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Case Report

Accidental Carbon monoxide poisoning with Neurological Sequelae

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Abstract:

Carbon monoxide (CO) intoxication is one of the leading causes of accidental poisonings[1]. It often leads to diagnostic errors, because of its presenting symptoms are extremely nonspecific and confounding. Symptoms commonly include headache, dizziness, weakness, vomiting, chest pain and confusion. Large exposures can result in loss of consciousness, arrhythmias, seizures, or death. The most common location of exposures causing CO poisoning are in homes and less commonly in workplaces [2]. Unintentional, non-fire related CO poisoning is responsible for approximately 450 deaths and 21,000 emergency department (ED) visits each year in United States[3,4,5].

Key words: *Carbon monoxide poisoning, smoke inhalation, Brain Injury .*

Introduction:

Carbon monoxide (CO) is a colorless, odorless, nonirritating gas that is produced through the incomplete combustion of carbon-containing substances. Sources of CO include: boilers, furnaces, cars and trucks, generators and other gasoline or diesel-powered engines, gas and propane heaters, woodstoves, gas stoves, fireplaces, tobacco smoke, forklifts and fires. CO poisoning occurs from breathing in elevated air levels of carbon monoxide. Unusual sources include exposure to methylene chloride which is metabolized to CO, causes hemolysis with increased metabolism of hemoglobin. The diagnosis of carbon monoxide poisoning is frequently made obvious by the patient's own history, collateral history from attending paramedics or by co-presentation of others who shared a common environment. However, patients with carbon monoxide poisoning who present alone and do not, or can not give a history of exposure are acutely dependent upon their physicians' ability to recognize an aggressive multi-system presentation. Carbon monoxide intoxication is one of the main causes of diagnostic errors in emergency medicine[6], often misdiagnosed as influenza like illness, gastroenteritis or headache. High index of suspicion and a careful history taking helps in making the diagnosis. We present a case of accidental carbon monoxide poisoning presented at ER with unconsciousness.

Case Report:

A 36 years old female, Mrs. A with no known co-morbidities presented to our ER with sudden unconsciousness while she was working at her workplace. She was discovered by one of her co-workers in a closed room lying unresponsive on floor close to a running gas generator during power outage. Mrs A's past medical history was unremarkable. She had no known allergies. On admission to ER her GCS (Glasgow Coma Scale) was 5/15, no evidence of associated tonic-clonic movements, tongue bite, frothing from mouth, vomiting, bladder and bowel incontinence. There was no history of fever, seizure, headache, vomiting and substance abuse prior to this episode. At the time of admission her pupil was of normal size and reacting to bright light, planter was extensor bilaterally. Her spontaneous breathing was shallow and weak with Oxygen saturation was 89% with 10 L O_2/min via face mask, her pulse was 96 beats per minute, temperature 98.2^0 F. Other systemic examination was normal. Arterial blood gas (ABG) showed P^H 7.25, PCO_2 31, PO_2 72, HCO_3 14.5, BE -11.5. Lactate level 5.9 mmol/L, Blood sugar 8.3 mmol/L. Carboxyhemoglobin (COHb) level could not be obtained due to unavailability of test. She was immediately intubated at ER for airway protection & Type I respiratory failure and supplied 100 % Oxygen. During tracheal intubation upper airway was found edematous, also laryngeal edema was noted. Her laboratory works was unremarkable including normal cardiac enzymes except for increased level of serum lactate. Her subsequent ABG were also unremarkable. Her ECG showed normal sinus rhythm with no ST changes. Steroid was started to reduce laryngeal edema along with other supportive

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treatment. MRI of Neck was done which showed small amount of fluid collection in pre epiglottic space. MRI brain showed hyper intensity in both cerebellar hemispheres including vermis [Figure 1]. As patient's condition gradually improved, she was liberated from ventilator on the 8th day of admission. Patient was confused, suffered from slowing of psychomotor function, impaired short-term memory and

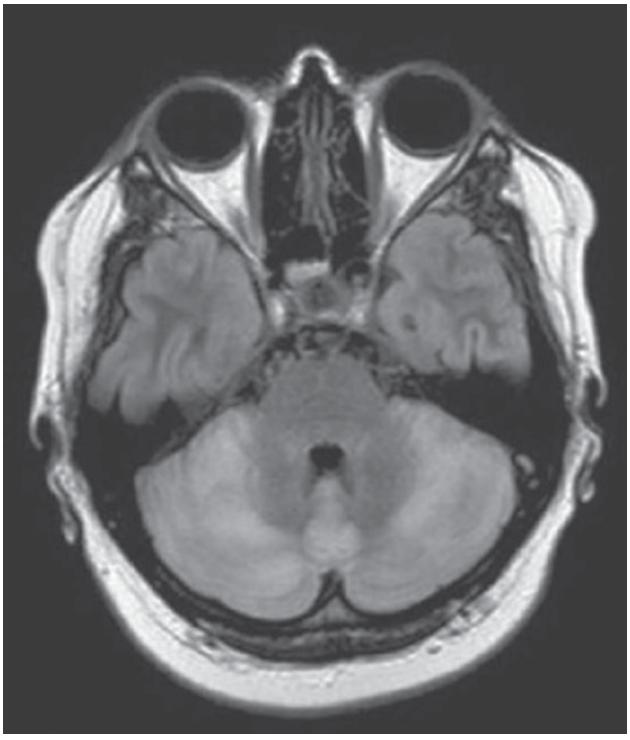

Figure 1

reduced sustained attention. She was transferred under department of neuromedicine with collaboration of physical medicine and rehabilitation department for further assessment of her neurological sequelae and planning for rehabilitation program.

Discussion:

CO toxicity occurs by competitive binding of CO to the heme group of hemoglobin resulting in increased affinity of the remaining of Hb for oxygen, shifting the oxygen-hemoglobin dissociation curve to the left[7]. CO binds to cardiac and skeletal myoglobin also. A "rebound effect" with delayed return of symptoms may be due to late release of CO from myoglobin with subsequent binding to hemoglobin[8].

The mean half-life of COHb is 320 minutes in room air, 80.3 minutes at 100% oxygen at one atmosphere, and 23.3 minutes at three atmospheres. Continued exposure to CO can lead to flu-like symptoms, headaches, dizziness, tiredness, and nausea that may progress to confusion, irritability and impaired judgment, memory impairment and incoordination or even death. Normal COHb levels are less than 5%, up to 9% in cigarette smokers. Serious toxicity is associated with levels above 25% and risk of fatality at 70%. In late presenting patients, a normal COHb level cannot be used to rule out poisoning[9].

In our patient, diagnosis was made on the basis of history, presence of obvious exposure history, response to supportive treatment and exclusion of other causes[10]. This patient also had residual neurological findings. The cause could be prolonged exposure to toxic levels of CO resulting in hypoxic brain damage.

Treatment includes immediate removal of the victim from the exposure and administration of high-flow or 100% oxygen by a nonrebreather reservoir oxygen mask[11]. Hyperbaric oxygen (HBO) is also used in the treatment of poisoning, but consensus has still not been reached for or against its use[12].

Prevention always takes precedence over everything else. Generator misuse leads to deaths from carbon monoxide (CO) poisoning, injuries and burns- all of which happen too often during power outages and storms. Portable generators should never be used in an enclosed space or indoors. There should be always at least 20 feet distance between generator and house/office with the engine exhaust directed away from windows and doors. Generators should not be used in the rain. If possible, a generator with built-in CO safety technology should be used.

Conflict of interest:

The authors declare that there are no conflicts of interest regarding the publication of this article.

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