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MORPHOLOGICAL ANALYSIS OF STRUCTURAL REMODELING OF THE MUCOSA AND FIBROTIC PROCESSES IN PYLORODUODENAL STENOSIS

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Abstract: This study evaluates the morphological and morphometric characteristics of the stenotic area in pyloroduodenal stenosis according to disease stages. The study included 54 patients, with biopsy samples obtained exclusively from the ulcer-scarred pyloroduodenal region at five points per patient. The specimens were analyzed at the levels of the mucosal, submucosal, and upper muscular layers. The results demonstrated progressive mucosal thinning, reduction of glandular volume, and intensification of inflammatory and fibrotic changes with advancing stages of pyloroduodenal stenosis.
Keywords: pyloroduodenal stenosis, morphological analysis, morphometry, fibrosis, mucosa.

In recent years, particular attention in medical science has been focused on cardiovascular diseases, especially ischemic heart disease and arterial hypertension, which is explained by their high prevalence and significant impact on life expectancy [1–3]. At the same time, the regulation of biochemical and metabolic processes occurring in the body, as well as the development of new pharmacological agents, including drugs with detoxifying and cytoprotective properties, remains a relevant issue [4,5].

Under these conditions, certain aspects of diseases of the digestive system—particularly the late complications of gastric and duodenal disorders—have been studied to a lesser extent. Nevertheless, pathologies of the pyloroduodenal region, especially pyloroduodenal stenosis, continue to represent one of the important problems in modern gastroenterology and surgery [6,7]. Due to the complexity of their etiology and pathogenesis, as well as the diversity of conservative and surgical treatment approaches, these conditions occupy a special place among diseases of the digestive system.

Although advances in modern diagnostic and therapeutic approaches have led to a certain reduction in the number of elective surgical interventions, profound morphological remodeling of tissues, the development of fibrotic processes, and their clinical significance in pyloroduodenal stenosis remain highly relevant issues [8,9]. Clinical practice indicates that the severity of the stenotic process is directly associated not only with functional disorders but also with structural changes occurring in the pyloroduodenal region.

Chronic inflammatory processes, progressive development of fibrous tissue, and remodeling of the mucosal, submucosal, and muscular layers play a crucial role in the pathogenesis of pyloroduodenal stenosis [10,11]. The identification of *Helicobacter pylori* infection has demonstrated the leading role of inflammatory processes in the formation of morphological changes in the pyloroduodenal zone. Against the background of chronic inflammation, protective mechanisms of the mucosa are weakened, resulting in impaired regeneration and excessive proliferation of fibrotic tissue [12–14].

At the same time, the literature insufficiently addresses the depth of morphological and ultrastructural changes in pyloroduodenal stenosis, their relationship with clinical manifestations, as well as their association with hormonal and immune mechanisms [15]. In particular, the

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degree of fibrotic processes, fibroblast activity, inflammatory cell infiltration, and morphometric parameters in relation to the severity of stenosis remain poorly studied.

One of the pressing issues is the comprehensive assessment of the structural condition of the mucosal, submucosal, and muscular layers in pyloroduodenal stenosis, as well as their proportions and variability. Morphological studies are of significant scientific and practical importance for a deeper understanding of the pathogenesis of stenosis, identification of its stages of development, and optimization of treatment strategies [16–18].

In addition, issues related to ontogenesis, regenerative potential of tissues, early aging processes, and their influence on the course of pyloroduodenal stenosis have not been sufficiently explored in the scientific literature [19]. Therefore, a thorough and systematic investigation of morphological changes occurring in the pyloroduodenal region remains highly relevant for modern surgery and gastroenterology.

The above considerations served as the basis for conducting the present study, aimed at investigating the morphological characteristics of tissues in the stenotic area in pyloroduodenal stenosis, analyzing structural changes, and evaluating their clinical significance.

Aim. To determine the morphological and morphometric characteristics of the mucosa in the stenotic area in pyloroduodenal stenosis and to assess the severity of structural changes.

Materials and Methods. The study was conducted in 54 patients with a confirmed diagnosis of pyloroduodenal stenosis. All patients underwent comprehensive clinical, endoscopic, and morphological examinations, enabling an integrated assessment of the degree of stenosis development and associated structural changes in the pyloroduodenal region. The patients' age ranged from 28 to 71 years, with a mean age of 49.6 ± 10.8 years. The study cohort included 38 men (70.4%) and 16 women (29.6%).

According to clinical and endoscopic criteria, patients were divided into three groups based on the stage of pyloroduodenal stenosis. Compensated stage I stenosis was identified in 17 patients (31.5%). Subcompensated stage II stenosis was diagnosed in 21 patients (38.9%). Decompensated stage III pyloroduodenal stenosis was observed in 16 patients (29.6%). This distribution allowed a stepwise analysis of the morphological severity of the stenotic process.

All patients underwent standard general clinical examinations, including complete blood and urine tests, biochemical blood analysis, electrocardiography, abdominal radiographic examinations, and fibrogastroduodenoscopy. During endoscopic evaluation, anatomical deformation of the pyloroduodenal region, the degree of stenosis, and the severity of mucosal hyperemia, erosions, and cicatricial changes were assessed.

For morphological analysis, biopsy specimens were obtained exclusively from the stenotic area of the pyloroduodenal region altered by ulcerative scarring. In each patient, biopsies were collected from five anatomical sites within the stenosis zone (proximal, distal, anterior wall, posterior wall, and lateral wall), yielding a total of 270 biopsy samples (54 × 5). Biopsy sampling was performed once in all patients during the initial endoscopic examination.

The obtained biopsy specimens included the following layers of the pyloroduodenal region: mucosa, submucosa, and the superficial portion of the muscular layer. This approach enabled a comprehensive assessment of both superficial and deep structural alterations associated with the stenotic process.

Histological examinations were performed according to a standard protocol. Biopsy specimens were fixed in 10% neutral buffered formalin, embedded in paraffin blocks, and sectioned at a thickness of 4–5 µm. Hematoxylin and eosin staining was applied in all 54 patients for general morphological evaluation. To assess the severity of fibrotic changes, special connective tissue

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staining techniques were used in 41 patients (75.9%). *Helicobacter pylori* infection was evaluated histologically in all 54 patients by assessing the degree of bacterial colonization.

Morphometric analysis was performed in all patients. The study assessed mucosal thickness, deformation of glandular structures, relative area of fibrotic tissue, fibroblast density, and inflammatory cell infiltration. Each parameter was measured in at least five microscopic fields of view, and mean values were calculated for each patient.

Statistical analysis was carried out using mathematical data processing methods. Data were expressed as mean (M) ± standard deviation (SD). Parametric and nonparametric biostatistical tests were applied to evaluate differences between groups. Differences in morphometric parameters among stenosis stages were assessed using analysis of variance. Correlations between clinical and morphological parameters were analyzed using correlation analysis. Statistical significance was set at $p < 0.05$.

Main morphometric parameters of the mucosa according to the stages of pyloroduodenal stenosis (M ± SD)

Indicators	I-step (n=17)	II-step (n=21)	III-step (n=16)
Mucosal thickness, μm	1120,4 ± 52,1	1048,6 ± 28,4	945,7 ± 34,9
Epithelial height, μm	21,2 ± 1,6	22,8 ± 1,9	24,6 ± 2,4
Relative volume of the glandular apparatus	0,61 ± 0,05	0,58 ± 0,04	0,52 ± 0,06
Depth of gastric pits (foveolae), μm	238,6 ± 14,2	224,9 ± 11,8	209,3 ± 10,5

The table presents the main morphometric parameters of the mucosa according to the stages of pyloroduodenal stenosis (PDS). Data are expressed as mean ± standard deviation (M ± SD). Differences between groups were assessed using one-way analysis of variance (ANOVA). A p-value < 0.05 was considered statistically significant.

Results. Morphological and morphometric parameters of the mucosa in the stenotic area in pyloroduodenal stenosis were found to change progressively in accordance with the stage of the stenotic process. Analysis of biopsy specimens demonstrated that structural remodeling in the pyloroduodenal region became more pronounced with increasing severity of stenosis.

In 17 patients with stage I pyloroduodenal stenosis, mucosal thickness was relatively preserved, with a mean value of 1120.4 ± 52.1 μm. The integrity of the epithelial layer was largely maintained, and the relative volume of the glandular apparatus was assessed at 0.61 ± 0.05 arbitrary units.

The proportion of intraepithelial lymphocytes was 5.2 ± 0.7%, indicating low inflammatory activity. The density of inflammatory infiltration was 13.8 ± 1.3%, while the degree of *Helicobacter pylori* colonization was 0.7 ± 0.2 points. At this stage, morphological changes were predominantly compensatory and reversible.

In 21 patients with stage II pyloroduodenal stenosis, mucosal thickness decreased compared with stage I, averaging 1048.6 ± 28.4 μm. The relative volume of glandular structures was 0.58 ± 0.04 arbitrary units, accompanied by architectural distortion and deformation of glands.

The proportion of intraepithelial lymphocytes increased to 6.6 ± 0.9%, and the density of inflammatory infiltration rose to 15.1 ± 1.6%. Moderate proliferation of fibrotic tissue in the

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submucosa was detected, along with increased fibroblast activity. The degree of *Helicobacter pylori* colonization was 0.8 ± 0.3 points.

In 16 patients with stage III pyloroduodenal stenosis, mucosal thickness markedly decreased to 945.7 ± 34.9 µm. The relative volume of the glandular apparatus was 0.52 ± 0.06 arbitrary units, with pronounced glandular atrophy and complete destruction in certain areas.

The proportion of intraepithelial lymphocytes reached 7.4 ± 1.2%, while inflammatory infiltration density increased to **17.6 ± 1.9%**. Extensive coarse fibrosis of the submucosal layer was observed, with extension of fibrotic changes into the superficial muscular layer and associated muscle fiber atrophy. The degree of *Helicobacter pylori* colonization was 0.9 ± 0.4 points, indicating deep and largely irreversible morphological alterations.

The obtained morphological and morphometric findings demonstrate that progression of pyloroduodenal stenosis is characterized by progressive thinning of the mucosa, expansion of fibrotic tissue, and intensification of inflammatory infiltration. At the same time, the degree of *Helicobacter pylori* colonization did not differ significantly among stenosis stages ($p > 0.05$), confirming that long-standing fibrosis and structural remodeling play a dominant role in the pathogenesis of pyloroduodenal stenosis.

Discussion. The conducted morphological and morphometric investigations demonstrated a clear relationship between the stepwise progression of the stenotic process in pyloroduodenal stenosis and the structural alterations occurring in the mucosa. According to the data presented in Table 1, at stage I PDS the thickness of the mucosa and the volume of the glandular apparatus were relatively preserved, indicating the adequacy of compensatory mechanisms at this stage.

At stage II, thinning of the mucosa, shortening of foveolar structures, and disruption of glandular architecture were identified. These changes suggest that during the subcompensated stage, morphological remodeling processes become stable and progressive. The data presented in Table 2 confirm a marked increase in inflammatory infiltration and fibrotic changes at this stage.

In stage III pyloroduodenal stenosis, deep and irreversible morphological alterations predominated in both the mucosal and submucosal layers. Extensive proliferation of fibrotic tissue and its extension into the superficial layers of the muscularis propria provided the morphological basis for severe luminal narrowing. This finding indicates that conservative treatment options are limited in decompensated stenosis.

It should be emphasized that the degree of *Helicobacter pylori* colonization did not show significant differences among the stages of PDS ($p > 0.05$). This suggests that, in the pathogenesis of pyloroduodenal stenosis, long-standing fibrosis and structural remodeling developing against a background of chronic inflammation play a more dominant role than the infectious factor itself.

Overall, the comprehensive analysis of morphometric and morphological parameters has important diagnostic value in determining the stage of pyloroduodenal stenosis and may serve as a basis for selecting optimal therapeutic strategies.

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