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Carbon Monoxide Poisoning.

"The Silent Killer"

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ABSTRACT:

Carbon monoxide has been described as a "silent killer" as it does not smell or have any taste or color.

Carbon monoxide toxicity is also something that should be considered in workplace or industrial deaths where machinery is operating in confined or poorly-ventilated spaces. For example, carbon monoxide toxicity was responsible for one third of deaths in a study of 523 fatal occupational inhalations in the United States . Mining had the highest fatality rate followed by firefighting .

Carbon monoxide toxicity continues to account for a significant number of deaths that present for medicolegal evaluation in most countries. (1)

Department of Health in UK in 2011 reporting that over 50 people die annually due to CO poisoning in England and Wales with an estimated 200 people requiring hospital admission.(2)

In line with the literature of the delayed onset of neurological symptoms following CO poisoning, the patient in our case experienced an abrupt development of neurological impairment about two weeks after being exposed to CO poisoning.

Close monitoring of individuals with obstructive lung disease risk factors while they get medical care for carbon monoxide poisoning is recommended .

Conclusion:

Even if COPD has not been officially diagnosed, we advise patients who have had carbon monoxide poisoning and have a history of heavy smoking to undergo routine ABG analyses while receiving treatment to be sure they are not progressing into a potentially fatal respiratory acidosis. Early intubation may be necessary in some situations because carbon dioxide retention in these patients restricts the use of uncontrolled high-flow oxygen. In such cases, hyperbaric oxygen therapy should only be very cautiously considered. Few studies have examined the relationship between smoking and the development of DNS, therefore more research is necessary.

Keywords:
Carbon monoxide, Neurological , Carboxyhemoglobin , Smoker.

Case Report :

A 70-year-old retired man showed up at the emergency room after a house fire. The patient and his family awoke at night when the apartment below them caught fire. Significant smoke inhalation was experienced by the patient and his wife due to the rising smoke levels from the floors and windows. After being rescued by their firemen and receiving high-flow oxygen therapy first from the emergency services, the man was noticeably more lethargic and less alert when he got to the hospital.

He was hypertensive , diabetic (type 2) and had disc prolapse .Bentofluazide, amlodipine, metformin, atorvastatin, and a daily multivitamin were prescribed . He did not have any allergies. The patient had no substantial family history, did not drink, but had a considerable history of smoking for more than fifty years, a minimum of 20 cigarettes a day, and continued to smoke.

Upon assessment, there was no stridor, hoarseness, facial burns, external burns, or singed nose hair. There was also no obvious signs of airway blockage. Nevertheless, carbonaceous sputum was being expectorated by the patient. The first values for blood pressure and pulse oximeter saturations on high-flow oxygen were 139/84 and 74 beats per minute, respectively. With a GCS of 14/15, the patient was alert but sleepy; they lost a point for being obliging. Oxygen was removed, and a subsequent arterial blood gas analysis showed pH 7.4 (NR 7.35–7.45), pO_2 10.1 kPa (NR > 10.6), and pCO_2 of 5.46 kPa (NR 4.6–6.0). High-flow oxygen at 15 litres/minute with a reservoir bag was recommenced as the patient's pulse oximeter saturations fell to 92% on air.

The patient remained getting harder to get up during the next hour, and a second arterial blood gas study revealed a pH of 7.32 and an increased pCO2 of 7.80 kPa. Despite constant oxygen saturations of 94%, the patient experienced no either objective or subjective dyspnea after switching to 0.24 FiO2 regulated flow oxygen. A subsequent arterial blood gas measurement revealed that the pCO2 level had returned to normal.

Aside from it, the blood results were unremarkable. contrasted to his wife, who had an initial COHb level of 11% but no symptomatology or clinical indications, from a COHb level of 9% (NR < 2%).

Four hours later, the patient's COHb level had decreased to 5%, compared to his nonsmoking wife's level of just 1%. After being monitored all night, both patients were

released the next morning, having experienced no immediate negative consequences from the exposure.

15 days of being discharged, the patient started to withdraw more, stopped talking, and started to move in strange, repetitive ways (such carefully crossing and uncrossing her legs and holding a sock in the air for minutes at a time). He was diagnosed with catatonic depression and admitted to a second inpatient mental health facility. His vital signs, chest x-ray, EKG, and the findings of screening testing were all unremarkable. But he was waxy flexible, had strange posture, often grimaced, silent, incontinent both urine and feces, and unresponsive to passive movement.

Brain MRI revealed partial necrosis of the right globus pallidus and new diffuse, bifrontal white matter T2 hyperintensities, which are compatible with anoxic damage from CO poisoning. His mood and social skills showed only slight improvement after taking 10 mg of dextroamphetamine daily. Following a sixteen-day hospital stay during which there was little change in her clinical condition, the patient was moved to a rehabilitation facility to get additional therapy.

Discussion:

Because carbon monoxide is colorless, tasteless, odorless, and non-irritating, an individual who comes in contact with it cannot detect it. CO is easily taken up by the lungs and enters the circulatory system, where it combines with hemoglobin (Hb) to create carboxyhemoglobin (COHb), a tightly bound, slowly reversible complex. The blood's ability to carry oxygen is reduced when COHb is present, which lowers oxygen availability to tissues in the body and causes tissue hypoxia.

Cellular oxidative metabolism may be hampered by a decrease in oxygen supply brought on by the elevated COHb level and worsened by reduced perfusion from hypoxic heart failure.

The concentration and length of exposure to CO affect the health concerns it poses. At low concentrations, the consequences range from mild effects on the heart and nervous system to death and loss of consciousness following extended or acute exposure to high levels of CO . Numerous outstanding papers have examined the risks connected to the comparatively low ambient amounts observed in the environment and in contaminated workplaces. [3]

The tight bond between CO and hemoglobin is caused by the significantly higher affinity of CO for hemoglobin than oxygen does. Though CO is only slowly replaced by oxygen, this bond is breakable and reversible. In addition, CO binds to myoglobin in the skeletal muscle and heart, impairing tissue oxygen transport. This causes cardiac dysfunction in the heart. By preventing the action of some enzymes, such cytochrome c oxidase, it also has direct impacts. Therefore, cardiac and neurological function deterioration may potentially be linked to CO poisoning.

The average level of CO-Hb in healthy, non-smoking individuals is less than 2%, and in smokers, less than 15%. Neurological symptoms, such as headache, nausea, and dizziness, are observed at CO-Hb levels over 10%, while increases in respiratory and heart rates, syncope, motor paralysis, and confusion are observed at CO-Hb levels of 30–50%. CO-Hb levels exceeding 50% can be fatal, so values in this range are central to the diagnosis of CO-poisoning. This explains the variation in CO-Hb levels between the smoker patient and his wife.

Headache, lightheadedness, exhaustion, and nausea are examples of nonspecific signs and symptoms. In clinical practice, differential diagnoses such as schizophrenia, cerebral metastases of tumor, stroke, and coagulation problems are necessary because behavioral changes such agitation, disorientation, and hallucinations might occasionally be noticed.[(4)]

Hyperbaric oxygen therapy is the gold standard treatment for acute CO poisoning. In this instance, a COPD patient who is susceptible to retaining carbon dioxide would undoubtedly induce a considerable increase of pCO2, as this significantly boosts the arterial oxygen level. Furthermore, hyperbaric oxygen therapy is somewhat contraindicated for COPD patients due to the possibility of emphysematous bullae rupture at high pressures.

Similar to what happened in this case, CO poisoning can result in delayed neuropsychiatric sequelae (DNS), which is characterized as brain damage that manifests itself a few days to a week after recovery from hypoxic injury. Many symptoms, including mobility disorders, mood disorders, and/or memory disorders, are indicative of DNS. Prior research has shown that prolonged CO exposure and deliberate suicide attempts have an impact on the development of DNS and have resulted in suboptimal neurologic

consequences. Studies on the relation between smoking and the development of DNS related to CO poisoning, however, are limited.[5]

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