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Autoimmune Gastritis: Focus on Endoscopic and Morphological Characteristics

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Aim: tosystematize the literature data on endoscopic semiotics and morphological changes in the gastric mucosa in autoimmune gastritis.
Key points. Autoimmune gastritis is associated with an increased risk of developing adenocarcinoma and neuroendocrine tumours of the stomach. Clarification of diagnostic criteria for autoimmune gastritis is essential for gastroenterological practice. The diagnosis is based on the results of endoscopic and histological examination, and on data from laboratory tests. Isolated atrophy of the mucous membrane of the body of the stomach, the presence of difficult-to-wash creamy mucus, changes in the mucous membrane like "shed skin", and the presence of whitish globule-like foci are typical endoscopic signs of autoimmune gastritis. Widespread pseudopyloric metaplasia, focal intestinal and pseudopancreatic metaplasia, hyperplasia of the ridges of the mucous membrane of the body of the stomach and their relationship to the glandular layer as in the antrum allow during a morphological study considering clinical data to suspect and verify autoimmune gastritis.
Conclusion. During instrumental examination, knowledge of endoscopic symptoms and pathognomonic morphological changes is important for the timely diagnosis of autoimmune gastritis.
Keywords: autoimmune gastritis, endoscopy, biopsy, atrophy, *Helicobacter pylori*
Conflict of interest: the authors declare that there is no conflict of interest.

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Аутоиммунный гастрит: в фокусе эндоскопические и морфологические характеристики

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Цель: систематизировать литературные данные об эндоскопической семиотике и морфологических изменениях слизистой оболочки желудка при аутоиммунном гастрите.
Основные положения. Аутоиммунный гастрит ассоциирован с повышенным риском развития аденокарциномы и нейроэндокринных опухолей желудка. Уточнение диагностических критериев аутоиммунного гастрита имеет существенное значение для гастроэнтерологической практики. Установление диагноза основано на результатах эндоскопического и гистологического исследования, на данных лабораторных тестов. Изолированная атрофия слизистой оболочки тела желудка, наличие трудноотмываемой кремоподобной слизи, изменение слизистой оболочки по типу «сброшенной кожи», наличие белесых глобулоподобных очагов служат типичными эндоскопическими признаками аутоиммунного гастрита. Распространенная псевдопилорическая метаплазия, очаговая кишечная и псевдопанкреатическая метаплазия, гиперплазия валиков слизистой оболочки тела желудка и их соотношение к железистому слою как в антральном отделе позволяют при морфологическом исследовании с учетом клинических данных заподозрить и верифицировать аутоиммунный гастрит.

Заключение. При инструментальном обследовании знание эндоскопических симптомов и патогномоничных морфологических изменений является важным для своевременного диагноза аутоиммунного гастрита.
Ключевые слова: аутоиммунный гастрит, эндоскопия, биопсия, атрофия, *Helicobacter pylori*
Конфликт интересов: авторы заявляют об отсутствии конфликта интересов.

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Atrophic gastritis is a precancerous condition, and its timely diagnosis is crucial for cancer prevention [1–3]. The frequency of *H. pylori*-associated gastritis is decreasing globally, which some researchers attribute to improved hygiene levels and positive outcomes of eradication therapy with antibacterial agents [4, 5]. Considering this trend, the study of autoimmune gastritis (AIG) as an independent significant cause of gastric mucosal atrophy becomes increasingly relevant [6, 7].

AIG is an organ-specific immune-mediated disease characterized by the destruction of gastric parietal cells, leading to the loss of intrinsic factor (Castle factor) and a reduction in gastric acid production. These changes result in iron and vitamin B_{12} malabsorption [8, 9]. The detection of autoantibodies against H^+-, K^+-ATPase and intrinsic factor, reduced serum pepsinogen I, and the ratio of pepsinogen I to pepsinogen II, along with increased gastrin-17, are crucial for AIG diagnosis [4, 9–12]. AIG often coexists with other autoimmune diseases such as autoimmune thyroiditis, less frequently with type 1 diabetes, Addison's disease, vitiligo, and chronic urticaria [9, 11].

The role of *H. pylori* infection in the initiation and progression of the autoimmune process in the stomach is still unclear [8]. For instance, there is speculation that *H. pylori*, through antigenic mimicry, may trigger autoimmune gastritis [13]. Antibodies to parietal cells can be detected in *H. pylori*-positive individuals without AIG [8]. According to one clinical study, up to 62 % of AIG patients had a positive *H. pylori* test result [14].

In a study by E.A. Losik, a high frequency of autoimmune gastritis was found in patients with autoimmune thyroiditis (51.9 %). It was noted that the prevalence of AIG increases with age (27.9 % in patients aged 20–39 years and 53.8 % in patients aged 70–79 years among those surveyed). The frequency of *H. pylori* infection in AIG patients did not differ from population rates, although individuals with pronounced atrophic changes in the gastric body encountered the infectious agent significantly less often [15]. Hematological abnormalities were identified in 25.4 % of patients with high titres of antibodies against parietal cells. Cases of anemia (11.3 %) manifested as both macrocytic and microcytic types.

An increase in the average volume of red blood cells and a low level of vitamin B_{12} were found in 16.9 % of patients, while a decrease in hemoglobin levels was observed in 25 % of cases [10].

Patients with AIG have an increased risk of developing gastric adenocarcinoma and type 1 neuroendocrine tumours of the stomach. During endoscopic observation of patients with pernicious anemia (from 0 to 20 years), the frequency of gastric adenocarcinoma was 3 %, and type 1 neuroendocrine tumours were 4 % [16]. In a cohort study involving 275 patients with atrophic gastritis of the gastric body observed for an average of 5 years (ranging from 1 to 17 years), the annual incidence of gastric cancer/high-grade dysplasia was 0.5 %, low-grade dysplasia — 0.6 %, and type 1 neuroendocrine tumour — 2.8 %. Age over 60 (risk ratio — 4.7), intestinal metaplasia in the absence of pseudopyloric metaplasia (risk ratio — 4.3), and pernicious anemia (risk ratio — 4.3) were associated with a high risk of gastric cancer/high-grade dysplasia and low-grade dysplasia [6]. The potential of AIG as a precancerous condition requires further investigation.

P. Krike et al. did not find a higher prevalence of antibodies against parietal cells and intrinsic factor in patients diagnosed with gastric adenocarcinoma compared to the control group [17]. During long-term follow-up of AIG patients without a history of *H. pylori* infection, no increased risk of gastric cancer was observed compared to the control group, and the authors suggest that this risk may be associated with preceding or unrecognized ongoing *H. pylori* infection [18].

Endoscopic semiotics of autoimmune gastritis

Japanese researchers conducted a retrospective analysis of endoscopic signs of autoimmune gastritis (AIG) in 222 patients and identified key features of AIG, including: predominant localization of mucosal atrophy in the gastric body area (frequency — 96 %); clear visualization of submucosal vessels and flattening/disappearance of longitudinal folds; variable absence of intestinal metaplasia in the mucosa of the antral region [19–21].

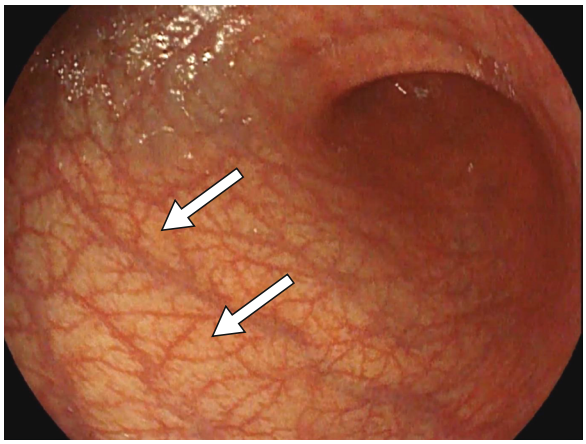

Figure 1. Endophotography during video esophagogastroduodenoscopy in white light: the mucous membrane of the gastric body is diffusely thinned with visible submucosal vessels of various sizes (arrows). (Hereinafter, endophotographies are presented from the archives of the Department of Diagnostic and Treatment Endoscopy of the University Clinical Hospital No. 2 of Sechenov University)

Рисунок 1. Эндофотография при видеоэзофагогастродуоденоскопии в белом свете: слизистая оболочка тела желудка диффузно истончена, с видимыми подслизистыми сосудами различного калибра (стрелки). (Здесь и далее представлены эндофотографии из архива отделения диагностической и лечебной эндоскопии Университетской клинической больницы № 2 Сеченовского университета.)

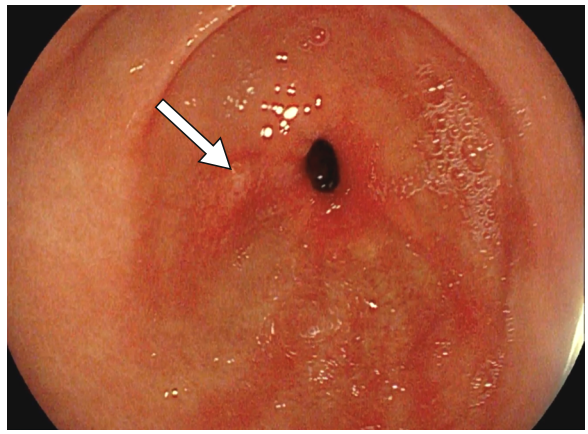

Figure 2. Endophotography during video esophagogastroduodenoscopy in white light (the same patient as in Fig. 1): the mucous membrane of the antrum of the stomach with linear hyperemia, a single epithelizing defect of the mucous membrane under fibrin (arrow)

Рисунок 2. Эндофотография при видеоэзофагогастродуоденоскопии в белом свете (тот же пациент, что и на рис. 1): слизистая оболочка антрального отдела желудка с линейной гиперемией, единичным эпителизирующимся дефектом слизистой оболочки под фибрином (стрелка)

Figure 1 and 2 show endoscopic images of a patient suffered from AIG with atrophy predominantly confined to the gastric body.

When evaluating the data from video esophagogastroduodenoscopy and identifying signs of gastric mucosal atrophy, its stage is determined based on the Kimura — Takemoto classification or the modified EGA (Endoscopic Grading of Atrophy) scale [22–24]. This classification and scale were recommended by the Kyoto Consensus for assessing the prevalence of mucosal atrophy in *H. pylori*-associated gastritis. However, in the case of AIG, considering the absence or minimal atrophic changes in the antral part of the stomach, the accuracy of these scales is limited, according to some researchers [25].

Additionally, five types of changes in the gastric body mucosa, characterized by the nature of visualized remnants of glandular epithelium during endoscopic examination, are described: flat-raised localized (frequency — 48.6 %); pseudopolypoid — raised islands of remaining non-thinned mucosa with pronounced atrophic phenomena at their periphery (22.9 %); island-like (18.6 %); extensively focal (7.1 %); granular (2.9 %) [19, 20].

Figures 3 and 4 show endophotographs demonstrating foci of preserved mucous membrane against the background of atrophy in a patient with AIG.

The mucous membrane of the antral part of the stomach in AIG may be intact or show nonspecific changes such as focal hyperemia, raised erosions, and, in some patients, foci of circular wrinkling of the mucous membrane (in 22 % of AIG cases). Against the background of hypochlorhydria and bacterial contamination, the presence of viscous, difficult-to-wash cream-like mucus with a pale-yellow colour can be visualized (frequency — 32.4 %) [19, 20]. This feature is presented in Figure 5.

When evaluating the pit pattern of the mucous membrane in a narrow spectrum, the phenomenon of the disappearance of centrally located gland orifices with changes in the mucous membrane resembling "shed skin" (frequency — 59 %) is observed [20, 26]. An additional endoscopic marker for the presence of AIG may be the identification of white globule-like foci during routine video endoscopic examination ("white globe appearance"), described as white spherical structures with a diameter of up to 1 mm with microvessels above them, reflecting the location of these structures beneath the gastric epithelium

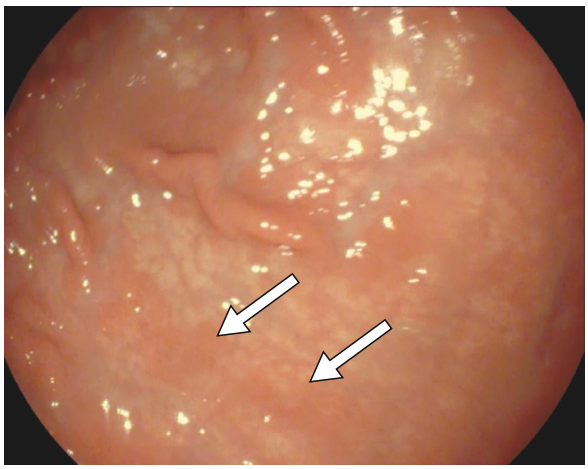

Figure 3. Endophotography during video esophagogastroduodenoscopy in white light: diffusely thinned mucous membrane of the body of the stomach is whitish in colour with islands of intact mucous membrane (arrows)

Рисунок 3. Эндофотография при видеоэзофагогастродуоденоскопии в белом свете: диффузно истонченная слизистая оболочка тела желудка белесого цвета с островками интактной слизистой оболочки (стрелки)

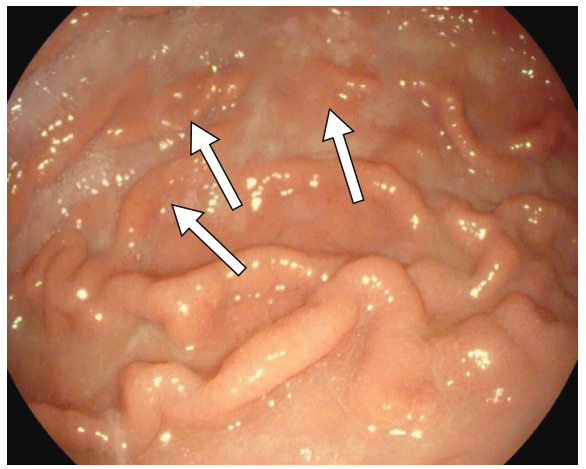

Figure 4. Endophotography during video esophagogastroduodenoscopy in white light: thinned mucous membrane of the body of the stomach is whitish in colour with raised islands of non-thinned mucous membrane of the longitudinal folds of the greater curvature of the body of the stomach (arrows)

Рисунок 4. Эндофотография при видеоэзофагогастродуоденоскопии в белом свете: истонченная слизистая оболочка тела желудка белесого цвета с приподнятыми островками неистонченной слизистой оболочки продольных складок большой кривизны тела желудка (стрелки)

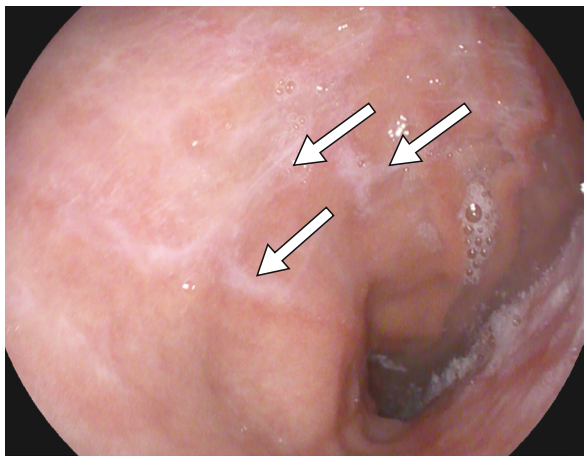

Figure 5. Endophotography during video esophagogastroduodenoscopy in white light: thinned mucous membrane of the body of the stomach with viscous, difficult-to-wash, creamy mucus of a whitish-yellow colour (arrows)

Рисунок 5. Эндофотография при видеоэзофагогастродуоденоскопии в белом свете: истонченная слизистая оболочка тела желудка с вязкой, трудноотмываемой кремоподобной слизью белесо-желтого цвета (стрелки)

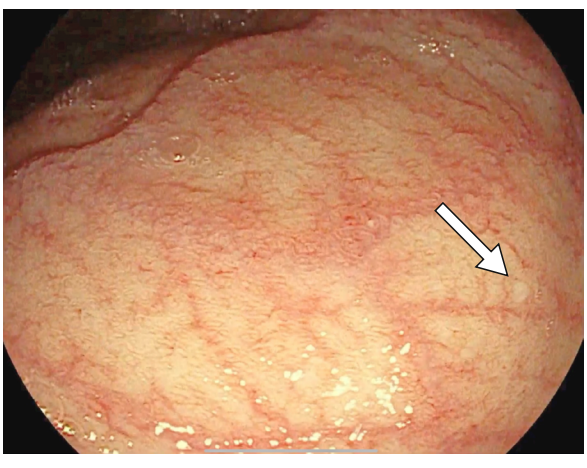

Figure 6. Endophotography during video esophagogastroduodenoscopy in white light: thinned mucous membrane of the gastric body with a whitish globule-like lesion (arrow)

Рисунок 6. Эндофотография при видеоэзофагогастродуоденоскопии в белом свете: истонченная слизистая оболочка тела желудка с белесым глобулоподобным очагом (стрелка)

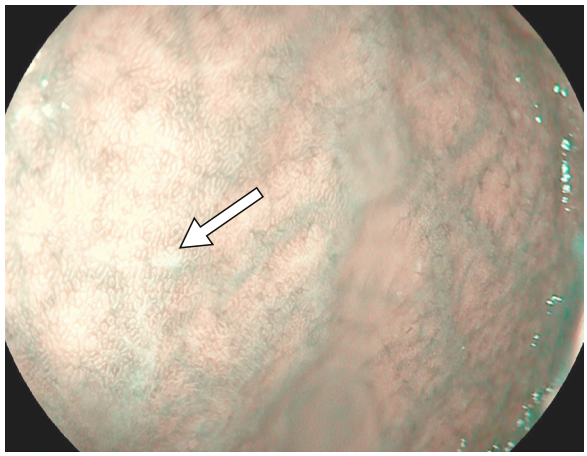

Figure 7. Endophotography during video esophagogastroduodenoscopy in a narrow spectrum of illumination: thinned mucous membrane of the body of the stomach with a whitish globule-like lesion against the background of contouring vessels (arrow)

Рисунок 7. Эндофотография при видеоэзофагогастродуоденоскопии в узком спектре освещения: истонченная слизистая оболочка тела желудка с белесым глобулоподобным очагом на фоне контурирующихся сосудов (стрелка)

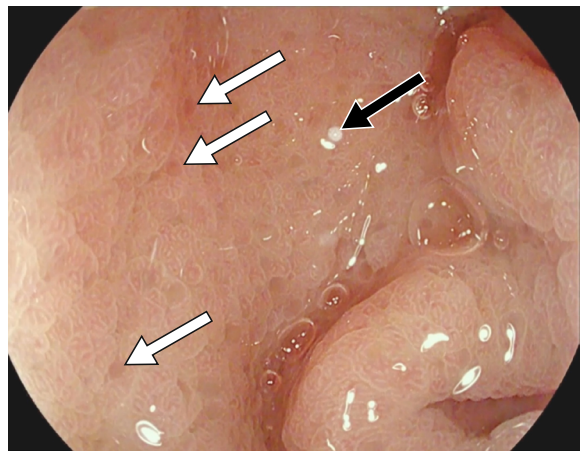

Figure 8. Endophotography during video esophagogastroduodenoscopy in white light: the mucous membrane of the body of the stomach with a whitish globule-like lesion (indicated by the black arrow) and multiple "glomus-like" neoplasms (indicated by the white arrows)

Рисунок 8. Эндофотография при видеоэзофагогастродуоденоскопии в белом свете: слизистая оболочка тела желудка с белесым глобулоподобным очагом (указана черной стрелкой) и множественными «гломус-подобными» новообразованиями (указаны белыми стрелками)

[27]. Figures 6 and 7 show endophotographs of patients with AIG with of white globule-like foci.

These formations are most commonly identified in malignant gastric lesions, but they can also be visualized in AIG, where they have an intermittent nature (may disappear within 12 months of observation) [27]. The appearance of these foci in AIG is explained by the obstruction of glandular ducts due to their destruction in the autoimmune process, leading to the formation of intramucosal cysts [27].

R. Kuvaev et al. described an additional feature of AIG during magnifying endoscopy, characterized by the presence of multiple round pale, but not white, "glomus-like" lesions (GLLs) on the mucous membrane of the gastric body, with branching capillaries on their summits (Fig. 8).

This phenomenon was observed in patients with AIG in 77 % of cases (sensitivity — 0.77; specificity — 1.00) and in 100 % of cases of AIG associated with neuroendocrine neoplasms. Morphological studies have shown that the basis of white "glomus-like" lesions lies in the hyperplasia of enterochromaffin-like cells against the background of atrophic changes [28].

Endoscopic semiotics of early manifestations of AIG has not been fully developed [29]. Literature describes individual cases of endoscopic semiotics of early AIG manifestations in the form of atrophic changes in the mucous membrane, predominantly localized in the area of the lesser curvature of the gastric body, with the presence of reddish nodules resembling pseudopolyps, against the background of atrophic but unchanged mucous membrane along the greater curvature. In the absence of atrophic changes in the early form of AIG, edema and mosaic-like appearance of the mucous membrane of the vault and/or body of the stomach can be observed [30–32].

Patients who have undergone *H. pylori* eradication with formed atrophic changes in the mucous membrane of the antral section pose a particular challenge for the endoscopic diagnosis of AIG. In such situations, the most accurate decision is to perform a gastric mucosa biopsy according to the Sydney protocol and serological blood tests to detect antibodies to parietal cells and intrinsic factor [33].

Histological picture of autoimmune gastritis

For morphological verification of AIG during video esophagogastroduodenoscopy, biopsy of the gastric mucosa is performed according to the modified Sydney protocol, with evaluation according to the OLGA (Operative Link for Gastritis Assessment) scale: two fragments — from the antral section at a distance of 3 cm from the pylorus along the greater and lesser curvature; two fragments — from the body of the stomach in the middle of the greater curvature and at 4 cm proximal to the angle along the lesser curvature; one fragment — from the angle of the

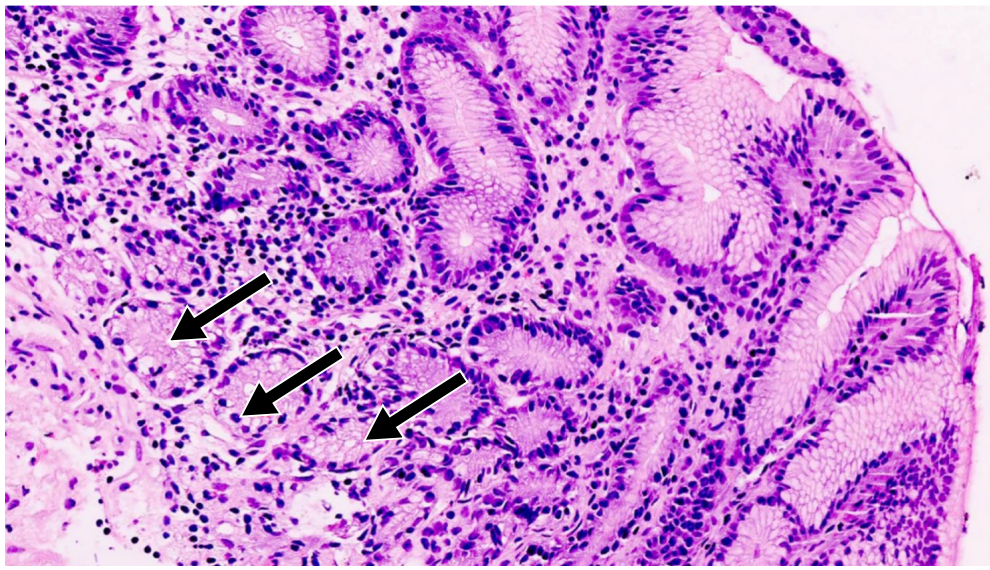

Figure 9. Chronic mild inactive atrophic gastritis of the body of the stomach with widespread pseudopyloric metaplasia (indicated by arrows) and hyperplasia of the foveal layer. Magnification ×200, stained with hematoxylin and eosin. (Microphotographs in this figure and below are from the archives of the Institute of Clinical Morphology and Digital Pathology of Sechenov University)

Рисунок 9. Хронический слабовыраженный неактивный атрофический гастрит тела желудка с распространенной псевдопилорической метаплазией (указаны стрелками) и гиперплазией фовеолярного слоя. Увеличение ×200, окраска гематоксилином и эозином. (Микрофотографии на этом рисунке и далее — из архива Института клинической морфологии и цифровой патологии Сеченовского Университета)

stomach. This biopsy can be combined with material obtained from the antral section of the stomach [4].

In AIG, the inflammatory infiltration consists predominantly of mononuclear cells and is directed against the oxyntic glands with parietal cells. In the case of *H. pylori* infection, inflammatory infiltration is predominantly localized in the superficial layers of the mucous membrane, altering the architecture of the pits. In AIG, the structure of the pits can often remain intact or less damaged, but recognizable by its round or oval forms. Characteristic morphological changes in the mucous membrane in AIG include widespread pseudopyloric metaplasia, focal intestinal metaplasia, and focal pseudopancreatic metaplasia. Morphological examination of biopsies taken from the body of the stomach is practically indistinguishable from biopsies of the mucous membrane of the antral section of the stomach [7, 8]. The typical picture of chronic mild non-active atrophic gastritis of the gastric body with widespread pseudopyloric metaplasia and hyperplasia of the foveolar layer, characteristic of autoimmune gastritis, is presented in Figure 9.

The morphological characteristics of biopsies of the mucous membrane of the antral section (Fig. 10) and the body of the stomach indicate the presence of groups of mucous glands (pyloric in the antral section and pseudopyloric in the body of the stomach) and a mild lymphoplasmacytic infiltrate. In the body of the stomach, there is hypertrophy of the rugae, and the ratio of rugae to the glandular layer is 2:1 (normal range — 1:3–4), approaching that of the antral section. This fact may be misinterpreted as a variant of normalcy and the absence of atrophy if the pathologist is not informed about the biopsy site or is unfamiliar with the morphological diagnostic features of autoimmune gastritis.

Other types of metaplasia (intestinal and pseudopancreatic) have a focal character and, in most cases, do not exceed 30 % of the biopsy area. Widespread pseudopyloric metaplasia is presented in Figure 11.

In some biopsy specimens, preserved areas of the main glands of the stomach body can be observed (Fig. 12), which, against the background of overall atrophy, may manifest as polypoid protrusions of the mucous membrane.

Polyps in the stomach body of patients with autoimmune gastritis (AIG) can also be formed by the hyperplastic foveolar layer (Fig. 13) and appear as elevated areas of rugal folds against the background of atrophic mucous membrane. They may be classified as hyperplastic polyps.

The surface pit epithelium, lining the rugae and pits, is tall and mature cylindrical. Occasionally, pits may expand, forming large cysts filled with

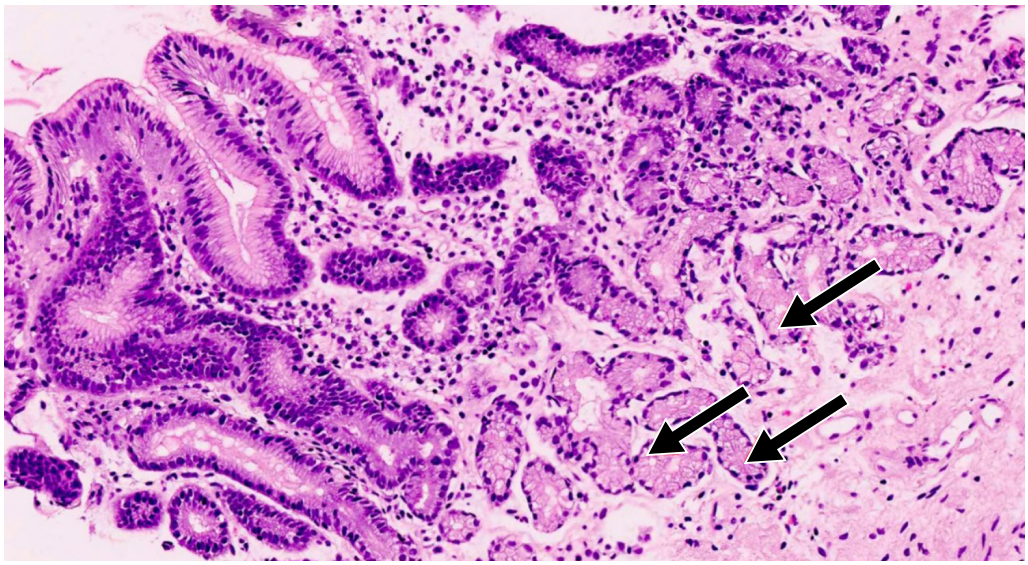

Figure 10. Chronic mild inactive superficial gastritis of the antrum of the stomach (mucous glands are indicated by arrows). Magnification ×200, stained with hematoxylin and eosin

Рисунок 10. Хронический слабовыраженный неактивный поверхностный гастрит антрального отдела желудка (слизистые железы указаны стрелками). Увеличение ×200, окраска гематоксилином и эозином

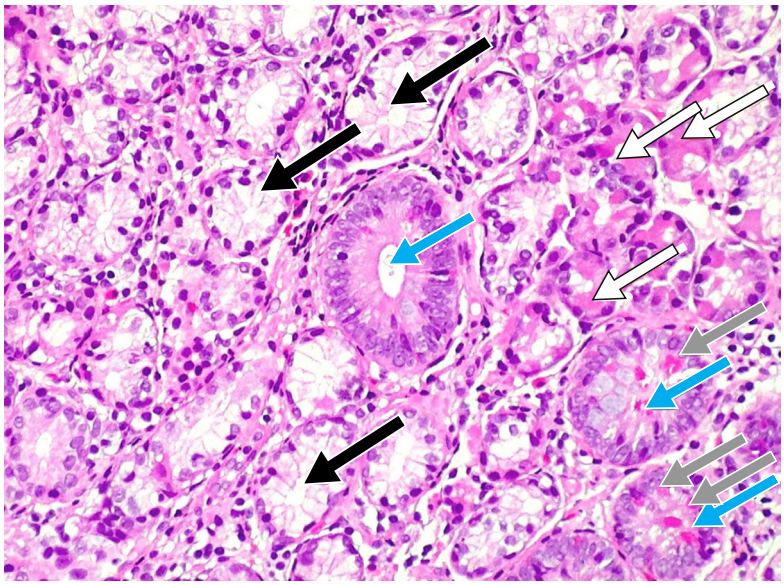

Figure 11. Widespread pseudopyloric metaplasia (black arrows) of the mucous membrane of the gastric body with individual intestinal crypts (blue arrows) with the presence of Paneth cells (grey arrows) (foci of complete intestinal metaplasia) and areas with the presence of eosinophilic staining of the cytoplasm of the glandular epithelium, resembling pancreatic acinar cells (white arrows) (focus of pseudopancreatic metaplasia). Magnification ×250, stained with hematoxylin and eosin

Рисунок 11. Распространенная псевдопилорическая метаплазия (черные стрелки) слизистой оболочки тела желудка с отдельными кишечными криптами (голубые стрелки) с наличием клеток Панета (серые стрелки) (очаги полной кишечной метаплазии) и участками с наличием эозинофильного прокрашивания цитоплазмы эпителия желез, напоминающих ацинарные клетки поджелудочной железы (белые стрелки) (очаг псевдопанкреатической метаплазии). Увеличение ×250, окраска гематоксилином и эозином

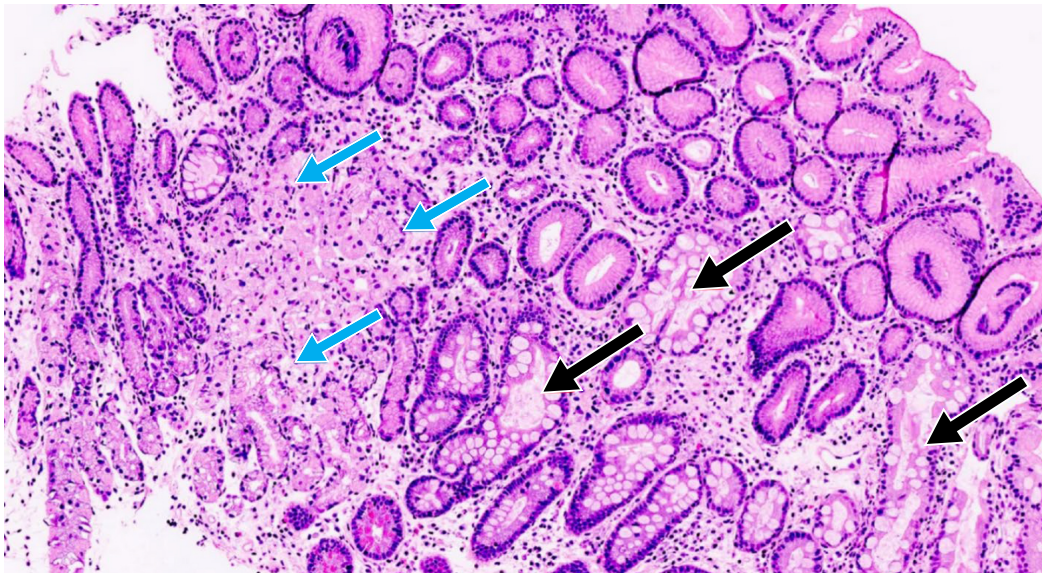

Figure 12. The preserved section of the main glands of the body of the stomach (blue arrows), along the rest of the course — mucous membrane with foci of complete intestinal metaplasia (black arrows) and hyperplasia of the foveal layer. Magnification ×200, stained with hematoxylin and eosin

Рисунок 12. Сохранившийся участок главных желез тела желудка (голубые стрелки), на остальном протяжении — слизистая оболочка с очагами полной кишечной метаплазии (черные стрелки) и гиперплазией фовеолярного слоя. Увеличение ×200, окраска гематоксилином и эозином

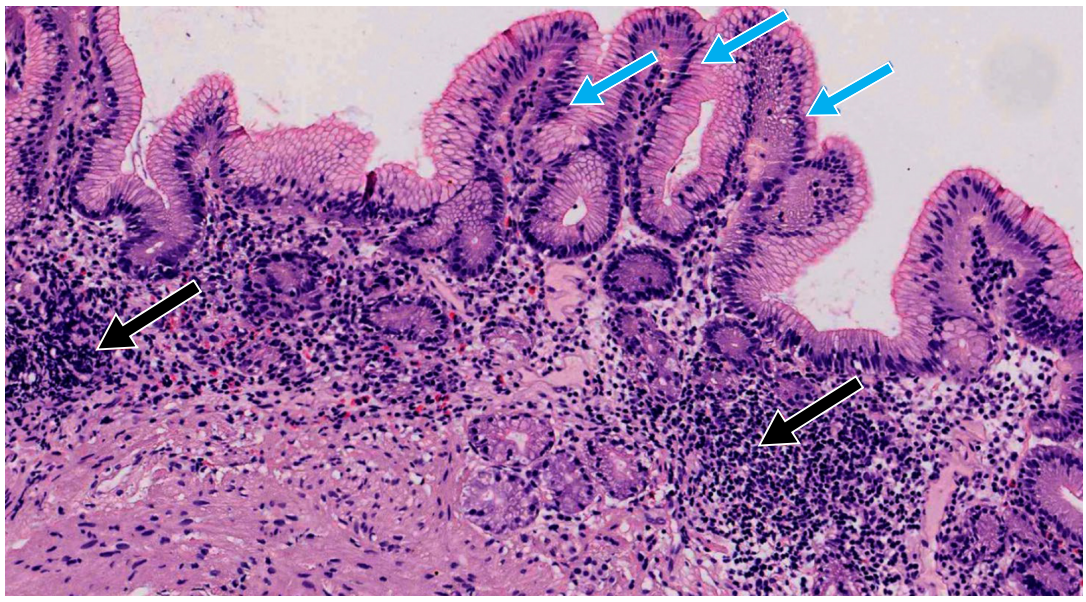

Figure 13. Areas of hyperplastic foveal layer (blue arrows) against the background of pronounced loss of glands and widespread lymphoplasmacytic infiltration with the formation of lymphoid accumulations (black arrows). Magnification ×200, stained with hematoxylin and eosin

Рисунок 13. Участки гиперплазированного фовеолярного слоя (голубые стрелки) на фоне выраженной убыли желез и распространенной лимфоплазмоцитарной инфильтрации с формированием лимфоидных скоплений (черные стрелки). Увеличение ×200, окраска гематоксилином и эозином

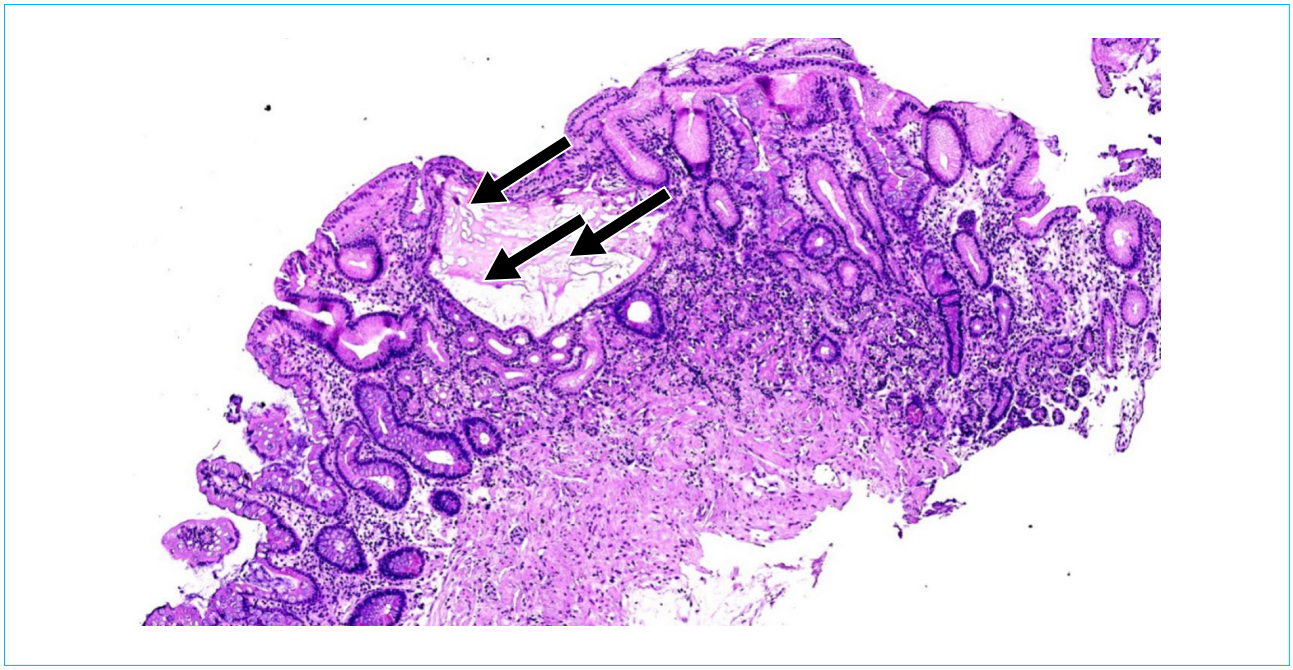

Figure 14. Chronic moderately severe inactive atrophic gastritis of the body of the stomach with focal pseudopyloric and focal complete intestinal metaplasia, hyperplasia of the foveal layer and the presence of a large cyst in the pit area with eosinophilic masses in the lumen (arrows). Magnification ×100, stained with hematoxylin and eosin

Рисунок 14. Хронический умеренно выраженный неактивный атрофический гастрит тела желудка с очаговой псевдопилорической и очаговой полной кишечной метаплазией, гиперплазией фовеолярного слоя и наличием крупной кисты в зоне ямок с эозинофильными массами в просвете (стрелки). Увеличение ×100, окраска гематоксилином и эозином

mucus in the form of eosinophilic masses in the lumen (Fig. 14).

Early morphological signs of AIG have a focal and mild nature, easily detectable in the morphological examination of biopsies. Pseudo-hypertrophic changes in parietal cells and small lymphoid infiltrates in the gastric body mucosa have been described upon detection of antibodies to parietal cells. With disease progression, destructive processes in the chief gland epithelium may occur, characterized by the presence of apoptotic bodies and features of emperipolesis, resulting from the immunological attack of activated T lymphocytes [8].

Conclusion

Endoscopic signs of AIG include isolated atrophy of the gastric body mucosa, the presence of viscous, difficult-to-remove cream-like mucus, changes in the mucosa resembling “shed skin”, and the presence of white globule-like lesions. Modern capabilities of endoscopic equipment and knowledge of the endoscopic AIG semiotics allow a matching of endoscopic and histological signs. Inflammation in AIG is limited to the body and fundus of the stomach, without affecting the antrum, which distinguishes this form of gastritis from other options that lead to atrophy, for example, *H.pylori*-associatd gastritis. This topography of atrophic gastritis during histological and endoscopic examination is the most important sign of the autoimmune nature of inflammation directed against the parietal cells of the oxyntic glands. Histologically, the characteristic features include widespread pseudopyloric metaplasia, focal intestinal metaplasia, focal pseudopancreatic metaplasia, hyperplasia of the rugae in the gastric body mucosa, and their ratio to the glandular layer, approaching that of the antral region. Foveal hyperplasia may endoscopically correspond to pseudopolyps. Intramucosal cysts serve as the morphological substrate of globule-like lesions, hyperplasia of enterochromaffin-like cells - “glomus-like” lesions. Early detection and verification of AIG are essential for determining patient prognosis and the subsequent management plan. It is EGD with histological examination that determines a timely diagnosis.

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