ADAPTIVE AND STRUCTURAL RESPONSES OF THE GASTRIC MUCOSA TO COMBINED ALIMENTARY TRACE ELEMENT DEFICIENCY

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ABSTRACT

Combined deficiency of essential trace elements remains a critical issue in experimental morphology due to its impact on cellular metabolism, antioxidant defense, and tissue homeostasis. This study aimed to investigate the adaptive and structural responses of the gastric mucosa under conditions of combined alimentary deficiency of magnesium, iron, zinc, and selenium. The experiment was conducted on white outbred rats divided into control and experimental groups. Trace element deficiency was induced through dietary restriction. Gastric tissue samples were processed using standard histological techniques and stained with hematoxylin and eosin for microscopic evaluation. The control group demonstrated normal histological organization of the gastric mucosa, including intact epithelial lining, well-structured gastric glands, and absence of edema or inflammation. In contrast, the experimental group exhibited significant morphological alterations. These included epithelial dystrophy, disruption of epithelial integrity, glandular disorganization, stromal edema, vascular dilation, and lymphohistiocytic infiltration. The observed changes indicate both adaptive and pathological remodeling processes in response to micronutrient deficiency. The findings highlight the essential role of trace elements in maintaining gastric mucosal integrity and suggest that their deficiency leads to impaired barrier function and increased susceptibility to tissue damage. This study contributes to understanding the mechanisms underlying gastric mucosal pathology associated with micronutrient imbalance.

KEYWORDS:

gastric mucosa, trace element deficiency, magnesium, iron, zinc, selenium, epithelial dystrophy, histology, experimental rats, mucosal homeostasis, inflammation, microcirculation, glandular structure

Relevance. Combined alimentary trace element deficiency remains an important issue in experimental morphology and biomedical research, since magnesium, iron, zinc, and selenium are involved in cellular metabolism, antioxidant protection, epithelial renewal, and tissue homeostasis. The gastric mucosa is characterized by active cellular regeneration and performs essential protective, secretory, and barrier functions. Therefore, it is highly sensitive to disturbances in trace element balance. Deficiency of these essential elements may lead to epithelial dystrophy, glandular disorganization, stromal edema, vascular disorders, and inflammatory cell infiltration. In this regard, the study of adaptive and structural responses of the gastric mucosa to combined trace element deficiency is relevant for understanding the mechanisms of gastric tissue damage and impairment of mucosal homeostasis.

Objective of the study. To investigate the adaptive and structural responses of the gastric mucosa to combined alimentary deficiency of magnesium, iron, zinc, and selenium.

Materials and methods.

The study was performed on white outbred rats divided into control and experimental groups. Animals in the control group were maintained on a standard diet. In the experimental group, combined alimentary trace element deficiency was modeled by limiting the intake of magnesium, iron, zinc, and selenium.

After completion of the experimental period, gastric tissue samples were collected and fixed in 10% neutral formalin. The material was processed according to standard histological techniques, embedded in paraffin, and sectioned. Histological sections were stained with hematoxylin and eosin. Microscopic examination was used to evaluate the condition of the surface-pit epithelium, gastric glands, lamina propria, microcirculatory vessels, stromal edema, and inflammatory cell infiltration.

Results.

n the control group, the gastric mucosa retained its typical histological organization. The surface epithelium was continuous, gastric pits were regularly arranged, and the glandular structures demonstrated normal orientation. The lamina propria showed no signs of pronounced edema, vascular congestion, or inflammatory infiltration.

In rats with combined alimentary trace element deficiency, marked structural changes were observed in the gastric mucosa. The surface-pit epithelium showed signs of dystrophic alteration, including cellular swelling, irregular epithelial arrangement, and partial loss of structural integrity in some areas. These findings indicated weakening of the protective epithelial barrier.

The glandular component of the gastric mucosa also demonstrated visible changes. Gastric glands became less regularly arranged, and some glandular structures showed deformation and signs of functional stress. These alterations reflected impaired trophic support and reduced structural stability of the mucosal layer under trace element deficiency.

Changes in the lamina propria were characterized by stromal edema, dilation of microcirculatory vessels, and moderate vascular congestion. Increased lymphohistiocytic infiltration was also observed, indicating activation of a local inflammatory response. The combination of epithelial dystrophy, glandular disorganization, vascular disturbances, and cellular infiltration demonstrated that the gastric mucosa responds to trace element deficiency through both adaptive and pathological structural remodeling.

Conclusion.

Combined alimentary deficiency of magnesium, iron, zinc, and selenium leads to significant adaptive and structural changes in the gastric mucosa. The most characteristic findings include epithelial dystrophy, weakening of the mucosal barrier, glandular disorganization, stromal edema, vascular disturbances, and inflammatory infiltration. These results confirm the essential role of trace elements in maintaining the structural homeostasis and protective function of the gastric mucosa.

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