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Radiodiagnosis

CARBON MONOXIDE POISONING - DESCRIPTIONS, DIAGNOSIS AND AWARENESS, FIRST KNOWN IDENTIFIED CASE IN BASTAR SAMBHAG, CHHATTISGARH : A RARE CASE REPORT.

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ABSTRACT Carbon monoxide (CO) poisoning is a kind of unintentional poisoning during childhood, making a global health issue. It is a fatal disease with the mortality rates range between 1% and 31%. We report a case of CO poisoning in a 1 yr male child. As the CO poisoning can give a wide range of clinical picture, it's oftnely not easy to get a clue for diagnosis. Imaging modalities (CT/MRI) play a major role to make the diagnosis. Unfortunately in our case the patient has died 10 days after the admission which is indicating it's fatal nature. Mostly the CO poisoning occur among the people with low socioeconomic status and required an awareness among them.

KEYWORDS : Carbon Monoxide (co) Poisoning, Child, Low Socioeconomic Status, Awareness.

INTRODUCTION

Carbon monoxide (CO) poisoning is a kind of unintentional poisoning during childhood, making a global health issue (1,2,3). Carbon monoxide (CO) has no color, odor, or taste. But it is very dangerous, you may not even be aware there is a problem in your home until it is too late. CO harming human health at levels exceeding 100 ppm (4). It can cause sudden illness and death. Clinicians must be alert to the signs and symptoms of CO toxicity because it accounts for most of the poisoning deaths in the United States. Approximately 300 to 500 people in the United States die annually from unintentional CO poisoning (5). Due to these characteristics, it has come to be known as the "silent killer"(6).

CO is formed during an incomplete combustion of organic material, for example, gasoline, coal, wood, propane, and natural gas. Common workplace sources of CO include fuel-powered engines (motor vehicles, forklifts, generators, pumps, etc.), fuel-burning heaters (furnaces, water heaters, boilers, space heaters, etc.), coke ovens, and blast furnaces. However, the most common sources of CO are faulty, poorly maintained, or inadequately ventilated gas appliances such as stoves and heaters(7).

CO gas is readily absorbed and is unchanged by the lungs. After absorption, it largely (90%) binds to haemoglobin, and rarely (10%), to myoglobin and cytochrome C-oxidase. Less than 1% is dissolved in plasma, and less than 1% of CO is oxidized to carbon dioxide. Cardiac injury has been associated with hypoxia in human and animal studies, and it has been reported that neurological and perivascular injuries were hypoxic as result of oxidative stress (reoxygenation) secondary to CO exposure. Damage to central nervous system (CNS) as result of hypoxia may lead to cardiovascular insufficiency, and effect of high doses of CO on smooth muscle may result in hypotension (8).

The most common symptoms of CO poisoning are headache, dizziness, weakness, nausea, vomiting, chest pain, confusion. Unfavourable cognitive sequelae (problems with memory, attention or concentration, and affect) can occur immediately after exposure and persist or can be delayed, but they generally occur within 20 days after carbon monoxide poisoning (9,10,11). Cognitive sequelae lasting one month or more (10) appear to occur in 25 to 50 percent of patients with loss of consciousness or with carboxyhemoglobin levels greater than 25 percent (10). It is often hard to tell if someone has CO poisoning, because the symptoms may be like those of other illnesses. People who are sleeping or intoxicated can die from CO poisoning before they have symptoms. The recommended treatment for acute carbon monoxide poisoning is 100 percent normobaric oxygen (9,10). Hyperbaric-oxygen therapy is often recommended for patients with acute carbon monoxide poisoning, especially if they have lost consciousness or have severe poisoning (9,10).

AIM

Aim of this research article is to show the neurological organic imaging changes in the brain due to CO poisoning. Making a diagnosis of CO poisoning on the basis of NCCT Brain with correlation of the patient's history, symptoms and blood analysis. Also to explain necessity of awareness about CO poisoning among the people of rural and tribal regions.

MATERIAL AND METHODS

On 29/04/2020 a one year old male child has brought by his parents to the department of Radiodiagnosis, Dimrapal Hospital, Lt. Baliram Kashyap Memorial Govt. Medical College, Jagdalpur, Bastar, Chhattisgarh, India. Requisition for the NCCT head was recommended by the Paediatrician from the paediatric department of the same hospital. At the time of presentation the patient's condition was semiconscious and had complain of multiple seizure attacks for 4-5 days. On history, in the beginning of the disease the patient had complains of vomiting, fever and loose stool, started a week ago as per his parents. Patient's weight was 6.6 kg. Vitals were recorded as pulse rate 110/min, HR 102 beats/min, respiratory rate 60/min, random blood sugar 230mg/dl and haemoglobin level was decreases about 8 mg/dl. On LFT SGOT was 41 IU/L, slightly raised SGOT 45 IU/L At the time of admission patient's oxygen saturation was 84% however it improves upto 94% by giving hyperbaric oxygen to patient at the rate of 6-8 litres per minute. Patient's ABG analysis has shown keto-acidosis as blood pH was slightly reduced.

NCCT head was performed on a GE EVO 128 slice machine. The study has revealed symmetrical hypodensity in bilateral Globus Pallidus. It represents the anoxic ischemic changes, which is a common associated findings in a patient with carbon monoxide poisoning however rest of the brain study was unremarkable in this case.

DISCUSSION

Carbon monoxide (CO) poisoning is common, potentially fatal, and probably under diagnosed because of its nonspecific clinical presentation. The diagnosis is made when a compatible history and examination correlated with the imaging findings and the with elevated carboxyhaemoglobin levels. The clinical findings of CO poisoning are highly variable and largely nonspecific and laboratory markers alone offer an imperfect assessment of intoxication severity. Brain imaging is usually only undertaken if there are concerns about alternate pathology to explain neurological decompensation (12). The CDC reported that in the United States in 2001-2003, children younger than 4 years had the highest incidence of unintentional CO exposure but the lowest death rates from CO poisoning (13). The risk of death from CO poisoning increases with age. The age-specific death rate is highest for those aged ≥85 years (6.00 deaths per million) and lowest for those aged 5 to 14 years (0.25 deaths per million) [14]. The incidents of CO poisoning usually occur during winter season (15). A study along with other case reports from India (16) and worldwide (17,18,19,20) showed that the main cause of CO poisoning is accidental collection of CO in a nonventilated room/closed space.

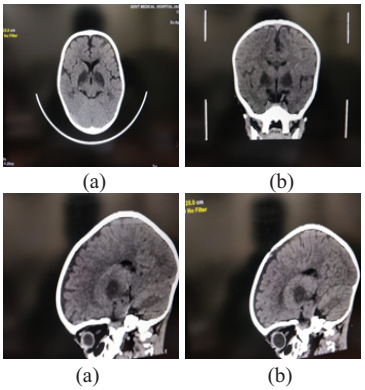
Early symptoms of CO poisoning usually are nonspecific, and nearly

all organ systems can be affected. When CO is inhaled and then absorbed into the bloodstream, it forms carboxyhemoglobin by binding to haemoglobin. CO has an affinity for haemoglobin 250 times that of oxygen. Not only is carboxyhaemoglobin unable to transport oxygen, it also reduces oxygen delivery to the tissues by interfering with the dissociation of oxygen from the remaining oxyhemoglobin. People who have higher metabolic rates and organ systems that have higher oxygen requirements are affected most. Therefore, infants and children are at greater risk, and neurologic, cardiovascular, and pulmonary manifestations are seen most frequently. Foetuses are particularly vulnerable, because maternal CO crosses the placenta(5). CO binds with the haemoglobulin and myoglobin, this is associated with reduced haemoglobin, reduced cardiac output, hypoxia and hypotension which may result in brain ischemia (21), consequently hypoxia leads to lactic acidosis and brain ischemia leads to drowsiness, unconsciousness and seizure.

The globus pallidus is the most common site of involvement in CO poisoning (22,23). The damage usually occurs immediately [23]. The predilection for the globus pallidus is unclear but may be related to the hypotensive effects of CO poisoning in the watershed territory of the arterial supply or to CO binding to the iron-rich globus pallidus [23, 24]. Necrosis of the globus pallidus is not necessarily related to the development of Parkinsonism and vice versa [25], probably because the damage to the nigrostriatal pathway is incomplete. CT usually shows symmetric hypodensity. The caudate nucleus, putamen, and thalamus occasionally are involved in CO poisoning but less so than the globus pallidus. Involvement of the brainstem and cerebellum may be a reflection of more severe poisoning because the posterior structures are more resistant to hypoxia [22]. Demyelination of the cerebral white matter is usually not a feature of the acute stage of CO poisoning [22]. The most commonly involved areas are the periventricular white matter and centrum semiovale [22]. In severe cases, however, demyelination can extend to the subcortical white matter, corpus callosum, and external and internal capsules. CT usually shows diffuse and confluent hypodensity in these areas. White matter demyelination is believed to be responsible for delayed neuropsychiatric syndrome. The most frequent symptoms of delayed neuropsychiatric syndrome are mental deterioration (amnesia, cognitive dysfunction), emotional disorder (depression, anxiety, mutism), urinary and faecal incontinence, and motor disorder (gait disturbance, Parkinson's disease–like symptoms) [26]. Carbon monoxide may be quantitated in blood using spectrophotometric methods or chromatographic techniques in order to confirm a diagnosis of poisoning in a person or to assist in the forensic investigation of a case of fatal exposure. A CO-oximeter can be used to determine carboxyhaemoglobin levels (27).

In our case patient was semiconscious, lab analysis shown raised blood sugar level, decreased haemoglobin and ketoacidosis. NCCT brain shown symmetrical homogenous hypodensity in bilateral globus pallidus however rest of the brain study was unremarkable. On history patient's parents told that they live in a single room house with a poor ventilation. They cook food by burning wood. They are also a local alcohol maker and they burn woods for making alcohol in that single room only which was the reason for the patient to get carbon monoxide exposure.On clinical scenario patient was having complain of vomiting and multiple seizure episodes. On the basis of all above findings along with the clinical history, it was diagnosed as CO poisoning.

Image 1



(a) (b)

(a) (b)

Image 1. NCCT Head (a) Axial, (b) Coronal, (c) sagittal right cerebral hemisphere, (d) sagittal left cerebral hemisphere, all show symmetrical homogenous hypodensity in bilateral globus pallidus.

Administration of 100% oxygen as early as possible is recommended for all patients with a relevant suspected diagnosis (in alert patients, for example, by means of non-invasive continuous airway pressure (CPAP), or respiration using a mask with a demand valve, or administration of 15 L/min O2 through a reservoir mask) (28). In suspected CO poisoning, an early diagnosis has a crucial role in initiating targeted and timely treatment. In principle, the diagnosis of CO poisoning is based on clinical symptoms and suspected or confirmed exposure (29). For the purposes of verification, carboxyhemoglobin (COHb) should be measured in a blood gas analysis (BGA).

In the differential diagnosis of CO poisoning, viral infections of the upper respiratory tract, hypoxic encephalopathy, encephalitis, meningitis, intracranial CNS pathologies, gastroenteritis, drug overdose (sedatives, hypnotics, salicylates), ethanol or methanol intoxication, cyanide intoxication, methemoglobinemia due to opiate use, migraine, hypertension headache, trauma, depression, and other psychiatric disorders should be taken into consideration (30).

CONCLUSIONS

As a result of various pathophysiologic mechanisms, a number of patterns of brain injury can be seen in patients with CO poisoning. The addition of CT/MR imaging to the diagnostic workup of CO-intoxicated patients offers additional information to clinicians to better gauge patient outcome, especially when other parameters are equivocal. Outpatient follow-up is required to assess for the development of Delayed Neuropsychiatric Syndrome (DNS), which can cause personality changes, memory difficulties and gait disturbance (31). CO poisoning should be included in the differential diagnosis in patients found to have cerebellar white matter lesions on imaging. Clinical symptoms, Lab analysis of blood and Imaging (CT/MRI) can make the diagnosis of CO poisoning however carboxyhaemoglobin (COHb) level or CO-oximeter are required for confirmation.

Prevention remains a vital public health issue, requiring public education on the safe operation of appliances, heaters, fireplaces, and internal-combustion engines, as well as increased emphasis on the installation of carbon monoxide detectors. People should be alarmed about the danger of CO collection and poisoning in a nonventilated room while using various room warming mechanisms such as firepots and room heater and by putting warning labels on various CO-producing appliances. They should be taught to avoid using these appliances without proper ventilation in the area to avoid collection of dangerous CO.

Mostly the CO poisoning occur among the people with low socioeconomic status. Albeit CO poisoning can occur in any region but the people specially the tribals, in the bastar division, are very poor socially and economically as well. The schedule tribes live mostly in hills and dense forest area, which are not easily accessible. They are mostly backward, poor, illiterate and indebted. These people possess their own culture, way of life, source of livelihood, religious beliefs, which are quite different from other sections of the Indian communities. In our case the patient has died after few days of admission. There is no other data available to compare CO poisoning in Bastar divison. Infact this case was the first known diagnosed case of CO poisoning in this area. CO poisoning is a fatal disease with the mortality rates range between 1% and 31% (32). Unfortunately in our case the patient has died 10 days after the admission.

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