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HISTOPATHOLOGICAL PATTERNS OF GASTRIC INJURY ASSOCIATED WITH MAGNESIUM, IRON, ZINC AND SELENIUM DEFICIENCY

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Abstract

This article presents an experimental histopathological assessment of gastric injury associated with magnesium, iron, zinc and selenium deficiency. The study was performed on 3- and 6-month-old white outbred rats. The gastric mucosa was evaluated under isolated deficiency of Mg, Fe, Zn and Se, as well as under combined Mg+Fe+Zn+Se deficiency. The results showed that each type of microelement deficiency produced a specific pattern of gastric injury. Magnesium deficiency was mainly associated with reduced secretory and regenerative activity, iron deficiency with damage to parietal cells, zinc deficiency with impairment of the epithelial barrier, and selenium deficiency with dystrophic and inflammatory changes. Combined deficiency caused the most severe injury, involving the epithelium, gastric glands, lamina propria, vascular bed and proliferative activity of the mucosa.

Keywords: stomach, gastric injury, histopathology, magnesium, iron, zinc, selenium, microelement deficiency, gastric mucosa.

Introduction

The gastric mucosa is a complex structural and functional system that provides digestion, secretion, protection and local tissue homeostasis. Its normal condition depends on epithelial integrity, glandular activity, adequate microcirculation and continuous cellular renewal.

Essential microelements play an important role in maintaining these processes. Magnesium is involved in enzymatic reactions and cellular metabolism, iron is important for oxygen transport and tissue trophism, zinc supports epithelial integrity and regeneration, while selenium participates in antioxidant defense and regulation of inflammation. Deficiency of these elements may lead to different forms of gastric mucosal injury.

Experimental comparison of isolated and combined microelement deficiency makes it possible to identify specific histopathological patterns of gastric damage and determine which structural components are most vulnerable.

Aim of the study

To identify histopathological patterns of gastric injury in 3- and 6-month-old white outbred rats under isolated and combined deficiency of magnesium, iron, zinc and selenium.

Materials and methods

The study was performed on gastric specimens of 3- and 6-month-old white outbred rats. The animals were divided into control groups and experimental groups with alimentary-induced deficiency of magnesium, iron, zinc and selenium, as well as a group with combined Mg+Fe+Zn+Se deficiency. In each age group, the changes were evaluated separately.

Histological sections were stained with hematoxylin and eosin. Microscopic analysis included assessment of the surface-pit epithelium, gastric glands, lamina propria, vascular bed, stromal edema, epithelial dystrophy, desquamation and inflammatory infiltration. Morphometric evaluation included mucosal thickness, gastric pit depth, gland length, number of gastric glands, parietal cells and chief cells. Ki-67 expression was used to assess proliferative activity of the gastric mucosa. These parameters correspond to the methodological recommendations developed for the assessment of the stomach in alimentary-induced microelementosis.

Results

In the control groups, the gastric mucosa preserved its typical histological structure. The surface epithelium was continuous, gastric pits and glands were regularly arranged, and the lamina propria showed no pronounced edema or inflammatory infiltration. In 6-month-old control rats, morphometric indicators were higher than in 3-month-old rats, reflecting age-related morphofunctional maturation of the stomach.

Magnesium deficiency was characterized by moderate thinning of the mucosa, smoothing of the mucosal relief, reduction of gastric pit depth and decreased glandular density. These changes indicated weakening of secretory and regenerative activity.

Iron deficiency produced a mainly dystrophic and atrophic pattern of injury. The most sensitive indicator was a decrease in the number of parietal cells. In 3-month-old rats, their number decreased to 24.5±2.5 per 10000 μm², and in 6-month-old rats to 27.5±2.5 per 10000 μm². This reflected impairment of the acid-forming function of the stomach.

Zinc deficiency was mainly associated with epithelial damage. The surface-pit epithelium showed signs of disorganization, and the protective barrier of the mucosa was weakened. In 3-month-old rats, mucosal thickness was 663.4±27.7 μm, gastric pit depth was 71.6±3.3 μm, and the number of glands was 19.5±2.5 per 100000 μm².

Selenium deficiency caused the most pronounced changes among isolated forms of microelement deficiency. In 3-month-old rats, mucosal thickness decreased to 618.9±25.2 μm, gastric pit depth to 66.2±4.2 μm, and gland number to 16.0±1.0 per 100000 μm². Morphologically, these changes were accompanied by cytoplasmic vacuolization, hydropic dystrophy of epithelial cells, stromal edema and lymphohistiocytic infiltration.

Combined Mg+Fe+Zn+Se deficiency caused the most severe gastric injury. In 3-month-old rats, mucosal thickness decreased to 604.1±25.7 μm, gastric pit depth to 61.1±3.3 μm, gland number to 16.0±2.0, parietal cells to 22.0±3.0, and chief cells to 35.0±4.0. In 6-month-old rats, the changes were even more pronounced: mucosal thickness was 675.2±26.9 μm, gastric pit depth was 88.7±4.0 μm, parietal cells decreased to 21.0±3.0, and chief cells to 34.0±4.0. The methodological data indicate that combined deficiency leads to deeper damage of the gastric mucosa than isolated deficiency forms.

Table 1.

Main histopathological patterns of gastric injury

Type of deficiency	Main histopathological pattern
Magnesium deficiency	Reduced secretory and regenerative activity
Iron deficiency	Predominant injury of parietal cells
Zinc deficiency	Damage to the epithelial barrier
Selenium deficiency	Dystrophic and inflammatory changes
Combined Mg+Fe+Zn+Se deficiency	Complex injury of epithelium, glands, stroma and vessels

Ki-67 expression also differed between groups. In the control group, Ki-67 expression was 7.0±2.0% in 3-month-old rats and 8.0±2.0% in 6-month-old rats. Under combined deficiency, Ki-67 expression increased to 22.0±4.0% and 27.0±5.0%, respectively. However, the distribution of positive cells became disorganized, indicating impaired regulation of epithelial renewal.

Conclusion

Isolated deficiency of magnesium, iron, zinc and selenium produces different histopathological patterns of gastric injury.

Magnesium deficiency is mainly associated with reduced regenerative and secretory activity, iron deficiency with parietal cell injury, zinc deficiency with epithelial barrier damage, and selenium deficiency with dystrophic and inflammatory changes.

Combined Mg+Fe+Zn+Se deficiency causes the most severe gastric injury, involving epithelial, glandular, stromal and vascular components of the mucosa.

Disorganized Ki-67 expression under combined deficiency indicates impaired regulation of epithelial renewal and reduced reparative capacity of the gastric mucosa.

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