Introduction

Carbon monoxide (CO) is tasteless, odorless, colorless and non-irritating gas. It is the product of incomplete combustion of hydrocarbons ***(Abelsohn et al., 2002).***

Carbon monoxide is associated with high incidence of severe morbidity and mortality. Epidemics of CO poisoning commonly occur during winter months ***(Varon et al., 1999)***.

Carbon monoxide is a product of the incomplete combustion of carbon - based fuels and substances. Human toxicity is often overlooked because CO is tasteless and odorless and its clinical symptoms and signs are non specific ***(Omaye, 2002)***.

Exposure to toxic amounts of CO occurs most often during inhalation of automobile exhaust or smoke, resulting either from faulty heating systems or industrial accidents ***(Piantadosi, 1999).***

Carbon monoxide is readily absorbed through the lungs and rapidly binds to hemoglobin (Hb). The brain and the heart may be severly affected after CO exposure with carboxyhemoglobin (COHB) levels exceeding 20%. Hypoxic brain damage predominates in the cerebral cortex, cerebral white matter and basal ganglia, especially in the globus pallidus. ***(Prockop and Chichkova, 2007).***

Early neurological manifestations include dizziness and headache. Increasing exposure may produce altered mental status, confusion, syncope, seizure, acute stroke like syndromes, and coma ***(Silver et al., 1999).***

Early cardiovascular effects of CO poisoning are manifested as a response to hypoxia. More significant exposures result in hypotension,

dysrythmia, ischemia, infarction, and, in extreme cases, cardiac arrest. Early deaths after CO exposure may be due to cardiac dysrythmia ***(Yanir et al., 2002).***

Carbon monoxide may cause shortness of breath, dyspnea on exertion and tachypnea are common lung examination is almost free, but in severe CO intoxication respiratory depression, pulmonary edema and hemorrhage may be seen secondary to left ventricular dysfunction or due to direct CO effect on the lung parenchyma ***(Piantadosi, 2002).***

The diagnosis of CO poisoning is often suggested by the circumstances in which the patient is found**.** Serum COHb levels should be obtained from patients suspected to CO exposure ***(Kao and Nanagas, 2004).***

High concentration oxygen should be delivered through a tight – fitting, non rebreathing, reservoir face mask or an endotracheal tube ***(Hand and Raffin, 1990).***

Hyperbaric oxygen benefits the brain more than normobaric oxygen by, e.g.: improving energy metabolism, preventing lipid peroxidation and decreasing neutrphil adherence ***(Stoller, 2007)***.