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Evaluation of Clinical Symptoms in Carbon Monoxide Poisoning with Biochemical Parameters

Keywords: **carboxyhemoglobin, carbon monoxide poisoning, pathogenesis**

Karbonmonoksit Zehirlenmelerinde Klinik Semptomların Biyokimyasal Parametrelerle Değerlendirilmesi

Anahtar Kelimeler: karboksihemoglobin, karbon monoksit zehirlenmesi, patogenez

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Dear editor,

We have read the article titled "Evaluation of Clinical Symptoms in Carbon Monoxide Poisoning with Biochemical Parameters" prepared by Çiftci et al. with great interest (1). We thank the authors for this informative and successful manuscript about carbon monoxide (CO) poisoning. We also would like to mention an important point about sonographic pathogenesis of tissue damage in carbon monoxide poisoning.

The binding of CO with respiratory pigments such as hemoglobin and myoglobin and enzymes such as cytochrome oxidase, nitric oxide synthetase and causing direct cellular damage forms the basis of toxic effects (2).

The affinity of hemoglobin to CO is 200 times greater than that of oxygen. The greater the affinity of fetal hemoglobin leads to a more serious fetal toxicity. About 85% of CO is bound to hemoglobin to form COHb, the rest is either soluble in plasma or intracellular, often bound to myoglobin (2). COHb composition shifts the normal oxyhemoglobin dissociation curve to the left and oxygen release to tissues decreases (2,3). Decreased oxygen level is perceived by the central nervous system and there is more ventilation. This leads to more CO intake and respiratory alkalosis (2).

Myoglobin, another heme protein, tends to bind to CO 60 times more than that of oxygen. CO also binds to cardiac myoglobin 3 times more than skeletal muscle myoglobin (3). The formation of carboxymyoglobin also causes a left-shifted oxygen dissociation (3). As a result, oxygen use in the muscles is impaired. Significantly binding of CO to myoglobin in high-oxygen muscles such as the heart reduces oxygen availability for aerobic metabolism, myocardial contractility, and cardiac output (3). This causes hypoxic cardiac dysfunction, ischemia, arrhythmia, and hypotension. In addition, direct skeletal muscle toxicity and rhabdomyolysis may develop (3,4).

Cytochrome-c oxidase is the final enzyme of the mitochondrial electron transport chain and catalyzes the reduction reaction of molecular oxygen to water (5). CO shows its effect on mitochondrial functions by decreasing cytochrome-c oxidase activity. The affinity of cytochrome c oxidase for oxygen is much higher than for CO. This enzyme binds CO only in cases of severe hypoxia (5). CO binds to cytochrome oxidase and inhibit. As a result of inactivation of mitochondrial enzymes, electron exit from the electron chain in mitochondrial cytochromes stops, cellular respiration and aerobic ATP production are impaired (5).

CO also causes endothelial dysfunction and vasodilation with the release of guanylate cyclase and nitric oxide (NO) (4). CO-related NO release may be one of the main factors for the cytotoxic effects of CO poisoning (4). Guanylate cyclase and NO release play a role in hypotension. Coexistence of relative hypoxia and hypotension may cause ischemia-reperfusion injury in cardiac myocytes as well as neural tissue (2).

In CO poisoning, the damaged endothelium will attract neutrophils leading to lipid peroxidation and eventually neuronal cell death and initiate an inflammatory cascade (4). It converts xanthine dehydrogenase to xanthine oxidase with the CO-triggered cascade. Xanthine oxidase generates free oxygen radicals (4). Ultimately, superoxide formation causes lipid peroxidation and neurological damage in neurons.

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