobe and the hippocampus. Basal ganglia mainly globus pallidus and occasionally caudate nucleus, putamen, and thalamus are involved in CO poisoning⁵. Involvement of the brainstem and cerebellum may be a reflection of more severe poisoning because the posterior structures are more resistant to hypoxia. The lesions usually appear as asymmetric hyperintense foci on T2-
weighted and FLAIR images. Cerebeller involvement predicts poor prognosis in such patients as its suggestive of severe hypoxia.

Such cases occurred when water used for bathing was heated by gas geysers fitted within ill-ventilated small bathrooms, and there was a clustering of such cases in winter months.

The mainstay of treatment is 100% oxygen. In 1895, Haldane demonstrated that a mouse could be kept alive by exposure to HBO at the same time as CO. This seminal experiment established role of HBO. HBO has many benefits. The half-life of carboxyhaemoglobin at 3 absolute atmospheres of oxygen is only 23 minutes. It showed improved mitochondrial function, impairment of platelet adhesion in the capillaries and inhibition of lipid peroxidation. But contrary to expectation, clinical trials of HBO have given conflicting results. Previous study demonstrated that HBO therapy reduced the incidence of neurological sequel as well demonstrated that neither clinical history nor the carboxyhaemoglobin level predicts which patients may show sequel after CO poisoning. But a recent Cochrane review suggested that firm guidelines regarding the use of HBO cannot be established. Ongoing trials will soon provide further information. In the absence of firm evidence most centres continue using HBO for severe neurological deficit including coma and for myocardial ischaemia. The decision about HBO will often depend on ease of access to a hyperbaric facility. The time-frame within which hyperbaric oxygen is most effective is not known.

Recently extracorporeal membrane oxygenation (ECMO) suggested for patients with respiratory failure of a reversible etiology including CO poisoning. The association between ECMO and improved neurologic function is uncertain. The effect of ECMO on cerebral blood flow or oxygenation metabolism is controversial.

While use of ECMO for severe potentially reversible cardio-respiratory failure could be a rescue strategy, we need more well-designed studies of ECMO compared to HBO in the context of neurological sequel. The availability of ECMO compared to HBO might be an important issue. In present case bilateral cerebellum involvement was suggestive of severe hypoxic injury due to CO poisoning and he was benefited with HBO therapy as shown on follow-up with improved clinical status and Addenbrookes score.

REFERENCES

2. Omaye ST. Toxicology 2002; 180:139–150.