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A Review on Toxicity of Carbon Monoxide on Human

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Abstract

Carbon monoxide toxicity becomes a momentous health issue in USA and many other countries. This toxicity causes severe morbidity and mortality and its symptoms resemble with other illness. Source of CO include, second hand smoke, fumes from heating systems burning fuels, exhaust fumes from motor vehicles. One of the most frequent sources of CO is gas from the exhaust of vehicles. CO poisoning should be considered when two or more patients are instantaneously sick. CO toxicity is not due to reduced oxygen carrying capacity of blood. Its symptoms are non-specific and may range from nausea and headache to intense central nervous system dysfunction. People may die even in low level of carbon monoxide without involving other toxic agents. This review will be helpful to understand the CO toxicity and also would be capable to minimize the CO toxicity.

Keywords: Toxic; Carbon monoxide; Haldane effect; Oxyhemoglobin

Introduction

Carbon Monoxide (CO) is a toxic gas. It has molecular structure which is produced when Incomplete Carbon combustion of carbonaceous material such as kerosene, coal, propane and gasoline take place. It is colorless, tasteless, odorless, and non-irritating has structure that's why it has been known as "Silent Killer" [1-3]. It can cause unconsciousness and even death of someone within minute. It also has been known as "great mimicker". It has low- density structure therefore it can easily diffuse. And even when no smoke and fire is present in environment it can exist [4].

The amount at which a substance (a toxin or poison) can harm humans and animals. Acute toxicity can harm an organism through short term contact. A toxic substance that cause effects for more than one year but less than the life time of exposed organism is known as sub chronic [5]. Chronic toxicity is the ability of a substance to cause dangerous effects over a prolonged period, usually when exposure is continuous, sometimes lasting for the entire life of the exposed organism.

Toxicants are compounds which cause toxicity and these are very harmful for human health even causes death. There are four major types of toxicants: Chemical, Biological, Physical, and Radiation. CO on average is 50 to 120 parts per million(ppm) in the environment. The quantity of CO in the northern hemisphere is more than in the southern hemisphere. The level of CO depends on the season and fluctuates with season. In summer its level is lower [6,7]. CO produce from human activities like incomplete burning of fuel etc, 60% of this CO is present in troposphere. The atmospheric pollution, occupational exposure, smoking and second hand smoke are source of CO. One of the most frequent sources of CO is gas from the exhaust of vehicles. Average concentration of CO inside the car is between 9 and 25 parts per minute. Environmental CO exposure may be higher in urban areas; typically it is less than 0.001%, or 10 parts per minute (ppm).
CO absorption by body depends on minute ventilation, concentration of CO, oxygen in the environment and duration of exposure. The indoor concentration of CO rise to 100 parts per minute due to gas stove. An estimated 400 to 500 parts per minute (ppm). CO is exposed to cigarette smoker during smoking. Exhaust of automobiles contain 10% (100,000 ppm) of CO. When the exposure of CO is 70 parts per minute (ppm) then the level of carboxyhemoglobin (CO-Hgb) at equilibrium is 10%. And the level of carboxyhemoglobin (CO -Hgb) at equilibrium is 40% at exposure to 350 parts per minute (ppm). The Occupational safety & health administration permit the certain level of CO exposure in workers over an 8-hours work day, which is averaged 50 ppm. CO poisoning has been reported in childrens which ride on the back of pickup trucks, recreational boaters, factory workers operating propane -powered forklifts, and persons in an ice skating ring using propane - powered resurfacing machines. In the cases of recreational boaters swimming underneath the swim platform near the boat exhaust and campers using gas-powered stoves in outdoor tents fatalities are reported. If we misuse the burning charcoal briquettes or a gas stove, in the winter for the purpose of heating then it is predictive of high level of CO-Hgb. Methylene chloride is a source of CO, which is converted to CO by liver. Methylene chloride is a solvent found in paint remover and aerosol propellants. During heme catabolism by hemeoxygenase take place, the endogenous production of CO occurs but the level of CO-Hgb in excess of 1% should not produce. However, CO-Hgb may increase 3% to 4% in hemolytic anemia [8]. When sepsis is severe then it elevates endogenous CO production. When sepsis is severe then it elevates the endogenous production of CO. At fatal level of CO, CO-Hgb not exist with its 50% intensive level. Toxicity of CO not only reduces the capacity of blood to carry oxygen. A person may be dying purely of CO, affected with 25% CO-Hgb.

A patient after suicide attempt with automobiles exhaust or from a house fire may not show a diagnostic dilemma. However,many patients or families with the symptoms of nausea, vomiting,headache etc these can easily diagnosed but doctors discharge them back to dangerous environment where they may suffer due to serious exposure.

Pathophysiology

In start it was thought that Pathophysiology of CO poisoning is due to exclusively to the cellular hypoxia which is imposed by modifying oxyhemoglobin with CO -Hgb and it also cause a relative anemia. CO with an affinity more than 200 times that of oxygen bind to hemoglobin. It decreases the assignment of oxygen to the tissues and cause tissue hypoxia. CO enters in body through lungs and bind with oxygen carrying hemoglobin. The oxygen carrying capacity of hemoglobin is decreases by binding the Fe+2 atoms of hemoglobin 200-250 times. The increase in affinity of CO to oxygen and decrease in the oxygen binding capacity of hemoglobin, called "Haldane Effect" [9]. Basically CO toxicity is a situation which is characterized by spoil oxygen transport and usage. CO bind with intracellular proteins (cytochrome-C, oxidase, and myoglobin) production of ATP decreases because mitochondrial functions disturbed and oxidative phosphorylation violate and tissue toxicity characterized by lactic acid, cause cell death. When CO binds to cytochrome a3; the blockage of oxidative metabolism causes production of oxygen free radicle. Cellular respiration may be spoil by inactivation of mitochondrial enzymes and also electron transport from oxygen redicals which are produced when CO is exposed. After normalization of CO -Hgb levels cellular spoil energy metabolism is inhibited which may explain the prolonged clinical influence after decrease of CO levels. The availability of oxygen in the heart and cause arrhythmias and dysfunction of heart due to CO binding with myoglobin also cause direct skeletal muscles toxicity and rhabdomyolysis. CO excites Guanylyl cyclase, due to this cyclic guanylyl monophosphate increases and resulting in cerebral vasodilation, which has been associated with loss of consciousness in an animal model of CO poisoning [10].

Neuronal necrosis or apoptotic cell death develops in acute CO intoxication. This process is accountable for some initial neurological abnormalities. In acute CO intoxication, Pathophysiological changes has been manoeuvre :explained by four ischemic nerve damage due to hypoxia induced by CO,direct peripheral nerve damage due to high level of CO,existence of petechial hemorrhage in peripheral nerves as in other organs,development of venous obstructive due to local edema and circulation impair [11,12]. CO causes release of" Nitric oxide (NO) "from endothelial cells and thrombocytes and outcome in the pro inflammatory reciprocate which develops due to the more formation of NO derivative oxidants that leads to endothelial damage and leukocytes sequestration, such as peroxynitrite.

NO may also influence the adherence of neutophils to the endothelium, by affecting the function of neutrophils adhesion molecule like B2-integrin. Adherence of neutrophil to the microvasculature appears to lead to Xanthine oxidase activation, oxidative radical formation, and oxidative damage and brain lipids peroxidation which are thought to be responsible for clinical syndrome of DNS. The damage caused by CO toxicity depends on its concentration, exposure interval and health condition of patient.

Effects

The effect of CO poisoning are diverse and can easily confused with other illnesses, such as nonspecific viral illness, benign headache, cardiovascular, neurological syndromes.

Signs and symptoms associated with CO poisoning: Headache, Confusion, Palpitation, Nausea, syncope, hypotension, Vomiting, chest pain, dysrhythmias, Dizziness, dyspnea, cardiac arrest, Blurred vision, weakness seizures, Tachycardia, Coma, Tachypnea, Myocardial ischemia.

As exposure of CO increases, patient show more severe symptoms with Brian and heart (oxygen dependent organ) show the rapid symptoms of injury. Early neurological symptoms are dizziness and headache. More exposure may produce modify mental status, confusion, syncope, seizure, acute stroke -like syndromes and coma. Central nervous system structural damage correlates with systemic hypotension due to CO poisoning.

The effect of CO is not restrained to the early period after exposure. Insistent or delayed neurological effects have been reported. Most interesting is a syndrome of clear recovery from acute CO poisoning followed by behavioral and neurological deterioration after a period of 2-40 days. This is often quoted as DNS, any neurological or psychiatric symptoms may manifest like memory loss, confusion, ataxia, seizures, urinary and fecal uncontrolled, emotional liability, disorientation, hallucinations, parkinsonism, mutism, cortical blindness, psychosis, gait and other motor disturbances [13]. The actual prevalence is difficult to determine of DNS, after CO poisoning the estimate ranging from less than 1%-47% of patient. In prevalence the big variability is at least partially explained, in defining DNS using clinical, subclinical, self -reported, combination criteria by a lack of consistency.

Different definitions are used by investigators of DNS; term may refer clinical signs, neuropsychomatric test abnormalities, or a combination of the two. To define DNS using total neurologic abnormalities may underestimate correct cognitive dysfunction; neuropsychometric testing may show subclinical and probably tentative cognitive dysfunction of anonymous clinical and prognostic significance. Abnormalities may be detected from neuropsychometric testing and patients may perform poorly on these tests, which are really ill, suicidal, depressed, or have congestion of other toxucants. Additionally these patients have no baseline for comparison. Neuropsychomatric testing supplies an objective measure of cognitive function that can be used to picture and follow CO poisoned patient.

Some writers hypothesized that chronic CO poisoning cause more morbidly and mortality than the currently recognized, partially because of the inherent difficulties in quantifying both degree of exposure and degree of neurologic impairment, the evidence to substantiate these claims is less than compelling. Case report that have been published describe a syndrome of headache, nausea, lightheadedness, cerebral dysfunction and cognitive and mood disorder in association with chronic, CO exposure at low-level. These all reports have uncontrolled confounding factors and data related to exposure is short. These symptoms usually finish once the patient is removed from the environment. Additionally chronic CO exposure has been linked with polycythemia and cardiomegaly, perhaps due to chronic hypoxia [14].

Professional hearing loss is very important health problem in our society. Scientific publications have discussed the many dangerous effects of noise exposure on hearing and on health, in past years. Besides the presence of noise in the workplace, other environmental factors especially CO united with noise present a possible hearing loss risk, even under condition of low noise exposure. The studies of CO exposure on auditory system of human were directed using acute exposure to CO. In these cases noise exposure is not an important factor for auditory problems.

Consequences of research on the toxic action of CO on the auditory system show that CO have direct influence on the cochlear use, various

biochemical and electrochemical constituent seems to be influenced in the same manner. Result also show the effects on the compound action potential produced by the auditory nerve fibers that are more related in comparison to the influences on the endococholear potential maintained by the stria vascularis and the effects of potential of the cochlear microphonic created by the external hair cells.

Intoxication of CO

CO intoxication shows regional variations and seasonal. CO is main source which is used for indoor heating that's why it is cause of more than 80% deaths. 14. 000 to 40. 000 cases detected in USA per year due to intoxication of CO From 1999 to 2003, average 439 deaths have been reported per year. In this regard, CO intoxication is the third in USA in the list of accidental deaths. Due to CO intoxication 5000 to 8000 patient per year admit to the hospitals in France, while in Italy from 1993 to 1994, this number was 6000. Within the same period deaths was more than 300. The value of subject rises because 1/3 of CO intoxication is not diagnosed.

Diagnosis

The diagnosis of CO poisoning is a clinical one. For diagnosis symptoms are required, but in CO poisoning no single symptom is either sensitive or specific. The most common symptoms are headache, dizziness, nausea, vomiting, confusion, fatigue, chest pain, shortness of breath, and loss of consciousness. A diagnosis of CO has no combination of symptoms at either confirm or excludes. There is no specific headache pattern typical CO poisoning, while headache is the most common symptom. The air, mixture of nitrogen and oxygen is slightly heavier than CO; gases diffuse rapidly through out an area so CO alarms can easily install [15].

In making the diagnosis of ocular co toxicity, a high index of suspicion is essential. Since myocardial damage development has been detected in 37% of patients intoxicated by CO. ECG and measurement of cardiac biomarkers in blood are essential in cases of serious CO intoxication. In patients intoxicated by CO with elevated cardiac enzymes and ECG changes, increased mortality rates have been found in the following few years, despite aggressive treatment. It has been observed that angina attacks increase in patients with chronic angina with a mild increase in the COHb levels, and it is possible to observe arrhythmias in patients with coronary artery disease when the level of COHb increases to 6%, and it was shown that in cases with severe CO toxicity ischemia can develop even if the patients have normal coronary arteries [16].

Prevention

CO has been common during storm-related power outages, while people turn to the indoor use of charcoal briquettes for cooking and heating, improper use of gasoline-powered electrical generators to supply electricity, and indoor use of gasoline powered pressure washer to clean up.

CO poisoning can be prevent through the use of CO significant alarms. The extensive practice of catalytic converters on automobiles and better. Emissions policies have resulted in a significant decline in accidental deaths due to CO poisoning [17-20]. Avoidance of high indoor concentrations of CO is Optimal and can be accomplished by frequent inspection and maintenance of furnaces, stoves, and fireplaces, avoidance of indoor unvented combustion sources such as net opportunities). A CO alarm in every house are recommend for disease control and prevention. They must be installed in the hallway outside the sleeping areas [15]. grills and space heaters, careful use of gas stoves, and installation of CO detectors. In the United States, CO alarms are designed to activate within 189 minutes of 70 ppm exposure, 50 minutes of 150 ppm exposure, or 15 minutes of 400 ppm exposure Although the effectiveness of CO detectors may be limited in the significant proportion of victims of fatal CO poisoning who succumb while asleep or under the influence of alcohol, appropriate and widespread use is likely to decrease the incidence of occult indoor CO poisoning.

Treatment

Treatment of co poisoning starts with supplemental oxygen and aggressive supportive care, airway administration, blood pressure support and stabilization of cardiovascular status. When occult toxicity of CO is known patients survive at the scene and warned and empty until the source is known, environment is protected [21-23].

In the treatment of CO poisoning, high flow oxygen by mask or endotracheal tube is frontline. O2 enhances the deduction of COHb, alleviates tissue hypoxia as compare to air.

However, It is known that no clinical experiment have done superior efficacy of norm baric 100% oxygen over air. By adding CO2 to O2 for spontaneously inhaling individuals was advocated immediately COHb removal enhances alveolar ventilation. At ambient temperature 100% O2 supply 2.09% vol done third of body's needs, 5.62% vol is provided by absolute 2.5 atm. In porcine study to supply enough dissolved O2 to provide the body's requirements in the near absence of hemoglobin, HBOT at 3. 0 ATA was look.

The half-life of CO-Hgb reduces when partial pressure of O2 Increase. The half-life of CO-Hgb is 240-320 minutes at room air, at 100% O2 40 to 80 minutes, approximately 20 minutes at 100% HBOT ATV 2.5 to 3.0 ATA. Long-term exposures may result in prolonged half-life and wide individual changes exists [24-26].

Prognosis

Generally, the CO intoxication cases are non-fatal. The patients of co intoxication can be discharged in 4-6 hours if symptoms are mild; if they have normal neurological signs and not need any therapy. But, it is still impossible to predict the long term fallout of co intoxication even with therapy [27-30].

Therapy in CO intoxication

FiO2 should be continued at the highest possible concentration, even if there is no smoke breathing associated different pulmonary damage in CO intoxication [30-32]. There is close relationship between elimination half-life of COHb and the PaO2 formation by FiO2. Half-life of COHb is 240-320 min at room air while it is 40 to 80 min with 100% oxygen at 2.5-3 atmosphere pressure [33-35]. Although HBO therapy is widely used today, there are no standard regarding the therapy time or frequency. However, a session of HBO at 2.5-3 atm is applied to the patient who meets the criteria. Additional sessions are evaluated considering the clinical findings and the elimination of the symptoms. For the patients who do not meet the criteria or application of HBO therapy is not possible, high flow of 100% oxygen is administered with a tight-fitting mask for 6-12 hours [36-40].

Several authors highlighted the dependability of HBO therapy. Worry and barotrauma to middle ear and sinuses which are seen in 0%–80% of the patients who have had HBO therapy are among the most common problems [41-43]. Seizures, oxygen toxicity, pulmonary edema and pulmonary bleeding, pneumothorax and air embolism are among the less often complications [44-46]. The single complete contraindication for HBO therapy is the unprocessed pneumothorax Claustrophobia, otosclerosis or other middle ear diseases, intestinal obstruction, chronic obstructive lung disease with bulla formation or the need to certain processes like aspiration, defibrillation, cardioversion and intubation are among the relative contraindications. It should be kept in mind that minutes are wanted for carefully of the patient [47-50].

Conclusion

This study concluded that carbon monoxide toxicity is very dangerous and even cause death of humans within minutes. Carbon monoxide cannot be controlled but its exposure can be minimized in this way the rate of mortality and morbidly due to CO toxicity would be decreases. In vehicles CO free fuel would be used instead of CO rich fuel. Car would not run in garage of home even home gate is open. Homes would not be heated with gas stove. Don't burn anything in gas stove that is not vented. Don't use generator, charcoal grill and other gasoline burning devices in house. To minimize the exposure of CO, alarms and detector would be used, if detectors or alarm ever sound immediately call 911. Heating system, water heater and other appliances should be serviced by qualified technician every year.

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