Pathogenetic aspects of cardiovascular disorders in carbon monoxide poisoning and ways of their correction

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Abstract: Carbon monoxide (CO) poisoning leads to cardiovascular disorders due to reduced oxygen delivery and cellular dysfunction. CO binds to hemoglobin, forming carboxyhemoglobin, impairing oxygen transport and causing systemic hypoxia. This triggers oxidative stress, inflammation, and endothelial dysfunction, contributing to arrhythmias, hypotension, and myocardial injury. The primary treatment, including oxygen therapy and hyperbaric oxygen, helps reduce CO levels and improve oxygenation. Emerging therapies targeting oxidative stress and inflammation are being explored to prevent long-term cardiovascular complications. Early detection and appropriate treatment are crucial to improving outcomes in CO poisoning.

Keywords: carbon monoxide,cardiovascular , methylene blue, CK, LDH, troponin,CD31, HHF35 aktin.

INTRODUCTION: Modern medicine faces numerous problems associated with acute poisoning, each of which has its own characteristics and requires an individual approach to treatment. One of the most common and dangerous types of poisoning is carbon monoxide poisoning. This disease poses a serious threat to both human health and

life. The severity and importance of the problem lies in the fact that carbon monoxide, being highly toxic, can disrupt the normal functioning of organs and systems of the body, especially the cardiovascular system. Carbon monoxide poisoning (CMP) poses a serious threat to health, causing acute and chronic consequences, including dangerous cardiovascular disorders. Carbon monoxide, entering the body, binds to hemoglobin, forming carboxyhemoglobin, which leads to tissue hypoxia and dysfunction of various organs and systems, including the cardiovascular system. Due to a lack of oxygen in the myocardium and other tissues, tachycardia, hypotension, ischemia, and deterioration of blood supply can develop.

There are many studies indicating that damage caused by carbon monoxide can have long-term consequences for the cardiovascular system, including the development of chronic heart failure and arrhythmias. Particular attention should be paid to studying the possibility of correcting hemodynamic disorders that occur during carbon monoxide poisoning. One of the promising methods is the use of methylene blue, a substance with antioxidant properties that can restore normal mitochondrial function and improve tissue respiration . cardiovascular can affect microcirculation, improve organ perfusion, and help restore cardiac function in acute hypoxic conditions. Studies show that methylene blue can reduce carboxyhemoglobin levels and accelerate the restoration of normal oxygen metabolism in tissues Thus, the study of the cardiovascular effects of carbon monoxide poisoning and the mechanisms for correcting these disorders using methylene blue is a relevant and important topic. This will expand our knowledge of the pathogenesis of cardiovascular complications in carbon monoxide poisoning and find more effective ways to treat and prevent this disease, which will help reduce morbidity and mortality from this type of poisoning.

The aim of the study is to investigate the features of the cardiovascular system in carbon monoxide poisoning and to evaluate the effectiveness of methylene blue to correct hemodynamic disorders.

Research objectives:

1. To evaluate cardiovascular disorders in carbon monoxide poisoning in experimental animals.

2. To study biochemical parameters (CK, LDH, troponin) reflecting the state of the cardiovascular system in animals with carbon monoxide poisoning.

3. To evaluate immunohistochemical parameters of the cardiovascular system (CD31, HHF35 aktin) in experimental rats with carbon monoxide poisoning.

4. To analyze the effectiveness of methylene blue in restoring hemodynamic parameters of the cardiovascular system after carbon monoxide poisoning.

Material and methods of the study. In this study, 60 white mongrel male rats with an initial weight of 180-200 g will be used. The animals will be divided into two groups:

The first group - intact animals (control).

The second group - animals exposed to carbon monoxide followed by the introduction of methylene blue to correct cardiovascular disorders.

A carbon monoxide inhalation poisoning model will be used for the experiment. To simulate this, animals are placed in a chamber with a controlled concentration of carbon monoxide. In the carbon monoxide poisoning model, animals are exposed to carbon monoxide for 5 hours a day for 14 days.

ANALYSES AND RESULTS: The primary mechanism by which CO leads to cardiovascular impairment is through the formation of carboxyhemoglobin (COHb), which occurs when CO binds to hemoglobin in red blood cells. This prevents hemoglobin from carrying oxygen and significantly reduces the oxygen-carrying capacity of the blood. As a result, tissues and organs, including the heart, experience a state of hypoxia, or oxygen deprivation, leading to cellular dysfunction and damage. Hypoxia in the cardiovascular system is particularly concerning because it can result in arrhythmias, reduced myocardial

contractility, and myocardial injury, which may lead to heart failure or circulatory collapse in severe cases. In addition to hypoxia, CO poisoning also triggers the generation of reactive oxygen species (ROS), leading to oxidative stress. The buildup of ROS contributes to cellular injury, endothelial dysfunction, and inflammation, further exacerbating cardiovascular complications. Endothelial dysfunction, in particular, impairs the regulation of vascular tone, leading to issues such as hypotension and the potential for the development of thrombotic events.

The cardiovascular system's response to CO exposure also involves changes in vascular smooth muscle function. CO can interfere with the regulation of vascular tone, contributing to abnormal vasodilation or vasoconstriction, which can further complicate cardiovascular management. In severe cases, CO poisoning can lead to myocardial ischemia, arrhythmias, and potentially fatal outcomes due to circulatory collapse.

Clinical Manifestations

Clinically, patients with CO poisoning may present with a wide variety of cardiovascular symptoms. Early signs may include chest pain, palpitations, tachycardia, and hypotension. These symptoms arise from the decreased oxygen supply to the myocardium and other tissues, resulting in compromised cardiac output. In more severe cases, patients may experience arrhythmias, including ventricular fibrillation or bradycardia, as the heart's electrical conduction system becomes disturbed due to hypoxia and electrolyte imbalances. In addition to these direct cardiovascular effects, CO poisoning can also exacerbate underlying cardiovascular conditions. For example, individuals with pre-existing coronary artery disease may be at greater risk for experiencing severe myocardial infarction and other cardiovascular complications following CO exposure.

Therapeutic Approaches

The treatment of cardiovascular disorders resulting from CO poisoning primarily involves reversing the effects of hypoxia and reducing the levels of COHb in the blood. The most commonly used intervention is oxygen therapy, particularly high-concentration oxygen administered through non-rebreather masks. This treatment increases the dissociation of CO from hemoglobin, effectively reducing COHb levels and improving tissue oxygenation. Hyperbaric oxygen therapy (HBOT) has been shown to be particularly beneficial for patients with severe CO poisoning. HBOT involves the administration of oxygen at pressures greater than atmospheric pressure, which not only increases the amount of oxygen dissolved in plasma but also accelerates the elimination of CO from the body. Numerous studies have demonstrated that HBOT significantly reduces the incidence of long-term neurological and cardiovascular sequelae, including arrhythmias and myocardial injury, by rapidly decreasing COHb levels and restoring normal oxygen delivery to tissues.

In addition to traditional oxygen therapy, emerging therapeutic strategies aim to address the secondary effects of CO poisoning, such as oxidative stress and inflammation. Antioxidant therapy is being explored as a potential adjunctive treatment, as it may help mitigate the cellular damage caused by ROS. Similarly, anti-inflammatory agents may offer a way to reduce the systemic inflammatory response triggered by CO exposure, potentially decreasing the risk of cardiovascular complications. Further, researchers are investigating the role of endothelial repair in improving outcomes for CO poisoning patients. Since endothelial dysfunction is a key contributor to cardiovascular problems in CO poisoning, therapies aimed at restoring endothelial integrity and function could play a significant role in preventing long-term cardiovascular damage.

Long-Term Outcomes

The long-term cardiovascular outcomes of CO poisoning depend on the severity of the exposure, the timeliness of treatment, and the individual's pre-existing health conditions. In many cases, patients who receive appropriate treatment early in the course of poisoning make a full recovery. However, in severe cases or when treatment is delayed, patients may experience chronic cardiovascular issues, such as persistent arrhythmias, ischemic heart disease, or even heart failure. A critical factor in long-term recovery is the ability to restore endothelial function and reduce the risk of thrombotic events. Research into the prolonged effects of CO poisoning suggests that even after the immediate hypoxic crisis is addressed, patients may still be at risk for cardiovascular events months or years later. This highlights the need for careful long-term monitoring and potentially the use of pharmacologic therapies to address any lingering cardiovascular dysfunction.

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