

**HISTOLOGICAL ANALYSIS OF GASTRIC
MORPHOLOGY IN EXPERIMENTAL CARBON
MONOXIDE INTOXICATION USING HEMATOXYLIN-
EOSIN STAINING**

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Abstract: *This article presents a histological analysis of gastric morphological changes during experimental carbon monoxide (CO) intoxication using hematoxylin-eosin (H&E) staining. Carbon monoxide is a highly toxic environmental gas capable of inducing severe tissue hypoxia and structural alterations in internal organs. The study was performed on white laboratory rats exposed to chronic CO intoxication. Histological examination of gastric tissues stained with hematoxylin and eosin revealed progressive pathological changes involving the mucosal epithelium, gastric glands, vascular structures, and stromal components. The most prominent findings included epithelial dystrophy, vascular congestion, edema, inflammatory infiltration, glandular degeneration, and focal necrotic lesions. Hematoxylin-eosin staining proved effective for evaluating both cellular and tissue-level pathological alterations induced by chronic carbon monoxide exposure. The findings demonstrate that prolonged CO intoxication causes significant structural damage to gastric tissues through hypoxia-associated mechanisms.*

Keywords: *Carbon monoxide, stomach, hematoxylin-eosin staining, histology, gastric mucosa, hypoxia, dystrophy, necrosis, inflammation, experimental intoxication.*

Main Part

Introduction

Environmental contamination with toxic gases remains an important public health issue worldwide. Among toxic gaseous compounds, carbon monoxide (CO) occupies a special place because of its widespread occurrence and high toxicity. Carbon monoxide is produced during incomplete combustion of carbon-containing materials and is commonly encountered in industrial emissions, vehicle exhaust, household heating systems, and fires.

The toxic effects of CO are mainly associated with its ability to bind hemoglobin with high affinity, forming carboxyhemoglobin and impairing oxygen transport to tissues. As a result, generalized tissue hypoxia develops, leading to disturbances in cellular metabolism, oxidative stress, vascular dysfunction, and structural tissue injury.

The stomach is highly sensitive to hypoxic damage due to its intensive metabolic activity and dependence on adequate blood supply. Gastric mucosal integrity relies on normal epithelial regeneration, effective microcirculation, and balanced secretory function. Hypoxia-induced disruption of these processes may lead to severe morphological damage.

Histological examination using hematoxylin-eosin staining is one of the most widely used methods for evaluating tissue pathology. Hematoxylin stains cell nuclei blue-purple, while eosin stains cytoplasmic and extracellular structures pink, allowing clear visualization of tissue architecture and pathological alterations.

Despite numerous studies investigating carbon monoxide toxicity, the histological characteristics of gastric injury during chronic CO intoxication remain insufficiently explored. Therefore, the present study aimed to analyze gastric morphological changes in experimental carbon monoxide intoxication using hematoxylin-eosin staining.

Aim of the Study

To investigate histological changes in gastric tissues during experimental carbon monoxide intoxication using hematoxylin-eosin staining.

Materials and Methods

The experimental study was conducted on white laboratory rats weighing 180–220 g. Animals were divided into two groups:

1. Control group;
2. Experimental group exposed to carbon monoxide.

The experimental animals were subjected to controlled carbon monoxide exposure in special chambers for 30 days. Standard laboratory conditions were maintained throughout the experiment.

At the end of the experiment, gastric tissue samples were collected for histological examination. The specimens were fixed in 10% neutral buffered formalin and processed using standard paraffin embedding techniques.

Histological sections measuring 5–7 μm in thickness were prepared and stained with hematoxylin-eosin according to routine histological protocols.

Microscopic analysis focused on the following parameters:

- integrity of the gastric mucosa;
- epithelial morphology;
- condition of gastric glands;
- vascular changes;
- inflammatory infiltration;
- dystrophic and necrotic lesions;
- stromal edema.

Results

Histological Features in the Control Group

Microscopic examination of gastric tissues from the control group demonstrated normal histological architecture.

The gastric mucosa was intact and lined by a well-preserved columnar epithelium. Gastric pits and glands exhibited regular organization and normal structural arrangement.

Parietal cells showed eosinophilic cytoplasm with centrally located nuclei, while chief cells displayed basophilic cytoplasm rich in secretory granules.

The lamina propria contained a normal amount of connective tissue and capillaries without signs of vascular congestion or inflammatory infiltration.

Submucosal and muscular layers preserved their normal morphology.

Histological Changes in Experimental Carbon Monoxide Intoxication

Histological analysis of gastric tissues from animals exposed to carbon monoxide revealed significant pathological alterations.

The superficial epithelium demonstrated varying degrees of dystrophic changes. Vacuolar degeneration of epithelial cells was one of the most common findings. Cytoplasmic swelling and decreased staining intensity were observed in multiple areas.

Focal epithelial desquamation was noted along the mucosal surface. In some sections, superficial erosive defects were identified.

Gastric glands showed marked structural disorganization. Parietal cells exhibited cytoplasmic vacuolization, irregular nuclear contours, and decreased eosinophilia. Many chief cells demonstrated reduced basophilia and loss of secretory granules.

In advanced lesions, glandular atrophy and partial destruction of glandular architecture were evident.

Vascular and Stromal Alterations

Pronounced microcirculatory disturbances were observed in the gastric wall.

Capillaries and venules appeared dilated and congested with erythrocytes. In some areas, vascular stasis and erythrocyte aggregation were present.

The vascular walls demonstrated increased permeability accompanied by perivascular edema. The submucosal layer appeared swollen due to fluid accumulation.

Occasional microhemorrhages were identified within the lamina propria and submucosal tissue.

Endothelial cells of blood vessels exhibited swelling and degenerative changes, suggesting hypoxia-induced vascular injury.

Inflammatory and Necrotic Changes

Inflammatory infiltration was observed in both mucosal and submucosal layers. The infiltrate mainly consisted of lymphocytes, neutrophils, and macrophages.

The severity of inflammation varied among different regions of the stomach but was generally associated with areas of epithelial damage and vascular pathology.

Focal necrotic lesions were detected in several specimens. Necrotic areas were characterized by eosinophilic homogeneous cytoplasm, nuclear pyknosis, karyorrhexis, and loss of cellular boundaries.

In some sections, coagulative necrosis involving superficial glandular structures was identified.

The combination of dystrophic, inflammatory, and necrotic alterations reflected progressive tissue injury caused by chronic carbon monoxide intoxication.

Discussion

The present study demonstrated that chronic carbon monoxide intoxication causes severe histological alterations in gastric tissues.

The earliest pathological changes involved vascular congestion and mild epithelial dystrophy, indicating the initial effects of tissue hypoxia. As exposure continued, structural injury progressed to severe glandular degeneration and focal necrosis.

The mechanism of tissue injury is closely related to hypoxia caused by carboxyhemoglobin formation. Reduced oxygen delivery impairs mitochondrial oxidative phosphorylation and ATP production, resulting in cellular dysfunction and degeneration.

Hematoxylin-eosin staining allowed clear visualization of these pathological processes. Hematoxylin highlighted nuclear alterations such as pyknosis and karyolysis, while eosin effectively demonstrated cytoplasmic degeneration and necrotic changes.

Microcirculatory disturbances played a major role in the progression of tissue damage. Vascular stasis, endothelial injury, and edema further impaired oxygen supply and aggravated hypoxic injury.

Oxidative stress also contributes significantly to cellular damage during CO intoxication. Reactive oxygen species damage membranes, proteins, and intracellular organelles, leading to irreversible cellular injury.

The observed histological findings confirm that chronic carbon monoxide exposure produces cumulative structural damage in gastric tissues.

Conclusion

1. Chronic carbon monoxide intoxication induces significant histological alterations in gastric tissues.
2. Hematoxylin-eosin staining effectively reveals dystrophic, inflammatory, vascular, and necrotic changes in the gastric wall.
3. The main pathological features include epithelial degeneration, glandular disorganization, vascular congestion, edema, and inflammatory infiltration.
4. Prolonged intoxication leads to focal necrosis and destruction of gastric glandular structures.
5. Tissue hypoxia, microcirculatory disturbances, and oxidative stress are the principal mechanisms responsible for gastric injury during CO intoxication.

6. Histological examination using hematoxylin-eosin staining remains an important method for evaluating toxic tissue damage in experimental pathology.

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