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CARBON MONOXIDE POISONING

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I**ntroduction:** Carbon monoxide is a dangerous gas as excess inhalation of it can result in death. CO was used by Greeks and Romans for the execution of criminals. Carbon monoxide toxicity accounts for more deaths when compared to toxicity from other gases.

Sources of Carbon monoxide: Common sources for carbon monoxide are exhausts from gasoline engines, coal mines, gases from guns, deep wells and underground drainage system.

Toxic effects of CO poisoning: Carbon monoxide is a toxic gas because it readily displaces oxygen from hemoglobin by binding to the same site in hemoglobin for oxygen. So, oxygen transport and oxygen carrying capacity of the blood decreases, significantly. Hemoglobin has got 20 times more affinity for carbon monoxide than for oxygen. So, even when CO partial pressure in alveoli is only 0.4 mm Hg. nearly 50% of hemoglobin is saturated with it. It can get gravely severe if the partial pressure increases to 0.6 mm Hg, (1/1000 of volume concentration in air) in the alveolar air. Presence of carboxyhemoglobin decreases the release of oxygen from hemoglobin and the oxygen-hemoglobin dissociation curve shifts to left. It is still more dangerous because, during carbon monoxide poisoning, the partial pressure of oxygen in blood may be normal despite low oxygen content in the blood. So, the regular feedback stimulation of respiratory centre by hypoxia does not take place because of normal partial pressure of oxygen. The low oxygen content in blood affects the brain, resulting in unconsciousness. The condition becomes fatal if immediate treatment is not given. Additionally, CO is toxic to the cytochrome system in cells, affecting the cell survival.

Symptoms of CO poisoning: Symptoms depend upon CO content of blood. While breathing air with 1% of CO, saturation of hemoglobin with CO becomes 15% to 20%. Mild symptoms like headache and nausea appear. When breathing air containing carbon monoxide more than 1%, the saturation becomes 30% to 40%. It causes convulsions, cardiorespiratory arrest, loss of consciousness and coma. When haemoglobin saturation is above 50%, death occurs.

Influence of CO on different systems of the body:

Effect of CO on respiration: The ability of respiratory system to distribute O_2 around the body decreases because CO decreases the O_2 content of blood and deprives the blood cells of O_2.

Effect of CO on nervous system: High CO-Hb concentrations are treated poisonous for central nervous system (CNS). The effects of CO poisoning includes confusion, dizziness, giddiness, disorientation, nausea, vomition, weakness and may eventually lead to coma.

Effect of CO on cardiovascular system: Exposure of CO on cardio vascular system (CVS)

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leads to fall in oxygen- carrying capacity of blood in addition to reducing venous as well as tissue oxygen tension.

Effect of CO on health: CO inhalation is dangerous as CO displaces oxygen in the blood and causes O_2 deprivation of the brain, heart and other vital organs with O_2 requirement for optimal functions. Large amounts of CO causes suffocation and loss of consciousness.

Effect of CO on lungs: CO is an odourless, colourless and tasteless gas and CO is treated as toxic agent. Breathing CO decreases the ability of blood to carry oxygen.

Production of CO by human body: CO is produced in the human body as natural product of hemoprotein turnover. Most of the CO is originated particularly from circulating hemoglobin. Under physiological conditions, the rate of endogenous CO production occurs at the rate of 18 micro-mole/hour.

Pathogenesis of CO toxicity

1. Mitochondrial inhibition and Free radical generation: CO shows its influence regarding inhibition on mitochondrial respiration by binding the ferrous heme A3 in the active site of CO and effectively shuts downs oxidative phosphorylation, equal to the effects of cyanide and nitric oxide (NO). Because of competitive binding of O2 and CO to cyclooxygenase (COX), CO mediated mitochondrial inhibition is greatest particularly under hypoxic conditions. Because of COX inhibition, oxidative phosphorylation is decreased and reduces ATP production in heart and other aerobic tissues, as important complexes of electron transport chain shuttle electrons continuously and produce superoxide anions damaging, cells and tissues.

2. Platelet and Inflammatory Effects: Excess CO stimulates platelets by displacement of NO from platelet surface hemoprotein. Displaced free NO reacts with superoxide to produce peroxynitrite and inhibits mitochondrial activity and enhances platelet activation. Activated platelets activate neutrophils to degranulate and release myeloporoxidase (MPO). MPO intensifies the inflammatory effect by triggering more neutrophil activation and degranulation. Proteases from neutrophils oxidizes endothelial cell xanthine dehydrogenase to xanthine oxidase and generate reactive oxygen species (ROS).

3. Mechanisms of Brain ischemia:

CO mediated reductions in oxygen delivery and mitochondrial oxidative phosphorylation cause ischemia, as well as anoxic brain injury and lead to the occurrence of cognitive deficits in survivors. Brain injury from ischemia happens from acidosis, excitotoxicity, depolarization, ionic imbalance, oxidative stress, inflammation and apoptosis.

Diagnosis of CO toxicity: CO poisoning may be ideally diagnosed by a clinically important triad of symptoms that are consistent with

a) CO Poisoning; b) History of recent CO exposure and c) Elevated CO-Hb levels.

Treatment for CO poisoning includes the following options:

A. Immediate removal of the subject from the source of exposure to CO

B. Providing adequate ventilation as well as artificial respiration.

C. Administration of 100% oxygen gas to facilitate replacement of CO in the blood

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D. Administration of air with few percent of co_2 to activate the respiratory centre so that ventilation is enhanced

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