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Spectrum of Chronic Gastritis Based on Morphological Examination of Gastric Biopsies

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Aim: in the retrospective study to assess the frequency of various gastritis variants based on the results of morphological examination of gastric biopsies over a 5-year observation period.
Methods. The study included 3162 individuals who underwent esophagogastroduodenoscopy with biopsy collection between 2017 and 2022. Pathological examination of biopsies was conducted using the updated Sydney system, determining the histological variant and, when possible, the etiology of gastritis. In some cases, chronic gastritis was assessed using the OLGA/OLGIM system.
Results. The most frequently diagnosed type was active *H. pylori*-associated gastritis (36.7 %), followed by chronic atrophic gastritis with intestinal metaplasia in cases where *H. pylori* infection was not detected (28.4 %), including immunohistochemical examination, classified as gastritis after successful *H. pylori* eradication. Atrophic forms of chronic gastritis constituted 34.8 %. Minimal or mild changes close to normal histological characteristics were observed in 19.2 % of patients. Reactive gastropathy was present in 7.6 % of cases. Autoimmune gastritis ranked fourth in frequency, with a relatively high percentage of observations (8.6 %).
Conclusion. According to the analysis of gastric biopsies in the Russian population, a high frequency of *H. pylori*-associated gastritis was observed, and autoimmune gastritis is not uncommon. The high prevalence of atrophic gastritis emphasizes the importance of dynamic patient monitoring within cancer prevention programs.
Keywords: gastritis, *H. pylori,* reactive gastropathy, atrophic gastritis, autoimmune gastritis
Conflict of interest: the authors declare that there is no conflict of interest.

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Спектр хронических гастритов по результатам морфологического исследования гастробиоптатов

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Цель: в ретроспективном исследовании оценить частоту различных вариантов гастрита на основании результатов морфологического исследования биоптатов желудка за пятилетний период наблюдения.
Методы. В исследование были включены 3162 человека, которым в период с 2017 по 2022 г. проведена эзофагогастродуоденоскопия со взятием биопсий. Патолого-анатомическое исследование биоптатов проводили на основе обновленной Сиднейской системы с определением гистологического варианта и, по возможности, этиологии гастрита. В части случаев была проведена оценка хронического гастрита по системе OLGA/OLGIM.
Результаты. Чаще всего был диагностирован активный *H. pylori*-ассоциированный гастрит — 36,7 %; в 28,4 % случаев установлен хронический атрофический гастрит с кишечной метаплазией, при котором инфекция *H. pylori* не была обнаружена, в том числе с помощью иммуногистохимического исследования. Эти случаи были расценены как постэрадикационный гастрит. Процент атрофических форм хронического гастрита составил 34,8 %. У 19,2 % пациентов изменения в биоптатах носили минимальный или слабовыраженный характер и были близки к нормальным гистологическим характеристикам слизистой оболочки. Реактивная гастропатия встречалась в 7,6 % случаев. Аутоиммунный гастрит занимает четвертое место по частоте встречаемости с достаточно высоким процентом наблюдений (8,6 %).

Выводы. В российской популяции, согласно анализу гастробиоптатов, наблюдается высокая частота *H. pylori*-ассоциированного гастрита, нередким заболеванием является аутоиммунный гастрит. Высокая частота атрофического гастрита обуславливает важность динамического наблюдения за пациентами в рамках программы канцеропревенции.
Ключевые слова: гастрит, *H. pylori,* реактивная гастропатия, атрофический гастрит, аутоиммунный гастрит
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Introduction

Helicobacter pylori infection is considered the most common cause of chronic inflammation of the gastric mucosa, leading to the recognition of chronic gastritis as an infectious disease [1]. The prevalence of *H. pylori* demonstrates significant geographical variability, mainly dependent on the socio-economic status of the population [2, 3]. Regions with high infection rates include Africa (70.1 %; 95 % CI: 62.6–77.6 %), South America (69.4 %; 95 % CI: 63.9–74.9 %), and Asia (66.6 %; 95 % CI: 56.1–77.0 %), while Western Europe (34.3 %; 95 % CI: 31.3–37.2 %) and North America (37.1 %; 95 % CI: 32.3–41.9 %) have lower infection rates [2]. The decreasing prevalence of *H. pylori* infection is considered a significant global trend [3, 4].

A systematic review indicates a global decrease in *H. pylori* infection from 58.2 % (95 % CI: 50.7–65.8 %) in the 1980–90s to 43.1 % (95 % CI: 40.3–45.9 %) in the period 2011–2022 [4]. In Russia, a decrease in *H. pylori* frequency has also been observed, with the lowest prevalence in individuals under 18 years old (20.2 %) and the highest in the 41–50 years old age group (43.9 %) [5].

The Kyoto Global Consensus on *Helicobacter* gastritis recommends classifying gastritis based on etiological factors, emphasizing the need for reflection in the International Classification of Diseases of the 11th revision [1]. The prevalence and morbidity of other etiological variants of gastritis (autoimmune, biliary, alcoholic, Crohn's disease-related, etc.) are understudied in our country and worldwide. Forty years after the discovery of *H. pylori* as the main cause of gastritis, considering the temporal trends of *H. pylori* infection in the population, it is important to assess the frequency of various gastritis variants.

Aim of the research

In this retrospective study, we aimed to assess the frequency of various gastritis variants based on the results of morphological examination of gastric biopsies over a 5-year observation period.

Materials and methods

Cases of chronic gastritis were selected from the database of the Central Pathoanatomical Department (CPAD) of the Clinical Center of Sechenov University based on the analysis of gastric biopsies from patients who underwent esophagogastroduodenoscopy (EGD) from January 2017 to May 2022. The upper gastrointestinal tract was examined according to the standardized EGD protocol. At least two biopsies were taken from the antral region and two from the body of the stomach along the lesser and greater curvature based on the updated Sydney system [6]. Routine biopsy of the gastric angle was not performed in all cases, as its impact on the final diagnosis is considered ambiguous [7, 8]. Histological findings were recorded in the standardized reporting system of the CPAD of the Clinical Center of Sechenov University, developed on the basis of the 1C Accounting program.

Biopsies were fixed in 10 % neutral buffered formalin, embedded in paraffin, sectioned at a thickness of 3–4 um on 4–5 levels, and stained with hematoxylin and eosin. *H. pylori* infection was assessed using Giemsa staining, and in doubtful cases, immunohistochemistry (IHC) was performed with polyclonal antibodies to *H. pylori* (Dako, Denmark) on the Bond Max automated immunostainer (Leica Biosystems, USA).

Inflammatory infiltrate, variations in its density, activity, degree of *H. pylori* colonization, and the extent of intestinal metaplasia were evaluated using visual analog scales presented in the updated Sydney system [6]. Diagnostic criteria for reactive gastropathy included foveolar hyperplasia, edema and hypertrophy of smooth muscle fibers in the lamina propria, vasodilatation, and hyperemia of the mucosal capillaries, along with significant inflammatory infiltrate [9]. Diagnosis of autoimmune gastritis was based on chief cell damage with atrophy and intestinal metaplasia affecting the body of the stomach. Pseudopyloric metaplasia, characterized by the replacement of chief cells with mucous glands, and pseudopancreatic metaplasia

with the appearance of cells resembling acinar cells of the pancreatic gland were frequently observed. In contrast to the body of the stomach, the mucosa of the antral region in autoimmune gastritis could appear normal or show minor inflammatory changes [6]. In cases with a previously established diagnosis of Crohn's disease, gastric involvement was diagnosed against the background of focal active gastritis with or without epithelioid granulomas, aphthous lesions, and/or fissured erosions/ulcers [10].

Results

A total of 3162 patients were included in the study, comprising 1688 (53.4 %) females and 1474 (46.6 %) males, with a female-to-male ratio of 1.15 : 1. The average age was 53 years (mean — 52.3 years, range — 19–86 years). Patients with a diagnosis of gastric cancer were excluded from the study. From 2017 to 2022, the established diagnosis of "chronic gastritis" ranged from 329 cases in the first half of 2022 to 670 cases in 2018 (Table).

A diagnosis of *H. pylori*-associated gastritis was confirmed in 1029 (36.68 %) out of 3162 individuals (Figs. 1–4). *Helicobacter pylori* gastritis was characterized by neutrophilic infiltration in 100 % of cases, with inflammation predominating in the antral part of the stomach in 74 % of patients, in the body — in 8.5 %, and evenly distributed in all stomach regions in 17.5 %.

Chronic atrophic gastritis with intestinal metaplasia was identified in 899 (28.4 %) individuals (Fig. 5), where *H. pylori* infection could not be detected, even with IHC examination. For these patients, a history of *H. pylori* eradication therapy could be assumed, although such information was lacking in the histological examination referral. Therefore, this group of patients was tentatively classified as post-eradication gastritis. Biopsy collection in these cases was explained by the endoscopic monitoring protocol for patients with atrophic gastritis, following the Clinical Recommendations of the Russian Gastroenterological Association and the RENDO Endoscopic Society for the diagnosis and treatment of gastritis [11].

A diagnosis of autoimmune gastritis was established in 272 (8.6 %) patients (Figs. 6, 7). Reactive gastropathy (chemical gastritis) was identified in 241 (7.6 %) (Fig. 8), characterized by foveolar hyperplasia, edema and hypertrophy of smooth muscle fibers in the lamina propria, vasodilation, hyperemia of mucosal capillaries, and minimal inflammatory infiltrate. Autoimmune gastritis often coexisted with reactive gastropathy in the antral part (in 35.3 % of 272 patients)

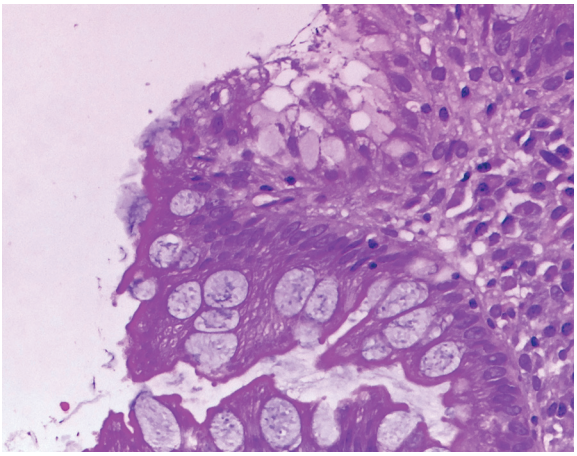

Figure 1. A small area of preserved integumentary pitted epithelium with signs of damage and the presence of microbial contamination on the surface of epithelial cells; above and below the located foci of intestinal metaplasia with goblet cells do not contain bacteria (×400, hematoxylin and eosin staining)

Рисунок 1. Мелкий участок сохранившегося покровно-ямочного эпителия с признаками повреждения и наличием микробной обсемененности на поверхности эпителиоцитов; выше и ниже расположенные очаги кишечной метаплазии с бокаловидными клетками не содержат бактерий (×400, окраска гематоксилином и эозином)

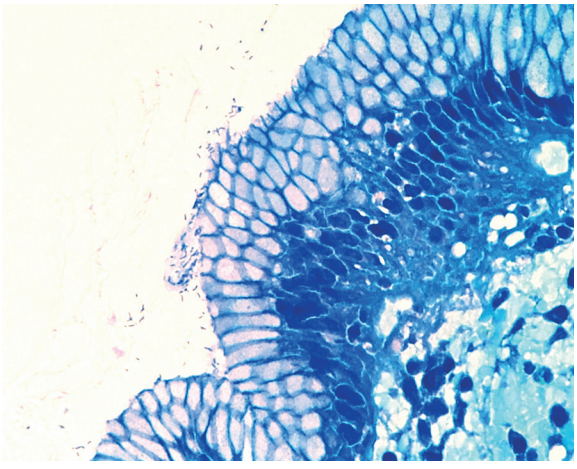

Figure 2. Clusters of positively stained bacteria on the surface of the biopsy specimen (×400, Giemsa stain)

Рисунок 2. Скопления позитивно окрашенных бактерий на поверхности биоптата (×400, окраска по Гимзе)

and rarely with *H. pylori*-associated gastritis (in 3.3 % of 272 patients).

Changes in biopsies for 608 (19.2 %) individuals exhibited minimal or mild characteristics,

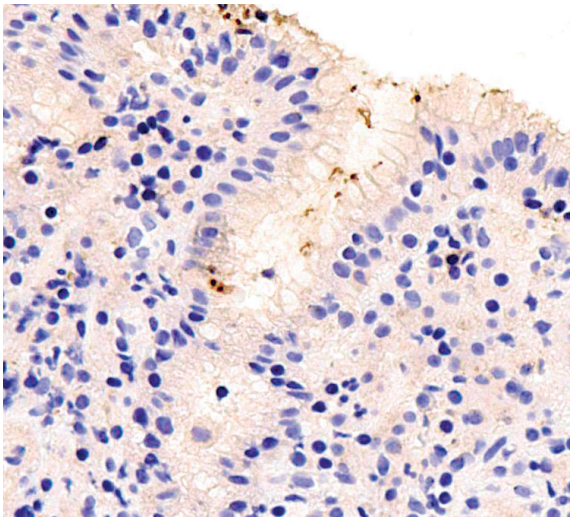

Figure 3. Positively stained brown bacteria, some of which on a cross section look like pinpoint inclusions in the covering mucus on the surface of the biopsy specimen and in the lumen of the pits (×400, IHC reaction with an antibody to *H. pylori*)

Рисунок 3. Позитивно окрашенные в коричневый цвет бактерии, часть из которых на поперечном срезе имеет вид точечных включений в покрывающей слизи на поверхности биоптата и в просвет ямок (×400, ИГХ-реакция с антителом к *H. pylori*)

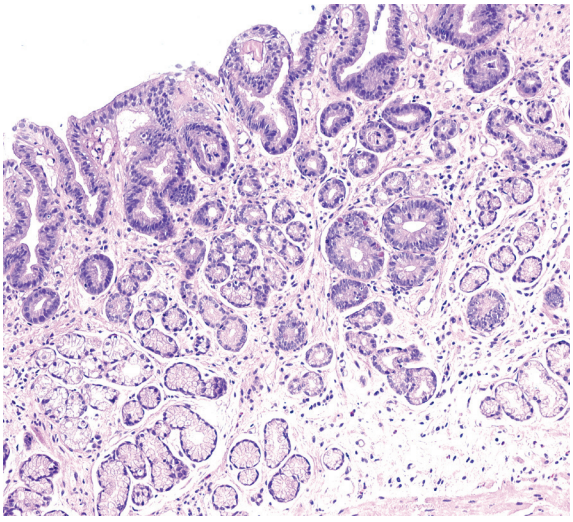

Figure 5. Chronic mild inactive atrophic gastritis of the antrum with focal complete intestinal metaplasia — up to 30 % of the biopsy area (×200, hematoxylin and eosin staining)

Рисунок 5. Хронический слабовыраженный неактивный атрофический гастрит антрального отдела желудка с очаговой полной кишечной метаплазией — до 30 % площади биоптата (×200, окраска гематоксилином и эозином)

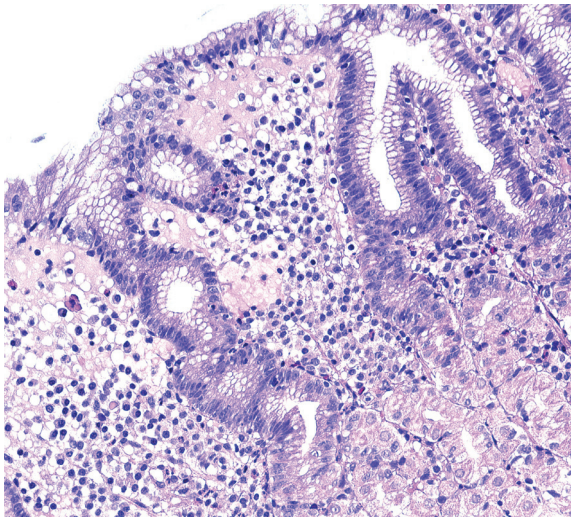

Figure 4. Chronic moderately active superficial *Helicobacter* gastritis of the body of the stomach (×200, hematoxylin and eosin staining)

Рисунок 4. Хронический умеренно выраженный активный поверхностный хеликобактерный гастрит тела желудка (×200, окраска гематоксилином и эозином)

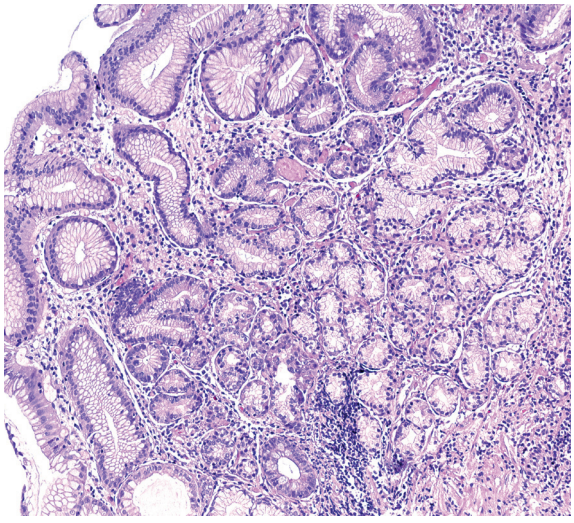

Figure 6. Autoimmune gastritis; chronic moderately severe inactive atrophic gastritis of the gastric body with widespread pseudopyloric metaplasia, focal complete intestinal metaplasia, hyperplasia of the foveal layer and the formation of lymphoid accumulations (×200, hematoxylin and eosin staining)

Рисунок 6. Аутоиммунный гастрит; хронический умеренно выраженный неактивный атрофический гастрит тела желудка с распространенной псевдопилорической метаплазией, очаговой полной кишечной метаплазией, гиперплазией фовеолярного слоя и формированием лимфоидных скоплений (×200, окраска гематоксилином и эозином)

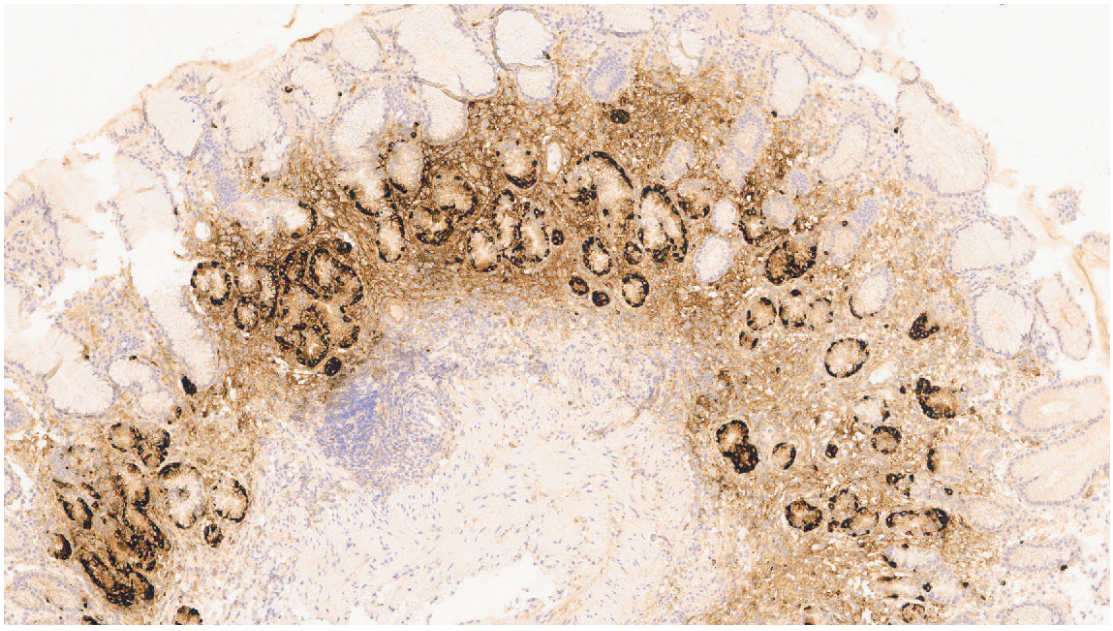

Figure 7. Autoimmune gastritis; widespread hyperplasia of neuroendocrine cells in the glands of the body of the stomach (×100, IHC reaction with antibody to Chromogranin A)

Рисунок 7. Аутоиммунный гастрит; распространенная гиперплазия нейроэндокринных клеток в железах тела желудка (×100, ИГХ-реакция с антителом к Хромогранину А)

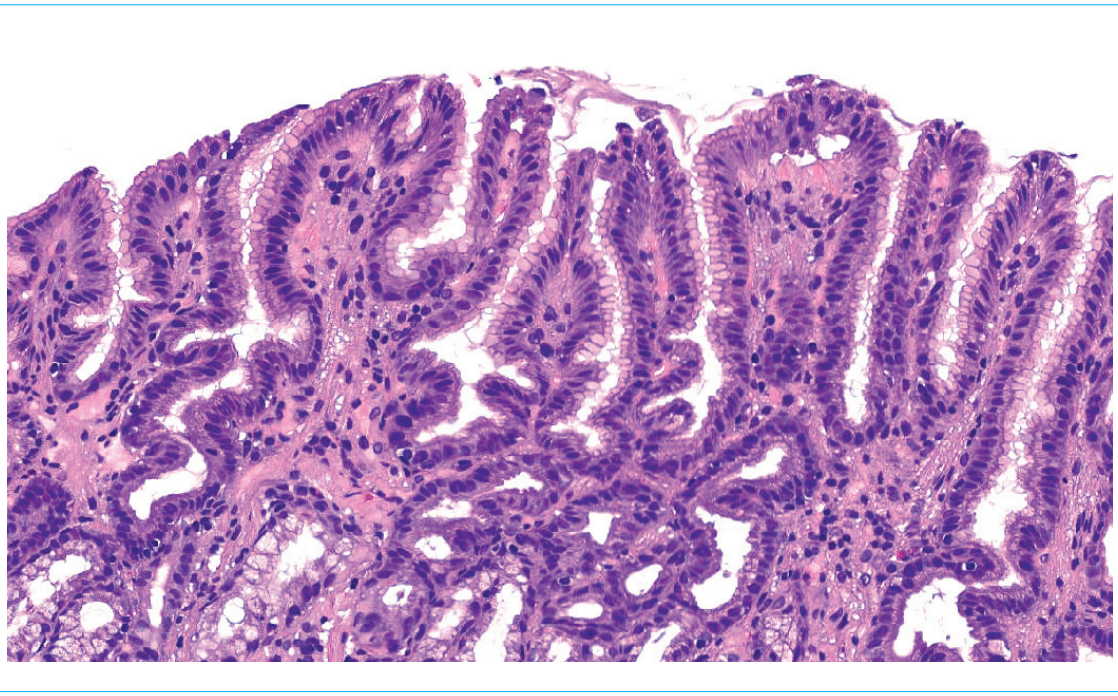

Figure 8. Reactive/chemical gastritis/gastropathy; biopsy of the mucous membrane of the antrum of the stomach with pronounced hyperplasia of the foveal layer, convoluted deep pits, proliferation of smooth muscle cells and paretic plethora of capillaries, the lamina propria contains a few lymphocytes (×200, hematoxylin and eosin staining)

Рисунок 8. Реактивный/химический гастрит/гастропатия; биоптат слизистой оболочки антрального отдела желудка с выраженной гиперплазией фовеолярного слоя, извитыми глубокими ямками, пролиферацией гладкомышечных клеток и паретическим полнокровием капилляров, собственная пластинка содержит немногочисленные лимфоциты (×200, окраска гематоксилином и эозином)

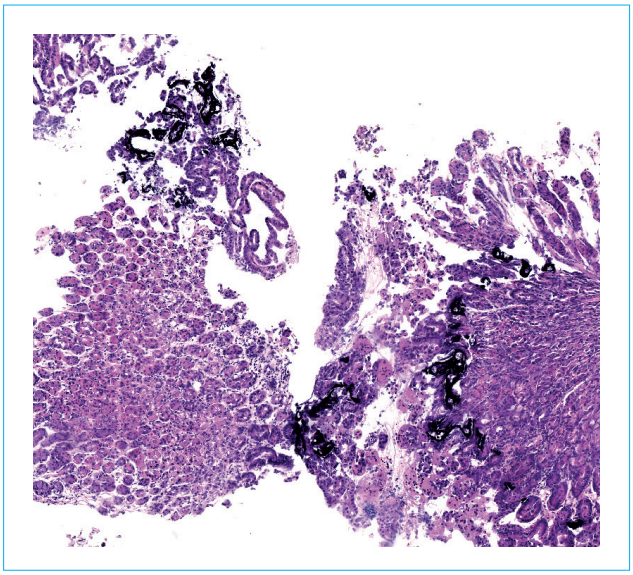

Figure 9. Mineralization of the mucous membrane of the gastric body; biopsy samples of the mucous membrane of the body of the stomach with damage and desquamation of the integumentary epithelium and deposition of basophilic mineral masses in the area of the ridges (×60, stained with hematoxylin and eosin)

Рисунок 9. Минерализация слизистой оболочки тела желудка; биоптаты слизистой оболочки тела желудка с повреждением и десквамацией покровного эпителия и отложением базофильных минеральных масс в зоне валиков (×60, окраска гематоксилином и эозином)

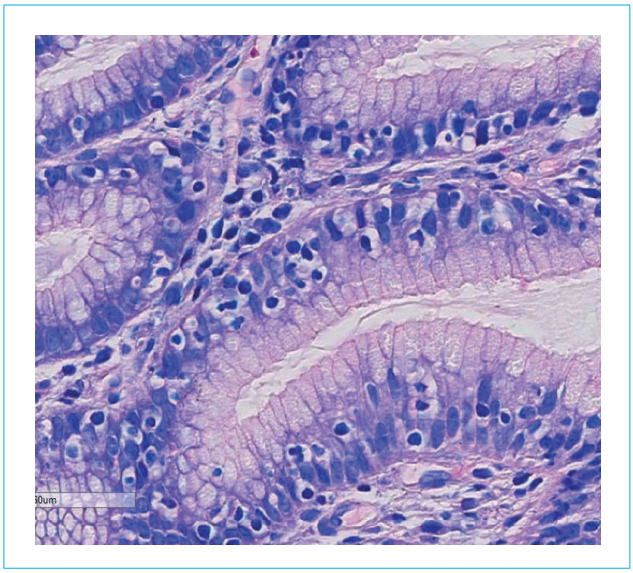

Figure 10. Lymphocytic gastritis; a large number of interepithelial lymphocytes in the integumentary pitted epithelium — lymphocytes have a clearing zone surrounding each lymphocyte located among the epithelial cells (×400, Giemsa staining)

Рисунок 10. Лимфоцитарный гастрит; большое количество межэпителиальных лимфоцитов в покровно-ямочном эпителии — лимфоциты имеют зону просветления, окружающую каждый лимфоцит, расположенный среди клеток эпителия (×400, окраска по Гимзе)

closely resembling normal histological features of the mucosal lining (Table).

This analysis included data from 273 patients with gastric and duodenal ulcer disease. In addition to biopsies from the ulcer margins, biopsies were performed according to the Sydney protocol. A significant portion of these patients had *Helicobacter pylori* gastritis — 78 (28.6 %) out of 273 people, while a small portion had reactive gastropathy — 15 (5.5 %). Consequently, based on the characteristics of gastric biopsies, an obvious cause of ulcer formation could not be identified in the remaining patients.

Other types of gastritis accounted for a small percentage of cases — 113 (3.6 %) out of 3162 individuals (Figs. 9–14).

Mineralization of the mucosal lining (calcification) was detected in 44 patients (1.4 %) (Fig. 9). According to literature data, gastric calcinosis may occur in the presence of hypercalcemia or hyperphosphatemia, typically associated with renal insufficiency or neoplastic conditions (metastatic form of gastric calcinosis). Inflammatory or fibrotic tissue changes with normal calcium and phosphorus levels in serum may also lead to calcinosis (distrophic form of gastric calcinosis). The cause of calcinosis is often challenging to

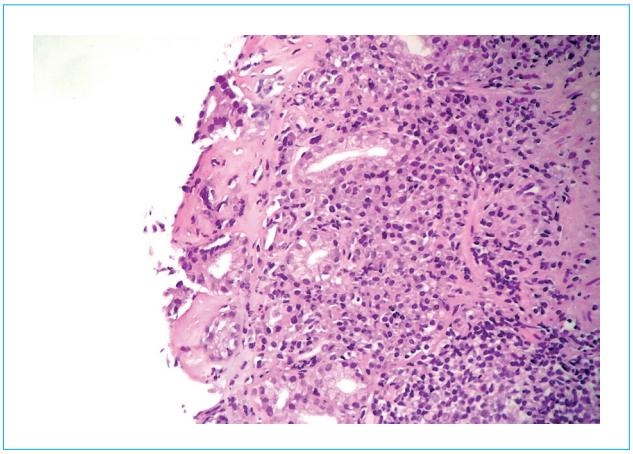

Figure 11. Collagenous gastritis; deposition of homogeneous eosinophilic masses in the subepithelial sections; the integumentary epithelium is desquamated over a grea-ter extent; lamina propria with pronounced lymphoplasmacytic infiltration with the formation of lymphoid follicles (×200, hematoxylin and eosin staining)

Рисунок 11. Коллагеновый гастрит; отложение гомогенных эозинофильных масс в субэпителиальных отделах; покровный эпителий на большем протяжении десквамирован; собственная пластинка с выраженной лимфоплазмоцитарной инфильтрацией с формированием лимфоидных фолликулов (×200, окраска гематоксилином и эозином)

Table. Distribution of cases of chronic gastritis by year
Таблица. Распределение случаев хронического гастрита по годам

Form of chronic gastritis *Форма хронического гастрита*	Year / *Год*						Total *Всего*
	2017	2018	2019	2020	2021	2022	
H. pylori-associated gastritis *H. pylori-ассоциированный (хеликобактерный) гастрит*	159	229	195	155	183	108	1029
Multifocal atrophic gastritis after successful *H. pylori* eradication *Постэрадикационный мультифокальный атрофический гастрит*	101	201	174	126	199	98	899
Chemical gastritis (reactive gastropathy) *Химический гастрит (реактивная гастропатия)*	67	67	51	30	18	8	241
Autoimmune gastritis / *Аутоиммунный гастрит*	32	42	49	50	57	41	272
Minimal changes in the mucous membrane *Минимально выраженные изменения слизистой оболочки*	66	98	97	138	141	68	608
Mineralization of the mucous membrane *Минерализация слизистой оболочки*	10	16	7	5	6	0	44
Lymphocytic gastritis / *Лимфоцитарный гастрит*	6	9	9	8	5	2	39
Collagenous gastritis *Коллагеновый гастрит*	0	0	0	0	1	1	2
Eosinophilic gastritis *Эозинофильный гастрит*	0	1	0	0	0	0	1
Changes in the mucous membrane in inflammatory bowel diseases *Изменения слизистой оболочки при воспалительных заболеваниях кишечника*	5	7	4	7	2	3	28

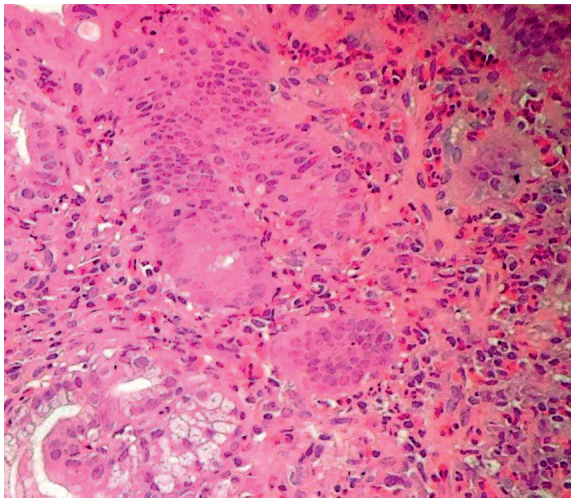

Figure 12. Eosinophilic gastritis; cellular infiltrate of the lamina propria with pronounced diffuse eosinophilic infiltration with a predominance of eosinophils in the infiltrate (×200, hematoxylin and eosin staining)

Рисунок 12. Эозинофильный гастрит; клеточный инфильтрат собственной пластинки слизистой с выраженной диффузной эозинофильной инфильтрацией с преобладанием эозинофилов в инфильтрате (×200, окраска гематоксилином и эозином)

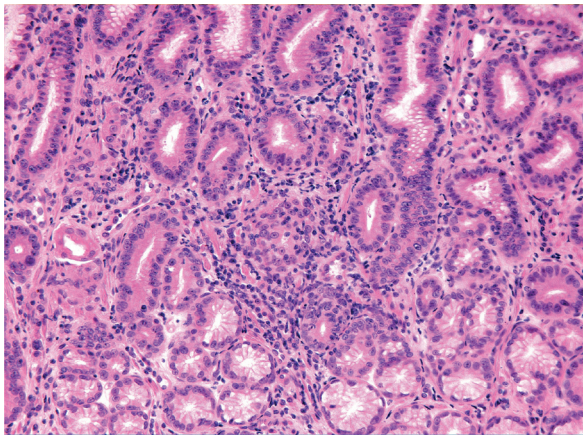

Figure 13. Focal/focal active gastritis; focal inflammatory cellular infiltrate, located predominantly in the projection of the cervical glands with the presence of segmented leukocytes in it, penetrating and damaging the epithelium (×200, hematoxylin and eosin staining)

Рисунок 13. Очаговый/фокальный активный гастрит; очаговый воспалительный клеточный инфильтрат, расположенный преимущественно в проекции шеечного отдела желез с наличием в нем сегментоядерных лейкоцитов, проникающих и повреждающих эпителий (×200, окраска гематоксилином и эозином)

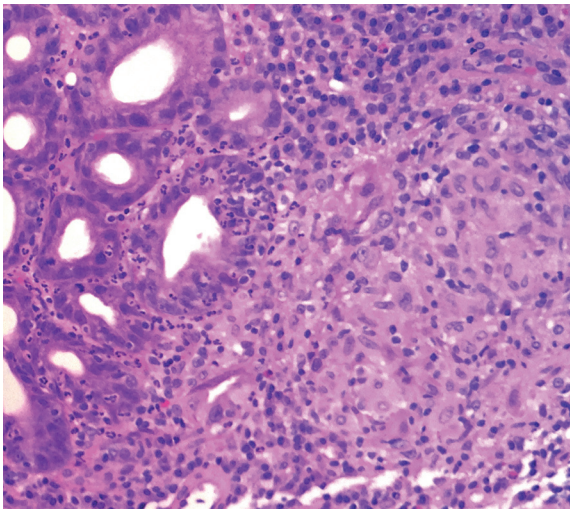

Figure 14. Granulomatous gastritis; epithelioid cell granuloma, without signs of necrosis; in the surrounding pits — accumulations of leukocytes penetrating the epithelium and a thick lymphoplasmacytic infiltrate (×200, hematoxylin and eosin staining)

Рисунок 14. Гранулематозный гастрит; эпителиоидноклеточная гранулема, без признаков некроза; в окружающих ямках — скопления лейкоцитов, проникающих в эпителий и густой лимфоплазмоцитарный инфильтрат (×200, окраска гематоксилином и эозином)

establish [12, 13]. Due to limited information available in the database, the reasons for mineralization in our patient group remain unknown. In our opinion, these cases may be classified as reactive gastropathies, as some instances were associated with the use of medications.

Lymphocytic gastritis was detected in 39 (1.3 %) patients. It was characterized by an increased content of interepithelial lymphocytes in the integumentary pitted epithelium (Fig. 10).

Rare variants of gastritis constituted a small percentage of observations and confirmed their rarity. The diagnosis of collagenous gastritis was established in two patients (0.06 % over a period of more than 5 years of observation) (Fig. 11), eosinophilic gastritis — in one patient (Fig. 12), and Menetrier disease in one patient.

Separately, a group with gastritis among patients with inflammatory bowel diseases should be highlighted — 28 (0.88 %) patients with Crohn's disease (Figs. 13, 14).

Intestinal metaplasia was identified in 1344 (43 %) out of 3126 patients. It was present in all cases (100 %) of multifocal atrophic gastritis and frequently in individuals with autoimmune gastritis (79.4 %), less commonly — in cases of atrophic *H. pylori* gastritis (19.5 %), and reactive gastropathy (11.6 %). It is noteworthy that in autoimmune gastritis, intestinal metaplasia was more often observed in the body of the stomach (in 70 % — in the body, in 9.4 % — in the antral part) compared to all other types of gastritis, where intestinal metaplasia was predominantly found in the antral region.

The implementation of the prognostic systems OLGA (Operative Link for Gastritis Assessment) and OLGIM occurred gradually. In 2017, biopsies were taken according to the protocol, and histological assessments were made using OLGA in 10.4 % of observations. By 2022, this increased to 69.6 %. The distribution by stages was as follows: Stage 0 accounted for 26.8 % of cases, Stage I — for 37.2 %, Stage II — 29.7 %, Stage III — 5.5 %, and Stage IV — 1.4 %. Thus, Stages 0–II demonstrated approximately equal percentages of observations, while Stages III–IV, associated with a high risk of gastric cancer and requiring subsequent endoscopic surveillance, comprised a total of around 7 % of cases (Fig. 15).

Discussion

The most prevalent cases in the examination of gastric biopsies were those of *H. pylori*-associated gastritis (36.68 %). The prevalence of Helicobacter gastritis in the Russian population reflects the trend of decreasing *H. pylori* infection rates in our country but remains higher than in European countries [15] and is comparable to Asian countries such as China, Japan, and South Korea [16].

Helicobacter gastritis has distinctive morphology, allowing for an etiological diagnosis through hematoxylin and eosin staining even before detecting the bacteria. Additional Giemsa staining, which we utilized, only confirmed the initially diagnosed *H. pylori*-associated gastritis. In cases where the histological examination was indicated based on a positive result of the rapid urease test during endoscopy, we were able to detect *H. pylori* in almost 100 % of cases. In doubtful cases, we preferred to resort to immunohistochemistry for detection. In certain situations (widespread intestinal metaplasia, proton pump inhibitor use), bacterial translocation to the body of the stomach can occur. In such cases, detecting *H. pylori* may be possible when examining biopsies taken from the body of the stomach, emphasizing the importance of adhering to the Sydney protocol [17].

Reactive gastropathy in the Russian population was identified in 7.6 % of cases. Our data diverge from European studies where reactive gastropathy was more common than active *Helicobacter*

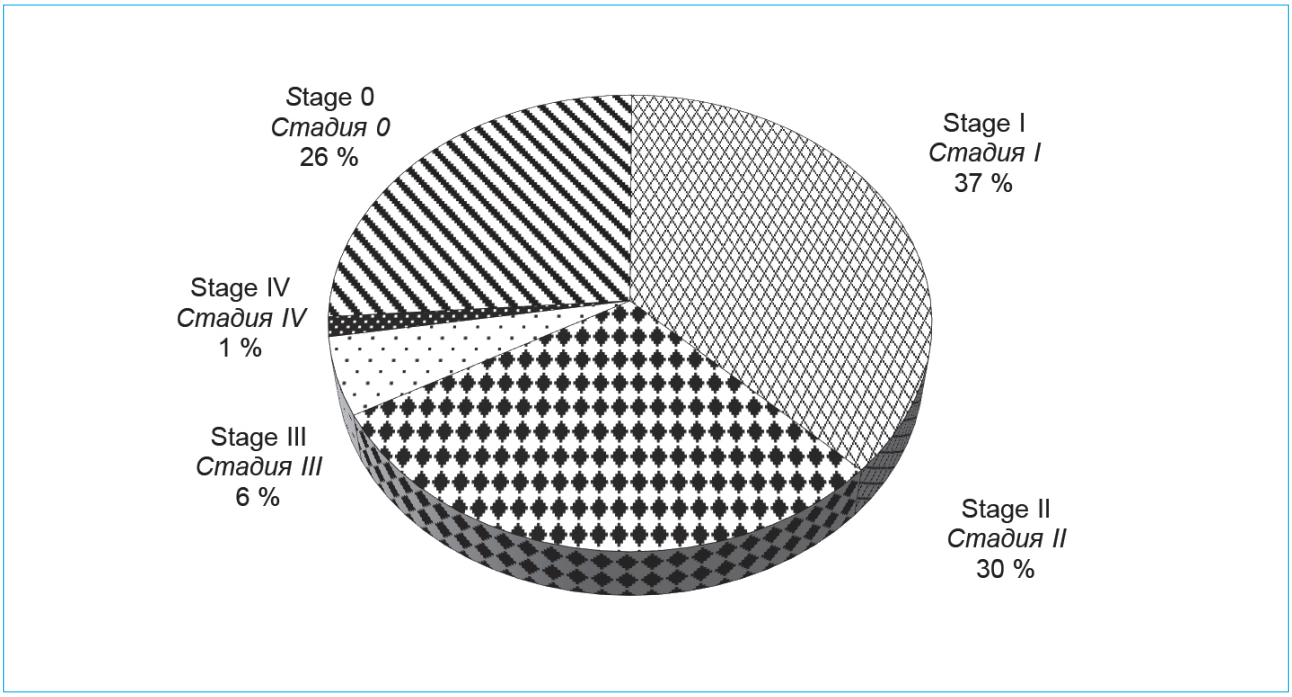

Figure 15. Distribution of cases of chronic gastritis according to the OLGA/OLGIM system

Рисунок 15. Распределение случаев хронического гастрита по системе OLGA/OLGIM

gastritis and post-eradication gastritis [15, 18]. A retrospective study in the United States, analyzing a large database, demonstrated that reactive gastropathy became the most common type of gastritis [19]. In the International Classification of Diseases, 11th edition (ICD-11), variants of reactive gastropathy are distinguished based on etiological factors, with biliary reflux and nonsteroidal anti-inflammatory drug use being the main contributors. However, the cause of chemical gastritis remains unrecognized in some cases. According to I. Maguilnik et al., reactive gastropathy is associated with Barrett's esophagus (odds ratio (OR) — 1.21; 95 % CI: 1.16–1.29), duodenitis (OR = 1.36; 95 % CI: 1.28–1.44), intraepithelial lymphocytosis of the duodenum (OR = 1.25; 95 % CI: 1.13–1.39), active ileitis (OR = 1.88; 95 % CI: 1.47–2.40), localized active colitis (OR = 1.57; 95 % CI: 1.33–1.86), and collagenous colitis (OR = 1.50; 95 % CI: 1.12–2.03) [19].

The second most common finding in our study was multifocal atrophic gastritis with intestinal metaplasia, where we could not detect *H. pylori* even with IHC staining. In the vast majority of cases, gastritis was inactive and interpreted as post-eradication gastritis by us. The widespread use of *H. pylori* eradication therapy in clinical practice will lead to an increase in the number of post-eradication gastritis cases, which, in the presence of a significant stage of atrophy, requires endoscopic surveillance for early detection of dysplasia [1, 11]. In this regard, it is important to universally include information about *H. pylori* eradication in the referral for histological examination. In a small portion of cases, signs of inflammatory activity were identified, suggesting another cause of inflammation, possibly not yet identified.

Atrophic gastritis with intestinal metaplasia is a well-recognized risk factor for the development of gastric cancer [1, 11, 20]. The assessment of biopsies according to the OLGA/OLGIM system was conducted in 70 % of cases. We identified 6.9 % of patients with Stage III and IV according to OLGA, belonging to the high-risk group for cancer development. Intestinal metaplasia is characterized by a change in the phenotype of gastric epithelium to that of the small or large intestine, with goblet cells. In our study, we diagnosed intestinal metaplasia in 43 % of patients with chronic gastritis; this high percentage may be partially explained by the participation of patients in cancer prevention programs and precise biopsy sampling. In our study, intestinal metaplasia predominated in the antral part in *H. pylori*-associated and post-eradication gastritis, and in the body of the stomach in autoimmune gastritis.

The high frequency of autoimmune gastritis in our study (8.6 %, fourth in frequency) may be due to an increase in disease incidence on the one hand and, on the other hand, the repeated admission of patients with a previously established diagnosis for dynamic observation. In our study, autoimmune gastritis was extremely rare in combination with *Helicobacter* gastritis.

Assessing the combinations of different histological variants of gastritis was challenging. Reactive gastropathy (in the antral part) was most often found in combination with autoimmune gastritis. The morphological criteria for diagnosing different types of gastritis occurring simultaneously in the same patient have not been extensively developed. As early as 2001, M. Stolte et al. raised the question of whether there is a mixed "chemically induced/reactive and *Helicobacter* gastritis" [21]. T.S. Chen et al. observed more pronounced foveolar hyperplasia in reactive gastropathy in combination with more pronounced inflammatory infiltrate when coexisting with *H. pylori* infection [22].

The etiological principle should become key in establishing the diagnosis of chronic gastritis. Our experience shows that gastritis is broader than the issue of *H. pylori* infection. Establishing a diverse spectrum of gastric mucosa changes, driven by various etiological factors, has become possible thanks to the interaction of a multidisciplinary team of specialists equipped with advanced endoscopic technology, patient concentration, and selection in university clinical hospitals.

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