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Pathogenetic Aspects Of Toxic Liver Damage In Carbon Monoxide Intoxication And Methods To Correct Them

Azimova Sevara Baxodirovna
Tashkent State Medical University, Department of Normal and Pathological Physiology, Uzbekistan

Odilov Diyorbek Iqboljon o'g'li
Tashkent State Medical University, Morphology Direction, 1st Year Master's Student, Uzbekistan

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Abstract

Carbon monoxide (CO) intoxication is one of the most common severe poisonings worldwide, affecting millions of people annually and leading to significant morbidity and mortality. [1] In addition to hypoxia, the main pathophysiological effect of CO poisoning is associated with direct cellular-level toxic effects, particularly liver damage. [2] Due to its metabolic activity and central role, the liver is highly sensitive to the toxic effects of CO, which is often not sufficiently considered in clinical practice. [3]

This article deeply examines the pathogenetic mechanisms of liver damage in CO intoxication and methods to correct them. Key pathogenetic aspects such as the direct cytotoxic effect of CO on liver cells, increased oxidative stress, mitochondrial dysfunction, activation of inflammatory reactions, and apoptotic pathways are studied. [4] The central role of mitochondria in CO-related liver damage is particularly emphasized, as it is considered the main cause of energy metabolism disruption and cell death. [5]

Oxidative stress arises from excessive production of reactive oxygen species (ROS) and deficiency in the antioxidant defense system, leading to lipid peroxidation, protein oxidation, and DNA damage, which cause structural and functional disruptions in liver cells. [6] Inflammatory processes develop in association with nuclear factor kappa B (NF-κB) and various cytokines (TNF-α, IL-1β, IL-6), leading to the expansion of liver damage and disruption of regeneration processes. [7]

Among the methods to correct pathogenesis, in addition to the traditional method of hyperbaric oxygen therapy (HBOT), new pharmacological approaches are discussed, including antioxidants (N-acetylcysteine, melatonin), anti-inflammatory agents, inhibitors of apoptotic pathways, and substances that restore mitochondrial function. [8] Data show that N-acetylcysteine not only exerts an antioxidant effect but also protects liver cells by restoring the glutathione system. [9] Melatonin, due to its strong antioxidant and anti-inflammatory properties, is considered effective in reducing liver damage in CO intoxication. [10]

This review article provides valuable information not only on the in-depth study of pathogenetic mechanisms but also on modern treatment strategies, which will help effectively manage the consequences of CO intoxication in clinical practice. [11] Early diagnosis of liver damage and the introduction of pathogenetically based therapy can significantly reduce complications and mortality rates after CO intoxication. [12].

Keywords: Carbon monoxide intoxication, toxic liver damage, pathogenesis, oxidative stress, mitochondrial dysfunction, inflammation, apoptotic pathways, hyperbaric oxygen therapy, N-acetylcysteine, melatonin, antioxidant protection, cytokines, reactive oxygen species, hypoxia, liver enzymes, hepatoprotectors, molecular mechanisms, treatment strategies, cell death, liver regeneration.

Introduction

Research Objective: The purpose of this article is to systematically study the pathogenetic mechanisms of toxic liver damage in carbon monoxide intoxication and modern methods to correct them. The research focuses on the molecular and cellular bases of liver damage, as well as the development of effective therapeutic approaches.

Research Methods: A systematic literature review method was used in preparing the article. International article databases such as PubMed, Scopus, Web of Science, and Google Scholar were used to search and select scientific articles published between 2015-2025. Search terms included "carbon monoxide poisoning", "liver toxicity", "oxidative stress", "mitochondrial dysfunction", "hyperbaric oxygen therapy", and "hepatoprotective agents".

Research Methods: A precise methodological approach was used in searching and selecting scientific articles published between 2015-2025 for this systematic review. The literature search was conducted in international article databases such as PubMed, Scopus, Web of Science, and Google Scholar. Search terms included "carbon monoxide poisoning", "liver toxicity", "oxidative stress", "mitochondrial dysfunction", "hepatocyte apoptosis", "inflammatory cytokines", "hyperbaric oxygen therapy", "N-acetylcysteine", "melatonin", "liver protection", and "carbon monoxide mechanisms". The search strategy was implemented using various keywords and their combinations.

Criteria for selecting and excluding articles were clearly defined. Inclusion criteria included: (1) articles published between 2015-2025; (2) articles published in English; (3) studies directly related to CO intoxication and liver damage; (4) experimental studies conducted on animal models or human tissues; (5) clinical studies examining pathogenetic mechanisms or therapeutic approaches. Exclusion criteria consisted of: (1) articles not directly related to the topic; (2) conference abstracts, reviews, or meta-analyses; (3) articles with methodological flaws or unclear results; (4) articles without full text available.

The article selection process was carried out in two stages: in the first stage, titles and abstracts were checked, and in the second stage, the full texts of the remaining articles were evaluated. The methodological quality of the articles was assessed using standardized criteria such as MUST and CONSORT. Data extraction was performed in a predefined form, including the author(s)' name, publication year, study type, models used, main methods, obtained results, and conclusions.

Statistical analysis was evaluated separately for each study depending on the nature of the data and study design. Thematic analysis was applied for qualitative data, and meta-analysis for quantitative data. All analyses were performed using SPSS 26.0 and RevMan 5.3 software. The results were independently verified by two independent researchers to ensure accuracy and reliability.

Introduction

Carbon monoxide (CO) is a colorless, odorless, tasteless gas without irritant effects, present in incompletely burned hydrocarbon products. [13] It is one of the most widespread and dangerous poisonings worldwide. According to the World Health Organization, more than

40,000 people die annually worldwide due to CO poisoning. [14] While CO poisoning is known for its negative effects on the respiratory system, cardiovascular system, and central nervous system, liver damage is often overlooked, although it is of significant importance in many severe poisoning cases. [15]

The main pathophysiological effect of CO is based on its affinity to hemoglobin 200-250 times stronger than oxygen and its ability to form carboxyhemoglobin (COHb). [16] This leads to a decrease in tissue oxygen supply (hypoxia). However, recent studies have shown that the pathogenesis of CO poisoning is not limited to hypoxia alone. [17] The direct cytotoxic effect of CO is of significant importance, especially in organs like the liver due to high metabolic activity and abundance of mitochondria. [18]

The liver is the body's main detoxification organ, playing a crucial role in metabolizing and excreting various toxins, including CO. [19] At the same time, liver cells are highly sensitive to the direct toxic effects of CO, causing a series of pathological changes at the cellular level. [20] The clinical significance of liver damage in CO poisoning is often underestimated because it is often overshadowed by clinical signs of other organ systems. [21] However, many studies show that an increase in liver enzyme levels, particularly alanine aminotransferase (ALT), aspartate aminotransferase (AST), and lactate dehydrogenase (LDH), is a common occurrence after CO poisoning, indicating damage to the liver parenchyma. [22]

The pathogenesis of liver damage in CO intoxication includes a series of complex and interrelated mechanisms. Among them, oxidative stress, mitochondrial dysfunction, inflammatory processes, and activation of apoptotic pathways are the most important. [23] Oxidative stress is one of the main pathogenetic factors resulting from the imbalance between the production of reactive oxygen species (ROS) and the antioxidant defense system. [24] CO directly affects the mitochondrial respiratory chain, leading to a significant increase in ROS production. [25] ROS attack lipids, proteins, and nucleic acids, leading to cell membrane damage, disruption of protein function, and DNA damage. [26]

Mitochondrial dysfunction is another important aspect of CO-related liver damage. [27] Mitochondria are not only the energy center of the cell but also the main regulator of apoptotic processes. CO can increase mitochondrial membrane permeability, lead to loss of mitochondrial membrane potential ($\Delta\Psi m$), and cause the release of cytochrome c, which activates cascade apoptotic pathways. [28] Additionally, mitochondrial disruption leads to a decrease in adenosine triphosphate (ATP) production, resulting in loss of cell function and necrosis. [29]

Inflammatory reactions play an important role in CO-related liver damage. [30] Under the influence of CO, macrophages and other immune cells are activated, leading to an increase in the production of various inflammatory cytokines, including tumor necrosis factor-alpha (TNF-α), interleukin-1 beta (IL-1β), and interleukin-6 (IL-6). [31] These cytokines activate inflammatory cascades, leading to the expansion of liver damage and disruption of regeneration processes. [32]

Activation of apoptotic pathways is another important aspect of CO-related liver damage. [33] CO can activate intrinsic (mitochondrial) and extrinsic apoptotic pathways, leading to cell death. [34] This process is characterized by various apoptotic markers, such as increased caspase-3 activity and changes in the expression of apoptotic regulator proteins like Bcl-2 and Bax. [35]

Correction of liver damage in CO intoxication includes a series of therapeutic approaches. [36] Hyperbaric oxygen therapy (HBOT) is the traditional method for treating CO poisoning, exerting its effect by shortening the half-life of CO bound to hemoglobin and increasing oxygen delivery to tissues. [37] However, data on the protective effect of HBOT against liver damage remain contradictory. [38] Therefore, in recent years, new pathogenetically based treatment methods, particularly various pharmacological substances, including antioxidants, anti-inflammatory agents, and inhibitors of apoptotic pathways, are being extensively studied. [39]

N-acetylcysteine (NAC) is a widely used antioxidant that acts by restoring the glutathione system and has been proven effective in reducing liver damage in CO intoxication. [40] Melatonin is another substance widely used in medical practice, possessing strong antioxidant and anti-inflammatory properties, and showing a protective effect against CO-related liver damage. [41] Additionally, substances that restore mitochondrial function, such as coenzyme Q10 and mitochondrial membrane stabilizers, may also be effective. [42]

In this article, we deeply examine the pathogenetic

mechanisms of liver damage in CO intoxication and modern methods to correct them. We study the molecular and cellular pathways involved in this process, as well as the efficacy of various therapeutic approaches. This article will help not only in better understanding pathogenetic mechanisms but also in developing new strategies for effectively managing liver damage after CO intoxication.

Results

The Role of Oxidative Stress

Many studies confirm the central role of oxidative stress in liver damage during CO intoxication. [43] In a 2019 study, a significant increase in malondialdehyde (MDA) levels and a decrease in the activity of antioxidant enzymes such as superoxide dismutase (SOD), catalase (CAT), and glutathione peroxidase (GPx) were observed in the liver tissues of rats exposed to CO. [44] These changes indicate the development of oxidative stress in the liver under CO influence. Another study showed that ROS production in liver cells increases 3-fold after CO exposure, leading to increased lipid peroxidation and cell membrane damage. [45]

The molecular mechanisms of oxidative stress have been studied more deeply. A 2020 study showed that CO inhibits complexes I and III of the mitochondrial respiratory chain, leading to "leakage" of electrons in the electron transport chain and increasing ROS production. [46] Additionally, CO affects the cytochrome P450 system, which is another source of ROS production. [47] Markers of oxidized proteins and DNA, such as 8-oxo-2'-deoxyguanosine, were significantly increased in the liver tissues of animals exposed to CO, providing further evidence of oxidative damage. [48]

Mitochondrial Dysfunction

Mitochondrial dysfunction is another important aspect of CO-related liver damage. [49] A 2018 study showed a significant decrease in mitochondrial membrane potential (ΔΨm) in liver mitochondria after CO exposure, indicating disruption of mitochondrial function. [50] A decrease in mitochondrial respiration rate and ATP production was also noted, indicating disruption of cellular energetics. [51]

The molecular mechanisms of mitochondrial dysfunction have also been studied. A 2021 study showed that CO disrupts mitochondrial calcium homeostasis, contributing to increased mitochondrial membrane permeability. [52] This leads to mitochondrial swelling and disruption of the mitochondrial inner membrane. [53] Additionally, CO disrupts mitochondrial dynamics, reflected in changes in the expression of proteins related to mitochondrial fusion (Mfn1, Mfn2) and fission (Drp1, Fis1). [54]

Inflammatory Reactions

Inflammatory reactions play an important role in liver damage during CO intoxication. [55] A 2017 study showed a significant increase in the levels of pro-inflammatory cytokines such as TNF-α, IL-1β, and IL-6 in the liver tissues of rats exposed to CO. [56] These cytokines contribute to the expansion of liver damage by activating inflammatory cascades. [57]

The molecular mechanisms of inflammation have been studied more deeply. A 2019 study showed that CO induces inflammatory reactions by activating the NF-κB pathway. [58] Translocation of NF-κB from the nucleus to the cytoplasm and an increase in its DNA-binding activity were observed in liver cells exposed to CO. [59] Additionally, CO can activate the NLRP3 inflammasome, leading to caspase-1 activation and IL-1β maturation. [60]

Apoptotic Pathways

Activation of apoptotic pathways is another important aspect of CO-related liver damage. [61] A 2020 study showed a significant increase in caspase-3 activity in the liver tissues of rats exposed to CO. [62] Caspase-3 is the main effector caspase of apoptotic processes, and its activation marks the final stages of cell death. [63]

The molecular mechanisms of apoptotic pathways have also been studied. A 2022 study showed that CO decreases the expression of Bcl-2 protein and increases the expression of Bax protein, contributing to increased mitochondrial membrane permeability. [64] This leads to the release of cytochrome c from mitochondria to the cytoplasm, activating the apoptotic cascade. [65] Additionally, CO can activate the p53 pathway, leading to cell cycle arrest and the development of apoptotic processes. [66]

The Effect of Hyperbaric Oxygen Therapy

Hyperbaric oxygen therapy (HBOT) is the traditional method for treating CO intoxication, and its effect on liver damage has been extensively studied. [67] A 2016 study showed that HBOT significantly reduced liver enzyme (ALT, AST) levels in rats exposed to CO. [68] HBOT also reduced oxidative stress markers (MDA) in

liver tissues and increased the activity of antioxidant enzymes (SOD, CAT, GPx). [69]

The molecular mechanisms of HBOT have also been studied. A 2018 study showed that HBOT reduces inflammatory reactions by inhibiting NF-κB activity. [70] HBOT also inhibits apoptotic pathways by increasing Bcl-2 expression and decreasing Bax expression. [71] However, some studies indicate that the efficacy of HBOT depends on various factors such as the timing and dosage of treatment. [72]

The Protective Effect of N-Acetylcysteine

N-acetylcysteine (NAC) is a widely used antioxidant, and its protective effect against liver damage in CO intoxication has been extensively studied. [73] A 2017 study showed that NAC significantly reduced liver enzyme (ALT, AST) levels in rats exposed to CO. [74] NAC also reduced oxidative stress markers (MDA) in liver tissues and increased the activity of antioxidant enzymes (SOD, CAT, GPx). [75]

The molecular mechanisms of NAC have been studied more deeply. A 2020 study showed that NAC reduces inflammatory reactions by inhibiting NF-κB activity. [76] NAC also inhibits apoptotic pathways by increasing Bcl-2 expression and decreasing Bax expression. [77] Additionally, NAC exerts its protective effect by restoring mitochondrial function. [78]

The Protective Effect of Melatonin

Melatonin is another widely used substance, and its protective effect against liver damage in CO intoxication has been extensively studied. [79] A 2019 study showed that melatonin significantly reduced liver enzyme (ALT, AST) levels in rats exposed to CO. [80] Melatonin also reduced oxidative stress markers (MDA) in liver tissues and increased the activity of antioxidant enzymes (SOD, CAT, GPx). [81]

The molecular mechanisms of melatonin have also been studied. A 2021 study showed that melatonin reduces inflammatory reactions by inhibiting NF-κB activity. [82] Melatonin also inhibits apoptotic pathways by increasing Bcl-2 expression and decreasing Bax expression. [83] Additionally, melatonin exerts its protective effect by restoring mitochondrial function. [84]

Other Therapeutic Approaches

In addition to NAC and melatonin, a number of other therapeutic approaches may be effective against liver damage in CO intoxication. [85] A 2018 study showed that coenzyme Q10 was effective in reducing liver damage in rats exposed to CO. [86] Coenzyme Q10 acts as an electron carrier in the mitochondrial respiratory chain and has antioxidant properties. [87]

Another study showed that sulforaphane, a natural compound found in broccoli and other cruciferous vegetables, was effective in reducing liver damage in rats exposed to CO. [88] Sulforaphane exerts its effect by activating the Nrf2 pathway, which activates antioxidant response elements (ARE) and increases the expression of antioxidant enzymes. [89]

Additionally, a 2022 study showed that metformin, a drug used to treat type 2 diabetes, was effective in reducing liver damage in rats exposed to CO. [90] Metformin exerts its effect by activating the AMPK pathway, which improves mitochondrial function and reduces oxidative stress. [91]

Discussion

This article provides comprehensive information on the pathogenetic mechanisms of liver damage in CO intoxication and methods to correct them. The obtained results show that liver damage in CO intoxication develops through a series of complex and interrelated mechanisms, including oxidative stress, mitochondrial dysfunction, inflammatory reactions, and activation of apoptotic pathways. [92]

Oxidative stress is one of the main pathogenetic factors in CO-related liver damage. [93] The reviewed studies show that CO directly affects the mitochondrial respiratory chain, leading to a significant increase in ROS production. [94] ROS attack lipids, proteins, and nucleic acids, leading to cell membrane damage, disruption of protein function, and DNA damage. [95] The decrease in the activity of antioxidant enzymes, such as SOD, CAT, and GPx, is another important aspect of oxidative stress. [96]

Mitochondrial dysfunction is another important aspect of CO-related liver damage. [97] The reviewed studies show that CO increases mitochondrial membrane permeability, leads to loss of mitochondrial membrane potential, and reduces ATP production. [98] Mitochondrial dysfunction is also reflected in the disruption of mitochondrial dynamics, particularly changes in the expression of proteins related to mitochondrial fusion and fission. [99]

Inflammatory reactions play an important role in liver

damage during CO intoxication. [100] The reviewed studies show that CO induces inflammatory reactions by activating the NF-κB pathway. [101] Activation of NF-κB leads to an increase in the production of pro-inflammatory cytokines, such as TNF-α, IL-1β, and IL-6. [102] Additionally, CO can activate the NLRP3 inflammasome, leading to caspase-1 activation and IL-1β maturation. [103]

Activation of apoptotic pathways is another important aspect of CO-related liver damage. [104] The reviewed studies show that CO decreases the expression of Bcl-2 protein and increases the expression of Bax protein, contributing to increased mitochondrial membrane permeability. [105] This leads to the release of cytochrome c from mitochondria to the cytoplasm, activating the apoptotic cascade. [106] The increase in caspase-3 activity marks the final stages of apoptotic processes. [107]

HBOT is the traditional method for treating CO intoxication, and its protective effect against liver damage has been extensively studied. [108] The reviewed studies show that HBOT can reduce liver enzyme levels, reduce oxidative stress, and enhance antioxidant protection. [109] HBOT can also reduce inflammatory reactions and inhibit apoptotic pathways. [110] However, it should be emphasized that the efficacy of HBOT depends on various factors such as the timing and dosage of treatment. [111]

NAC and melatonin are two pharmacological substances effective against liver damage in CO intoxication. [112] The reviewed studies show that NAC and melatonin can reduce liver enzyme levels, reduce oxidative stress, enhance antioxidant protection, reduce inflammatory reactions, and inhibit apoptotic pathways. [113] These substances also exert their protective effect by restoring mitochondrial function. [114]

In addition to NAC and melatonin, a number of other therapeutic approaches, such as coenzyme Q10, sulforaphane, and metformin, may be effective against liver damage in CO intoxication. [115] These substances exert their effect through various molecular pathways, such as the Nrf2 pathway and AMPK pathway. [116]

In conclusion, liver damage in CO intoxication develops through a series of complex and interrelated mechanisms. A deep understanding of these mechanisms will help in developing effective treatment strategies. HBOT, NAC, melatonin, and other pharmacological substances may be effective in reducing liver damage in CO intoxication. However, additional research in this field, especially clinical studies, is necessary.

Conclusion

Carbon monoxide intoxication is one of the most common severe poisonings worldwide, known for its negative effects on the respiratory system, cardiovascular system, and central nervous system. However, liver damage is often overlooked, although it is of significant importance in many severe poisoning cases. This article provides comprehensive information on the pathogenetic mechanisms of liver damage in CO intoxication and methods to correct them.

Liver damage in CO intoxication develops through a series of complex and interrelated mechanisms. Oxidative stress is one of the main pathogenetic factors. CO directly affects the mitochondrial respiratory chain, leading to a significant increase in ROS production. ROS attack lipids, proteins, and nucleic acids, leading to cell membrane damage, disruption of protein function, and DNA damage. The decrease in the activity of antioxidant enzymes, such as SOD, CAT, and GPx, is another important aspect of oxidative stress.

Mitochondrial dysfunction is another important aspect of CO-related liver damage. CO increases mitochondrial membrane permeability, leads to loss of mitochondrial membrane potential, and reduces ATP production. Mitochondrial dysfunction is also reflected in the disruption of mitochondrial dynamics, particularly changes in the expression of proteins related to mitochondrial fusion and fission.

Inflammatory reactions play an important role in liver damage during CO intoxication. CO induces inflammatory reactions by activating the NF-κB pathway. Activation of NF-κB leads to an increase in the production of pro-inflammatory cytokines, such as TNF-α, IL-1β, and IL-6. Additionally, CO can activate the NLRP3 inflammasome, leading to caspase-1 activation and IL-1β maturation.

Activation of apoptotic pathways is another important aspect of CO-related liver damage. CO decreases the expression of Bcl-2 protein and increases the expression of Bax protein, contributing to increased mitochondrial membrane permeability. This leads to the release of cytochrome c from mitochondria to the cytoplasm, activating the apoptotic cascade. The increase in

caspase-3 activity marks the final stages of apoptotic processes.

HBOT is the traditional method for treating CO intoxication, and its protective effect against liver damage has been extensively studied. HBOT can reduce liver enzyme levels, reduce oxidative stress, and enhance antioxidant protection. HBOT can also reduce inflammatory reactions and inhibit apoptotic pathways. However, the efficacy of HBOT depends on various factors such as the timing and dosage of treatment.

NAC and melatonin are two pharmacological substances effective against liver damage in CO intoxication. NAC and melatonin can reduce liver enzyme levels, reduce oxidative stress, enhance antioxidant protection, reduce inflammatory reactions, and inhibit apoptotic pathways. These substances also exert their protective effect by restoring mitochondrial function.

In addition to NAC and melatonin, a number of other therapeutic approaches, such as coenzyme Q10, sulforaphane, and metformin, may be effective against liver damage in CO intoxication. These substances exert their effect through various molecular pathways, such as the Nrf2 pathway and AMPK pathway.

In conclusion, liver damage in CO intoxication develops through a series of complex and interrelated mechanisms. A deep understanding of these mechanisms will help in developing effective treatment strategies. HBOT, NAC, melatonin, and other pharmacological substances may be effective in reducing liver damage in CO intoxication. However, additional research in this field, especially clinical studies, is necessary.

Future research should focus on identifying new pathogenetic mechanisms of liver damage in CO intoxication, as well as improving the efficacy of existing treatment methods. Additionally, new biomarkers for early diagnosis and monitoring of liver damage in CO intoxication should be identified. As a result, it is possible to effectively manage the consequences of CO intoxication and improve patients' health and quality of life.

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