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Pathomorphological Alterations of The Gastric Mucosa in Individuals with Metabolic Syndrome and A Body Mass Index Ranging From 27.5 To 34.9 Kg/M²

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Abstract: In metabolic syndrome with a body mass index of 27.5 kg/m², the development of pronounced compensatory-adaptive mechanisms in the active zones of the gastric mucosa results in a number of morphological alterations. These include massive lymphocytic infiltration in the areas between the fundal glands of the mucosa, the covering epithelium, the submucosal layer, and the muscular layer; sclerosed stromal structures; atrophically altered glandular cells; and the presence of coarse fibrous connective tissue components between muscle bundles. In addition, marked atrophic and metaplastic changes of the glandular epithelium are observed in the antral and fundal regions of the stomach, as well as the formation of Paneth cells in the mucosa of the pyloroantral region. Such alterations are considered precancerous conditions and represent morphological criteria that constitute a direct indication for sleeve (longitudinal) gastric resection.

Keywords: Pathomorphology, stomach, metaplasia, atrophy, sclerosis, fundal, antral.

Introduction: Worldwide, the prevalence of metabolic syndrome characterized by increased body weight, visceral obesity, and disturbances in carbohydrate and lipid metabolism has increased by approximately 21% over the past decade. In the United States, this indicator has risen by 34% over the last five years, while in European countries it has increased by 20.8%. Metabolic syndrome accounts for approximately 56% among individuals over 40 years of age and 44% among those over 50 years of age. From an economic perspective, this leads to the premature morbidity of the working-age population and requires substantial financial expenditures within national healthcare systems.

In the Russian Federation and Central Asian countries, the average prevalence of metabolic syndrome is about 16.8%. It is observed in 51% of individuals over 50 years of age, 36% among those over 40 years of age, 11% among individuals aged 20–30 years, and 9% in other age groups. These data confirm the relevance of the problem and highlight the necessity of studying age-related morphological changes in the gastrointestinal tract of patients with this condition. Such investigations are essential for improving the quality of life of the population. In particular, in our country more than 4.2 million women suffer from metabolic syndrome, of whom 38% are of reproductive age. This is of great importance for maternal and child health protection and further emphasizes the urgency of the present topic. In Uzbekistan, expenditures allocated for the restoration of reproductive health in women with metabolic syndrome have been steadily increasing: in 2022, approximately 22% of the total healthcare budget was directed toward this purpose, while in 2023 this figure increased to 36%. The present study provides data on clinically and morphologically interrelated changes associated with metabolic syndrome, including gastric enlargement, increased gastric volume, and the development of gallstone

disease. However, in the scientific literature of foreign countries and the CIS states, there is limited information regarding the specific morphological and immunohistochemical changes of the stomach in metabolic syndrome. Therefore, further research in this field will make it possible to develop evidence-based recommendations for medical practice.

Research objective

To study the pathomorphological changes of the gastric mucosa in metabolic syndrome.

DISCUSSION AND RESULTS

In this group, strict indications for bariatric surgery of the stomach were identified. Macroscopically, the gastric mucosa was characterized by the presence of numerous erosive foci, as well as atrophic and sclerotic changes of the mucosal layer. In addition, excessive mucus secretion and swelling of the mucosal surface were observed.

Microscopically, the epithelial layer covering the mucosa showed numerous areas of desquamation. A large number of micro-scars composed of young connective tissue covered with loose fibrous connective tissue were identified. These pathological changes extended along the inner surface of the stomach from the cardiac region to the pyloric region and were also characterized by the presence of small ulcerative lesions in the antral and pyloroantral areas.

Most of the covering epithelial cells demonstrated polymorphic structural features. Their cytoplasm consisted of dense, homogeneous eosinophilic protein substrates and exhibited an uneven texture and variable thickness. In the cardiac region, the covering epithelium appeared thickened, with signs of mucoid swelling and a dull homogeneous cytoplasmic appearance, accompanied by multifocal hyperplastic foci. The cytoarchitectonics of the covering epithelium were altered, showing a multilayered structure: the superficial layers appeared fragmented, while the deeper layers showed enlarged cellular structures. In the fundal, antral, and pyloroantral regions, the covering epithelium exhibited hyperplastic changes of varying height, with the presence of metaplastic foci characterized by flattened multilayered epithelial structures. At this stage, a physiological process normally occurs in which easily digestible carbohydrates are rapidly broken down and absorbed in the stomach. Under the influence of the hormone leptin, the satiety center in the hypothalamus is stimulated, which typically leads to cessation of food intake within approximately 20–30 minutes after eating. However, the combined development of hyperplastic, metaplastic, and atrophic changes on the surface of the gastric mucosa creates resistance to the absorption of simple carbohydrates, which in turn leads to increased food intake in both quantity and volume.

At the same time, previously developed superficial active gastritis spreads to the middle and deeper layers of the mucosa. According to the OLGA classification, inflammatory changes involve all glandular cells of the fundal glands and extend to the deep mucosal and submucosal layers. As a result, pronounced development of chronic inflammatory foci in MALT structures was observed in the entire thickness (3/3) of the 0.5 mm mucosal layer and in the submucosa, accompanied by increased infiltration of intraepithelial lymphocytes. In the stromal structures of the mucosa, proliferation of coarse fibrous connective tissue was observed, along with irregular vascular branching, sclerotic changes in certain areas, signs of vascular congestion, and the presence of necrobiotic cells of the fundal glands. One of the most notable findings was the alteration in the functional state of the fundal glands (see Figure 1).

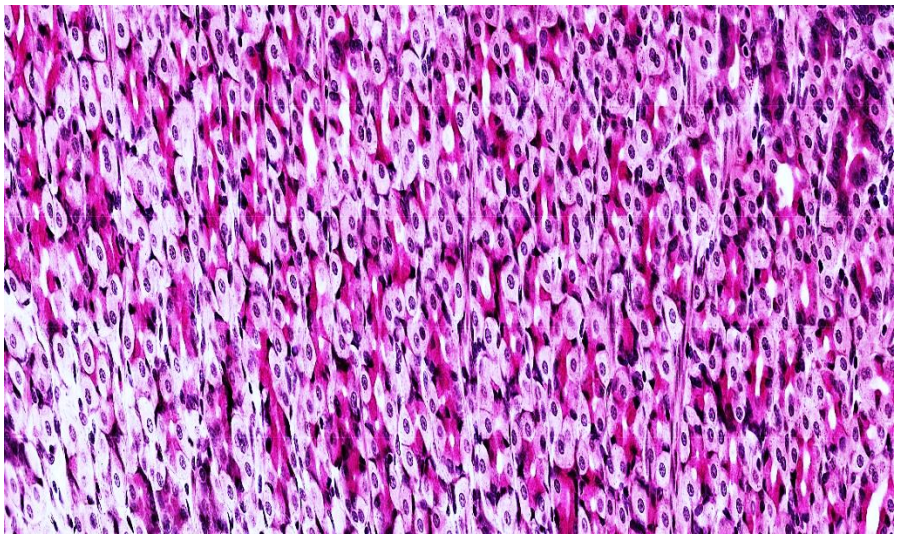

Figure 1. Group 2. Gastric mucosa. Massive accumulation and stagnation of secretions in the lumen of the

fundal glands are observed, appearing as pink homogeneous protein substrates. Parietal cells are increased in number, and their cytoplasm shows a pale eosinophilic appearance. A small number of lymphocytes are detected in the interstitial areas. Hematoxylin and eosin (H&E) staining. Magnification: ×10 ×10.

Cellular characteristics. Parietal cells appear separated and exhibit altered localization. They vary in size and show densely eosinophilic cytoplasm. The surrounding basal membrane is thickened, and the cells are located among fragmented fibrous structures. Foci of diapedetic hemorrhage are observed in the surrounding areas.

Within a ×100 microscopic field, parietal cells demonstrate considerable variability in size. Dense eosinophilic protein deposits are detected in the pericellular regions. The accumulation of these mucinous deposits indicates stagnation of mucin secretion caused by deformation of the isthmus–neck region of the fundal glands, suggesting the presence of functional insufficiency in these cells (see figure 2 and 3).

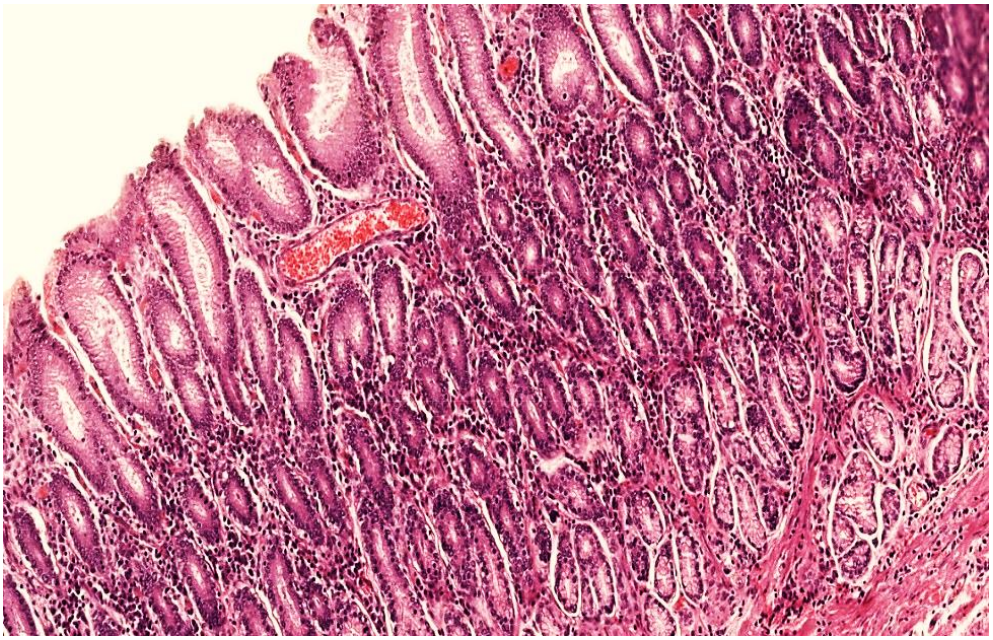

Figure 2. Group 2. Gastric mucosa. Accumulation of homogeneous pink protein substrates and mucin deposits is observed around the parietal cells of the fundal glands. Stagnation of secretions in the glandular lumen and atrophic changes in the fundal gland epithelium are also noted. Hematoxylin and eosin (H&E) staining. Magnification: ×10 ×10.

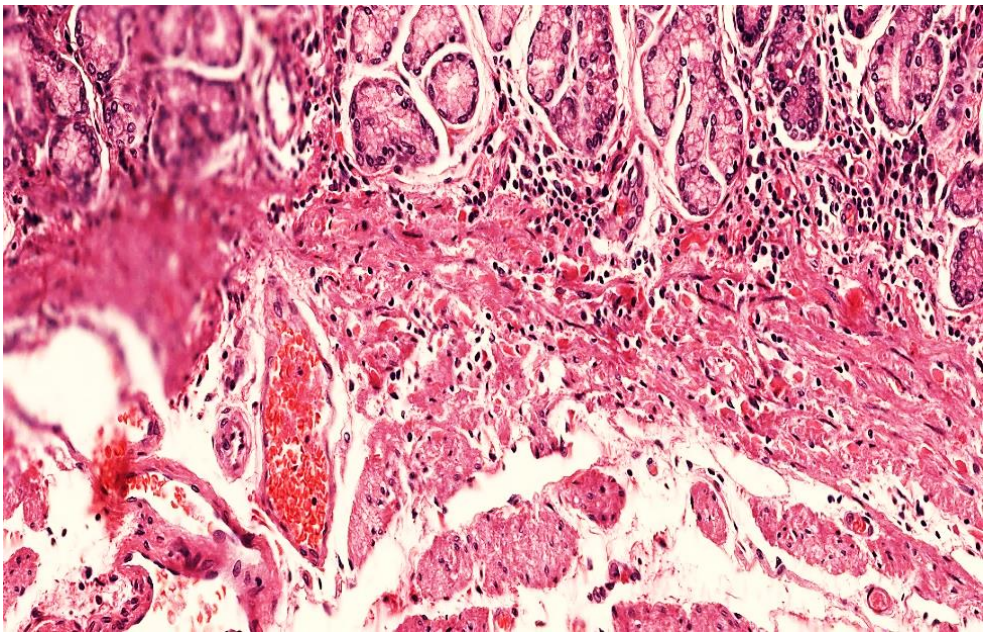

Figure 3. Gastric muscular and submucosal layers. Massive foci of lymphocytic infiltration are observed in the muscular layer and submucosal layer of the stomach. Most blood vessels appear congested. Hematoxylin and eosin (H&E) staining. Magnification: ×10 ×10.

In most areas of the mucosa, atrophic changes are observed in all cellular components of the fundal glands, along with an increased number of cells undergoing metaplastic transformation. Massive foci of lymphocytic infiltration are identified in the stromal structures. The blood vessels appear congested, and foci of plasmatic edema are detected in the submucosal and muscular layers (see Figures 4, 5, 6, and 7).

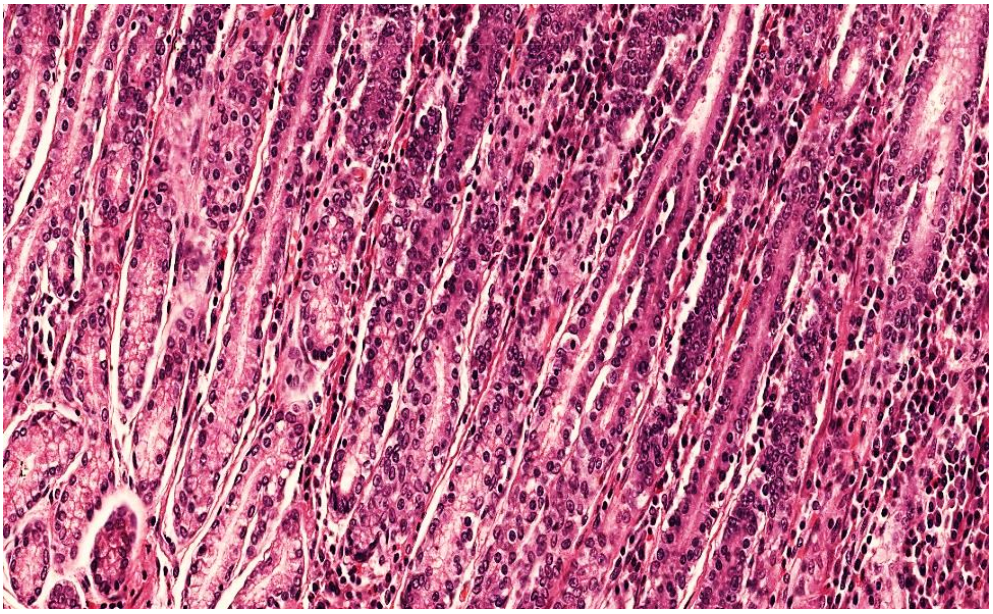

Figure 4. Gastric mucosa. Around the parietal cells of the fundal glands, homogeneous pink protein substrates and mucin deposits are accumulated in the form of precipitates. Stagnation of secretions in the glandular lumen and atrophic changes in the fundal gland epithelium are also observed. Hematoxylin and eosin (H&E) staining. Magnification: ×10 ×10.

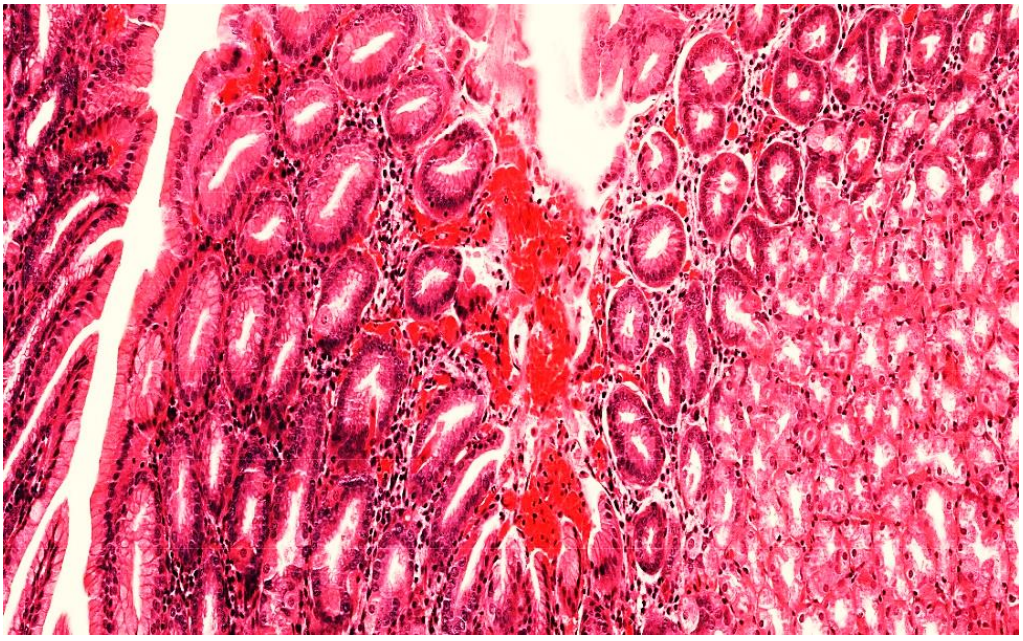

Figure 5. Group 2. Gastric mucosa. The blood vessels beneath the covering epithelium of the mucosa exhibit irregular congestion, with lymphocytic infiltration and interstitial edema present in the surrounding areas. Hematoxylin and eosin (H&E) staining. Magnification: ×10 ×10.

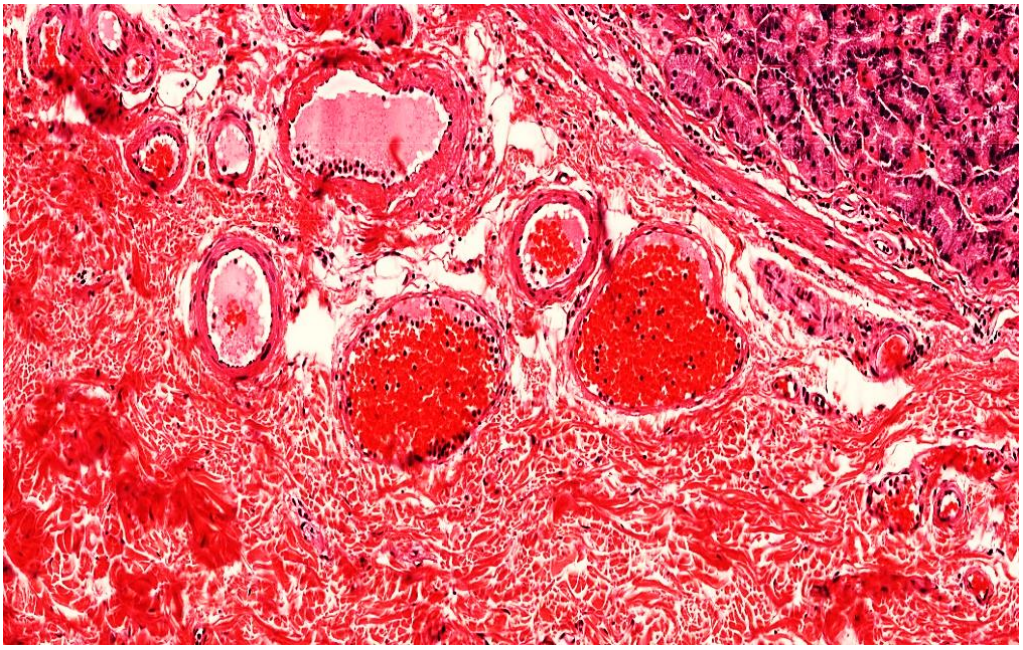

Figure 6. Group 2. Gastric muscular layer. The blood vessels in the muscular layer exhibit congestion, accompanied by interstitial edema. In the submucosal layer, massive interstitial edema is observed, primarily of a plasmatic nature. Inflammatory infiltrates are present around the vessels. Hematoxylin and eosin (H&E) staining. Magnification: ×20 ×10.

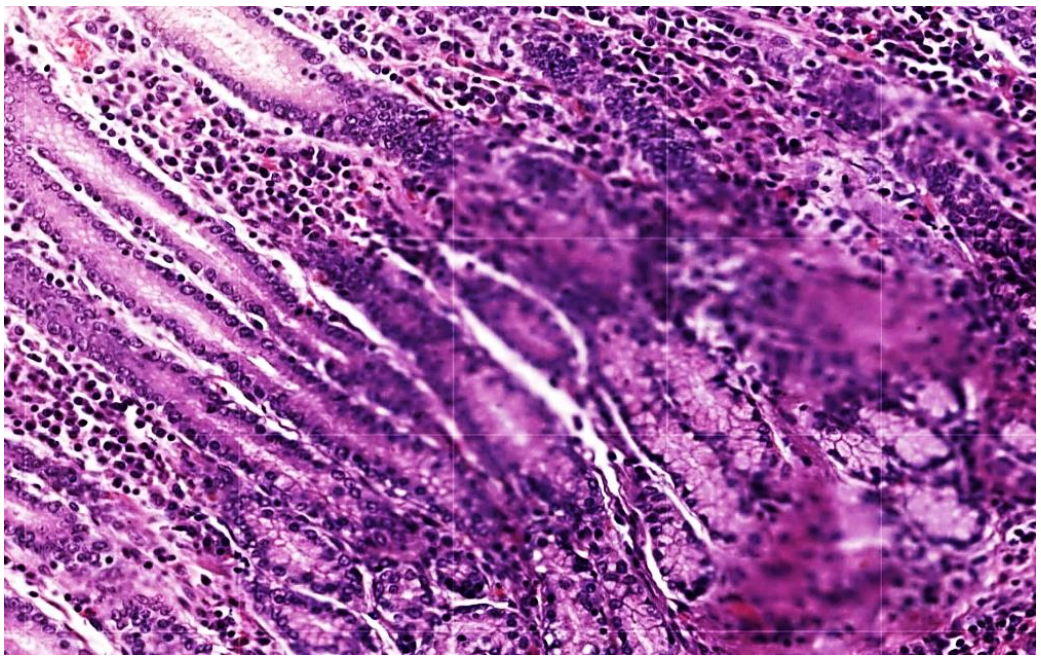

Figure 7. Gastric mucosa. The blood vessels beneath the covering epithelium of the mucosa show irregular congestion, with lymphocytic infiltration and interstitial edema present in the surrounding areas. Hematoxylin and eosin (H&E) staining. Magnification: ×10 ×10.

In this group, profound changes in the gastric mucosa were observed. According to the OLGA and OLGIM classifications, inflammatory infiltrates were present in the superficial, middle, and deep layers of the mucosa, and metaplastic foci were identified in all glandular cells. Fundal glands in the cardiac, antral, and pyloric regions showed transformation of epithelial cells into Paneth cells, with an increased number of atrophic cells. The interstitial stroma exhibited fibrous connective tissue proliferation, and perivascular plasmatic edema led to the development of metachromasia.

All of these morphological alterations were primarily chronic processes, reflecting compensatory adaptive mechanisms predominantly in the form of atrophic and hyperplastic changes, followed by metaplasia. Hyperplastic foci remained in the active zones of the glands. These changes were associated with chronic

gastritis, with 70% of cases showing Helicobacter pylori infection, which contributed to the development of ulcers and erosive lesions. In our study, patients with a body mass index (BMI) of 27.5 kg/m² or higher predominantly presented with hypertension, diabetes mellitus, obesity, hyperglycemia, and a loss of sexual activity. These individuals were at increased risk for precancerous changes in the gastric mucosa. Observed alterations included deep metaplasia of the fundal gland epithelium in the superficial, medial, and deep layers, atrophic and dysplastic changes in the glands, and mucosal involvement corresponding to OLGA stage 3 and OLGIM stage 3, for a total of 6 points.

Consequently, atrophic changes and metaplasia in the antral and fundal glands were classified as grade 3 for both parameters, summing to 6 points. The overall risk of gastric cancer in these patients was assessed as moderate to high (See Table 1).

Table-1

Algorithm for pathomorphological assessment (scoring system)

The assessment is performed based on changes observed in the antrum (3 biopsy samples) and the body (2 biopsy samples) regions.

Parameter	0 points	1 point (Mild)	2 points (Moderate)	3 points (Severe)
OLGA (Atrophy)	None	Loss of <30% of glands	Loss of 30–60% of glands	Loss of >60% of glands
OLGIM (Metaplasia)	None	Intestinal metaplasia <30%	Intestinal metaplasia 30–60%	Intestinal metaplasia >60%

Important note: In patients with metabolic syndrome, as the degree of atrophy (OLGA) increases, the density of ghrelin-producing cells decreases, which may weaken the metabolic response after surgery. Therefore, in patients from Group 1, bariatric surgery should be deferred, and therapeutic measures targeting the gastric mucosa should be applied instead. This approach can reduce metabolic disturbances, lower blood glucose levels, and contribute to a decrease in body weight.

CONCLUSION

1. Surgical approach based on OLGA/OLGIM stage: If a patient is diagnosed with OLGA/OLGIM stage III or IV, it is recommended to avoid the standard Roux-en-Y gastric bypass procedure. The reason is that the remaining stomach (blind pouch) becomes inaccessible for endoscopic monitoring, making early detection of potential malignancy impossible. In such cases, Sleeve Gastrectomy (longitudinal resection) is preferable, as it allows continuous visual inspection of the gastric mucosa.

2. Mandatory Helicobacter pylori eradication: Even if patients with metabolic syndrome are classified as OLGA/OLGIM stage I–II, eradication therapy for H. pylori should be completed at least 4 weeks before surgery. This helps halt the progression of atrophy and metaplasia and reduces the risk of postoperative anastomotic ulcers and inflammation in the residual stomach.

3. Biochemical monitoring of metabolic status: Before and after surgery, assessment should not be limited to the OLGA stage alone. The GastroPanel (Pepsinogen I, Pepsinogen II, and Gastrin-17) should also be evaluated. In metabolic syndrome, low ghrelin levels combined with decreased Pepsinogen I indicate gastric mucosal atrophy (OLGA III–IV). These markers allow prediction of the patient's postoperative ability to absorb vitamin B12 and iron.

4. Individualized endoscopic follow-up schedule: Postoperative endoscopic monitoring should be tailored according to the patient's OLGA/OLGIM stage:

- OLGA I–II: Perform esophagogastroduodenoscopy (EGD) once every 3–5 years.

- OLGA III–IV: Mandatory endoscopic monitoring every 1–2 years, with biopsy of any "suspicious" areas.

This strategy allows timely detection of secondary changes in the gastric mucosa in the context of metabolic syndrome.

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