Complications of Carbon monoxide poisoning

An Essay

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Abstract

Carbon monoxide is the most common and serious by-product of combustion and responsible for smoke-related morbidity and mortality.

As it is colorless, odorless, tasteless and non-irritating gas, the exposed person is usually unaware of its effect until serious disorders occur.

The main clinical magnifications of acute CO poisoning consist of symptoms caused by alterations of the cardiovascular system such as initial tachycardia and hypertension, and CNS symptoms such as headache, dizziness, paresis, convulsions and unconsciousness.

Carbon monoxide poisoning also produces myocardial ischemia, atrial fibrillation, pneumonia, pulmonary edema, erythrocytosis, leucocytosis, hyperglycemia, muscle necrosis, acute renal failure, skin lesions and changes in perception of the visual and auditory systems.

Of considerable clinical interest, severe neurological manifestations may occur days or weeks after acute CO poisoning. Delayed sequelae of CO poisoning are not rare, usually occur in middle or older, and are clinically characterized by symptom triad of mental deterioration, urinary incontinence and gait disturbance.

Controversy is present as regard hyperbaric oxygen as the main standard treatment of CO poisoning thus prevention remains a vital public health issue, requiring public education on the safe operation of appliances, heaters, fireplaces, and internal-combustion engines

Key ward :-

- ***Complications***
- ***Carbon monoxide***

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Introduction

Carbon monoxide poisoning occurs after the inhalation of Carbon monoxide (CO) is a product of combustion of organic matter under conditions of restricted oxygen supply, which prevents complete oxidation to carbon dioxide (CO2). Carbon monoxide is colorless, odorless, tasteless, and non-irritating, making it difficult for people to detect **(Tomaszewski .,2002)**.

Common sources of CO that may lead to poisoning include house fires, furnaces or heaters, wood-burning stoves, motor vehicle exhaust, and propane-fueled equipment such as portable camping stoves, ice resurfaces, forklifts, and engine-driven generators. CO poisoning can also occur in scuba diving due to faulty or badly sited diving air compressors. Another source is exposure to the organic solvent methylene chloride, which is metabolized to CO by the body **(Raub et al., 2000)**.

Carbon monoxide is a significantly toxic gas with poisoning being the most common type of fatal poisoning in many countries. Symptoms of mild poisoning include headaches and flu-like effects; larger exposures can lead to significant toxicity of the central nervous system and heart. Following poisoning, long-term sequelae often occur. Carbon monoxide can also have severe effects on the fetus of a pregnant woman. The mechanisms by which carbon monoxide produces toxic effects are not yet fully understood, but hemoglobin, myoglobin, and mitochondrial cytochrome oxidase are thought to be compromised. Treatment largely consists of administering 100% oxygen or hyperbaric oxygen therapy, although the optimum treatment remains controversial **(Christian, 2002)**

Toxicity is also increased by several factors, including: increased activity and rate of ventilation, pre-existing cerebral or cardiovascular disease, reduced cardiac output, anemia or other hematological disorders, decreased barometric pressure, and high metabolic rate **(Satran et al.,2005)**

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 monoxi0de detectors **(Yoon et al., 1988)**.

Aim of the work

To discuss all the complications of carbon monoxide poisoning, their effects on various body systems, Sequalee of these complications and how to deal with each one of them.

physical properities, Sources AND EPIDEMOLOGy of Carbon Monoxide

CO is a product of the incomplete combustion of hydrocarbons. The concentration of CO in the atmosphere is usually < 0.001%. The levels are higher in urban areas than in rural areas **(Ernst & Zibrak, 1998).**

It has a molecular mass of 28.01 Daltons. It has a density of 0.968 relative to air. Its absorption through the lungs depends on the duration of exposure, the concentration of CO in the environment and the alveolar ventilation rate about 85% of absorbed CO combines with hemoglobin (Hb) while the remainder attaches to myoglobin and other blood proteins **(Baek et al., 1999)**.

It is a colorless, odorless, non irritat gas produced primarily as a result of incomplete combustion of any carbonaceous fossil fuel **(Hampson, 1998)**.

It is the leading cause of poisoning mortality in the United States and may be responsible for $>^{1}/_{2}$ of all fatal poisonings worldwide. An estimated 5000 - 6000 people die in the United States (US) each year as a result of CO exposure **(Tomaszewski, 2002)**.

The Centers for Disease Control and Prevention reported that from 1968 - 1998, non–related CO poisoning caused 116,703 deaths, 70.6% of which were due to motor vehicle exhaust, and 29% were unintentional**.** The rate of accidental deaths seems to have declined from 1513/year in 1979 to approximately 500 to 600 per year in the 1990s, likely owing to improved

motor vehicle emissions policies and the use of catalytic converters **(Mott et al., 2002)**.

Although most accidental deaths are due to house fires and automobile exhaust, consumer products contribute to approximately 180-200 annual deaths **(Kao & Nanagas, 2004)**.

The US Consumer Product Safety Commission summarized the 180 unintentional consumer product–related, non–related CO deaths in 1998 as being associated with indoor heating systems (71%), stoves and other appliances (10%), charcoal grills (9%), camp stoves (6%) and water heaters (4%) **(Mah, 1998)**.

Patients > 65 years, men and ethanol-intoxicated patients seem to be at higher risk of dying as a result of fatal, unintentional, non–related CO poisoning **(Yoon et al., 1998)**. Unintentional deaths peak in the winter months, as heating systems are being used and windows are closed **(Cobb & Etzel, 1991)**.

Sources of Carbon monoxide:

Endogenous Sources of carbon monoxide:

Carbon monoxide gas is endogenously produced as a byproduct of Hb degradation. It forms a resilient heme ligand and approximately 1% of Hb's oxygen binding sites are blocked by CO from endogenous sources even in absence of air pollution **(Omaye, 2002)**.

Table (1): Common environmental sources of CO **(Omaye, 2002).**

Propane engines	Radiant heaters	Portable generators
Kerosene heaters	Gas fireplaces	Natural gas appliances
Motor vehicle exhaust	Fires	Paint
Hibachi cookers	Tobacco smoke	

It arises in biological systems during the oxidative catabolism of heme by the heme oxygenase enzymes. It plays a central role in many aspects of human health and disease including regulation of blood pressure, maintenance of organ specific vascular tone, neurotransmission, stress response, platelet activation and smooth muscle relaxation **(Morse and Sethi, 2002)**.

Carbon monoxide is emerged during heme degradation as an endogenously produced gaseous mediator, like nitric oxide (NO) and can activate guanylate cyclase by binding to the heme moiety of the enzyme stimulating the production of cyclic 3, 5 guanosine monophosphate (cGMP) formed from guanosine triphosphate (GTP) (Fig.1) **(Ryter et al., 2002)**.

The biphasic effects of CO might be an example of hormesis in action. Hormesis is the phenomenon where a substance has been found to exhibit beneficial effects at a low or very low concentrations in contrast to detrimental responses found at higher concentrations **(Calabrese and Baldwin, 1999)**.