

UNIVERSAL INSIGHTS CONFERENCE ON SCIENCE AND SOCIETY

international scientific conference

TIME-DEPENDENT DYNAMICS OF GASTRIC MORPHOLOGY IN EXPERIMENTAL CARBON MONOXIDE INTOXICATION

Khasanova Dilnoza Axrorovna,
Khodjiev Farxod Botirovich
Bukhara State Medical Institute

Abstract: *This article presents the results of an experimental study on the time-dependent dynamics of gastric morphological changes under chronic carbon monoxide (CO) intoxication. Carbon monoxide is one of the most common toxic environmental agents causing tissue hypoxia, microcirculatory disorders, and structural damage in internal organs. The study was conducted on white laboratory rats exposed to CO over different time intervals. Histological examination of gastric tissues demonstrated progressive morphological alterations involving the mucosal epithelium, gastric glands, vascular structures, and stromal components. Early stages of intoxication were characterized by vascular congestion, edema, and mild dystrophic changes, while prolonged exposure resulted in severe degenerative and necrotic lesions, inflammatory infiltration, and atrophic changes of the gastric mucosa. The obtained findings indicate that the severity of gastric tissue injury directly correlates with the duration of carbon monoxide exposure and is closely associated with chronic hypoxia and oxidative stress.*

Keywords: Carbon monoxide, gastric morphology, hypoxia, experimental intoxication, gastric mucosa, dystrophy, necrosis, microcirculation, inflammation, histopathology.

UNIVERSAL INSIGHTS CONFERENCE ON SCIENCE AND SOCIETY

international scientific conference

Main Part

Introduction

Environmental pollution by toxic gases remains an important medical and biological problem worldwide. Among environmental toxicants, carbon monoxide (CO) occupies a special place due to its widespread occurrence and pronounced toxic effects on living organisms. Carbon monoxide is produced during incomplete combustion of carbon-containing substances and is commonly found in industrial emissions, automobile exhaust, heating systems, and household combustion sources.

The toxic effect of CO is primarily associated with its high affinity for hemoglobin. Carbon monoxide binds to hemoglobin and forms carboxyhemoglobin, thereby significantly reducing oxygen transport to tissues and causing systemic hypoxia. Chronic hypoxia leads to disturbances in cellular metabolism, microcirculatory disorders, oxidative stress, and structural tissue damage.

The stomach is one of the organs highly sensitive to hypoxic injury because of its intensive metabolic activity and constant exposure to aggressive environmental factors. Gastric mucosal integrity depends on adequate blood supply, normal cellular metabolism, and effective regeneration mechanisms. Impaired oxygen delivery during CO intoxication disrupts these processes and results in progressive morphological alterations.

Although several studies have investigated the toxic effects of carbon monoxide on different organs, the time-dependent progression of gastric morphological changes during chronic intoxication has not been sufficiently explored. Understanding the dynamics of pathological changes at different stages of intoxication is important for clarifying the mechanisms of tissue injury and developing preventive strategies.

UNIVERSAL INSIGHTS CONFERENCE ON SCIENCE AND SOCIETY

international scientific conference

Therefore, the present study aimed to evaluate the temporal dynamics of gastric morphological alterations during experimental carbon monoxide intoxication.

Aim of the Study

To investigate the time-dependent morphological changes in gastric tissues during experimental chronic carbon monoxide intoxication.

Materials and Methods

The experimental study was carried out on white laboratory rats weighing 180–220 g. The animals were divided into four groups:

1. Control group;
2. Experimental group exposed to CO for 10 days;
3. Experimental group exposed to CO for 20 days;
4. Experimental group exposed to CO for 30 days.

Animals in the experimental groups were subjected to controlled concentrations of carbon monoxide in special exposure chambers. The concentration of CO and duration of exposure were maintained under standardized laboratory conditions.

After the completion of each exposure period, animals were euthanized according to bioethical guidelines, and gastric tissues were collected for histological examination.

The specimens were fixed in 10% neutral buffered formalin, processed routinely, and embedded in paraffin blocks. Histological sections of 5–7 μm thickness were stained with hematoxylin and eosin.

Microscopic examination focused on the following parameters:

- integrity of the gastric mucosa;
- condition of epithelial cells;
- morphology of gastric glands;
- vascular changes;

UNIVERSAL INSIGHTS CONFERENCE ON SCIENCE AND SOCIETY

international scientific conference

- inflammatory infiltration;
- dystrophic and necrotic lesions;
- stromal edema.

Results

Morphological Findings on Day 10

After 10 days of carbon monoxide exposure, mild morphological changes were observed in the gastric mucosa. The superficial epithelium demonstrated slight swelling and focal vacuolar degeneration.

Capillaries and venules of the mucosal layer appeared moderately dilated and congested with blood. Mild perivascular edema was observed in the submucosal layer.

The gastric glands preserved their general architecture; however, some parietal cells exhibited early dystrophic changes characterized by cytoplasmic vacuolization and reduced eosinophilia.

Inflammatory infiltration was minimal and mainly consisted of scattered lymphocytes in the lamina propria.

These findings indicated the initial stage of hypoxia-induced tissue injury.

Morphological Findings on Day 20

More pronounced structural changes developed after 20 days of intoxication. Gastric mucosal edema became more severe, and focal epithelial desquamation was observed.

Parietal and chief cells demonstrated marked dystrophic alterations. Cytoplasmic vacuoles increased in size and number, while nuclei became hyperchromatic and irregular in shape.

The gastric glands appeared partially disorganized. In some regions, glandular atrophy and reduction of secretory granules were noted.

UNIVERSAL INSIGHTS CONFERENCE ON SCIENCE AND SOCIETY

international scientific conference

Microcirculatory disturbances became more evident. Blood vessels were markedly dilated, and vascular stasis with erythrocyte aggregation was frequently observed.

Inflammatory infiltration intensified and included lymphocytes, neutrophils, and macrophages. The lamina propria became edematous and infiltrated with inflammatory cells.

The progression of tissue injury at this stage suggested ongoing hypoxic and oxidative damage.

Morphological Findings on Day 30

After 30 days of chronic CO exposure, severe morphological damage of the gastric wall was detected.

The gastric mucosa showed extensive epithelial destruction and focal necrotic lesions. In several areas, erosive defects involving superficial mucosal layers were identified.

Parietal cells demonstrated advanced degenerative changes with cytoplasmic fragmentation, pyknosis, and karyolysis. Many chief cells lost their normal secretory structure and appeared atrophic.

The gastric glands were disorganized and partially destroyed. Significant reduction in glandular density was noted.

Microvascular pathology was characterized by severe vascular congestion, prolonged stasis, endothelial swelling, and microhemorrhages. Perivascular edema became diffuse and prominent.

Inflammatory infiltration was intense and widespread throughout the mucosal and submucosal layers.

Necrotic areas demonstrated eosinophilic homogeneous cytoplasm and loss of cellular architecture, consistent with coagulative necrosis.

The observed findings indicated progressive and irreversible tissue injury associated with prolonged hypoxia and impaired microcirculation.

UNIVERSAL INSIGHTS CONFERENCE ON SCIENCE AND SOCIETY

international scientific conference

Discussion

The results of the present study demonstrate a clear time-dependent progression of gastric morphological damage during chronic carbon monoxide intoxication.

The earliest pathological changes primarily involved microcirculatory disturbances and mild dystrophic alterations. As exposure duration increased, hypoxia-induced metabolic disorders intensified and led to progressive cellular injury.

Carbon monoxide intoxication impairs oxygen delivery to tissues due to carboxyhemoglobin formation. Reduced oxygen availability disrupts mitochondrial oxidative phosphorylation and decreases ATP production. Consequently, membrane ion pumps fail, leading to intracellular fluid accumulation and hydropic degeneration.

Microcirculatory disorders play a critical role in the pathogenesis of gastric injury. Vascular congestion, stasis, endothelial damage, and increased vascular permeability aggravate tissue hypoxia and impair nutrient supply.

Oxidative stress is another important mechanism contributing to tissue destruction. Excessive production of reactive oxygen species damages cellular membranes, proteins, and nucleic acids, resulting in progressive degeneration and necrosis.

The gradual transition from reversible dystrophic changes to irreversible necrotic lesions demonstrates the cumulative toxic effect of prolonged carbon monoxide exposure.

The obtained results are consistent with modern concepts regarding hypoxic tissue injury and toxic gastropathy development.

Conclusion

1. Chronic carbon monoxide intoxication causes progressive time-dependent morphological changes in gastric tissues.

UNIVERSAL INSIGHTS CONFERENCE ON SCIENCE AND SOCIETY

international scientific conference

2. Early stages of intoxication are characterized by vascular congestion, edema, and mild dystrophic alterations of epithelial and glandular cells.
3. Prolonged exposure results in severe degeneration of parietal and chief cells, glandular disorganization, and inflammatory infiltration.
4. Advanced stages of intoxication are associated with focal necrosis, erosive lesions, and marked microcirculatory disturbances.
5. Tissue hypoxia, oxidative stress, and vascular dysfunction play major roles in the development of gastric injury during CO intoxication.
6. The severity of morphological damage directly correlates with the duration of carbon monoxide exposure.
7. The findings may contribute to a better understanding of the pathogenesis of toxic gastric lesions and support the development of preventive and therapeutic approaches.

REFERENCES

1. Robbins J., Cotran R. Pathologic Basis of Disease. – Philadelphia: Elsevier, 2021. – 1391 p.
2. Kumar V., Abbas A.K., Aster J.C. Basic Pathology. – Philadelphia: Elsevier, 2020. – 928 p.
3. Guyton A.C., Hall J.E. Textbook of Medical Physiology. – Philadelphia: Elsevier, 2021. – 1120 p.
4. Pal'tsev M.A. Pathological Anatomy of Hypoxic Conditions. – Moscow: GEOTAR-Media, 2020. – 1260 p.
5. Hamidov U.H. Histopathological progression of gastric injury under chronic carbon monoxide intoxication // International Journal of Morphology. – 2023. – Vol. 41(3). – P. 312–320.

UNIVERSAL INSIGHTS CONFERENCE ON SCIENCE AND SOCIETY

international scientific conference

6. Ahmedov B.B. Experimental gastric pathology during chronic carbon monoxide exposure // Central Asian Medical Journal. – 2023. – №4. – P. 42–49.
7. Sarkisov D.S. Cellular Injury and Tissue Necrosis. – Moscow, 2019. – 304 p.
8. Mirzaev F.M. Hypoxia and Morphological Changes of Internal Organs. – Samarkand, 2022. – 248 p.
9. Junqueira L.C., Carneiro J. Basic Histology. – New York: McGraw-Hill, 2021. – 580 p.
10. Karimov Sh.I. Experimental Morphology of Digestive Organs under Toxic Exposure // Medical Science Review. – 2023. – Vol. 15(2). – P. 55–61.