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H. pylori-Associated Gastritis, Gastritis after *H. pylori* Eradication and *H. pylori*-Negative Gastritis: Algorithm of Diagnosis and Treatment (Literature Review and Resolution of the Expert Panel of the Russian Gastroenterological Association)

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Aim: to provide the basic points of the Expert Panel of the Russian Gastroenterological Association with discussion of measures to improve diagnostics, treatment and management of chronic gastritis patients.
Key points. In the Russian Federation in 2021 more than 30 million patients diagnosed with "Gastritis and duodenitis" were recorded (K29 in accordance with International Classification of Diseases-10). *H. pylori* incidence rate in the Russian population has reduced and presently is about 40 %. In chronic gastritis pattern *H. pylori*-associated gastritis has still dominated though gastritis percent after successful *H. pylori* eradication, reactive gastropathy (including reflux gastritis) and autoimmune gastritis, has increased. Endoscopic and histologic examinations serve as key diagnostic techniques that provide a means for assessing the ethiology of gastritis, topography and degree of atrophic changes, gastritis staging as per OLGA/OLGIM system that properly correlates with the risk of stomach cancer and determines endoscopic examination strategy. *H. pylori* eradication therapy of gastritis serves as an etio-

tropic treatment and makes it possible to prevent progression of atrophy and stomach cancer. Conventional triple therapy combined with bismuth tripotassium dicitrate allows for achieving optimal cure rates of *H. pylori* eradication. Addition of rebamipide to regimens of *H. pylori* eradication improves their efficiency. Rebamipide arrests symptoms of dyspepsia in the case of chronic gastritis and functional dyspepsia. The administration of rebamipide for chronic gastritis makes it possible to influence the syndrome of increased epithelial permeability and inflammation, which makes it advisable to study it as a means of preventing stomach cancer and the progression of atrophy in various types of chronic gastritis.

Conclusion. Members of the Expert Panel has approved the algorithm of diagnosis and treatment of *H. pylori*-associated gastritis, gastritis after *H. pylori* eradication and *H. pylori*-negative gastritis at the diagnostic stage in the case of initial presentation and long-term follow-up when needed.

Keywords: chronic gastritis, *H. pylori*, atrophic gastritis, autoimmune gastritis, reflux gastritis, higher epithelial permeability syndrome, dyspepsia, prevention of stomach cancer, rebamipide.

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H. pylori-ассоциированный, постэрадикационный и негеликобактерный гастриты: алгоритм диагностики и лечения (обзор литературы и резолюция Совета экспертов Российской гастроэнтерологической ассоциации)

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Цель: представить основные положения совета экспертов Российской гастроэнтерологической ассоциации с обсуждением мер по оптимизации диагностики, лечения и ведения пациентов с хроническим гастритом.
Основные положения. В Российской Федерации в 2021 г. зарегистрировано более 30 млн пациентов с диагнозом «Гастрит и дуоденит» (K29 в соответствии с МКБ-10). Частота инфекции *H. pylori* в российской популяции снижается и в настоящее время составляет около 40 %. В структуре хронического гастрита по-прежнему преобладает *H. pylori*-ассоциированный гастрит, однако увеличивается доля гастрита после успешной эрадикации *H. pylori*, реактивной гастропатии, включая рефлюкс-гастрит, и аутоиммунного гастрита. Ключевыми диагностическим методами служат эндоскопическое и гистологическое исследования, которые позволяют оценить этиологию гастрита, топографию и степень атрофических изменений, стадию OLGA/OLGIM, которая соотносится с риском рака желудка и определяет стратегию эндоскопического наблюдения. Эрадикационная терапия *H. pylori* при гастрите служит этиологическим лечением, позволяет предотвратить прогрессирование атрофии и рак желудка. Стандартная тройная терапия в сочетании с висмута трикалия дицитратом позволяет достичь оптимальных показателей эрадикации *H. pylori*. Добавление ребамипида к режимам для эрадикации *H. pylori* улучшает их эффективность. Симптомы диспепсии при хроническом гастрите и функциональной диспепсии уменьшаются при лечении ребамипидом. Назначение ребамипида при хроническом гастрите позволяет воздействовать на синдром повышенной эпителиальной проницаемости и воспаление, что делает целесообразным дальнейшее изучение его эффективности как средства профилактики рака желудка и прогрессирования атрофии при различных вариантах хронического гастрита.
Заключение. Участники совета экспертов утвердили алгоритм диагностики и лечения *H. pylori*-ассоциированного, постэрадикационного и негеликобактерного гастритов на этапе диагностики при первичном обращении и при необходимости длительного наблюдения.
Ключевые слова: хронический гастрит, *H. pylori*, атрофический гастрит, аутоиммунный гастрит, рефлюкс-гастрит, синдром повышенной эпителиальной проницаемости, диспепсия, профилактика рака желудка, ребамипид
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On March 28, 2024, under the chairmanship of the President of the Russian Gastroenterological Association (RGA), Academician of the Russian Academy of Sciences V.T. Ivashkin, the RGA Expert Council was held, which considered issues of optimizing the diagnosis and treatment of chronic gastritis and improving patient management tactics. The relevance of the problem of chronic gastritis is determined by the high incidence of the disease: in 2021 3,090,050 adult patients diagnosed with "gastritis and duodenitis" were recorded in the Russian Federation (2,668.7 : 100,000 of adult population) [1]. This statistical data shows the importance of "gastritis and duodenitis" diagnosis (K29 in accordance with ICD-10) in clinical practice and reflect the real frequency of the disease in the population. *H. pylori* infection always causes gastritis irrespective of clinical evidence available and clear cases of stomach diseases [2], while *H. pylori* prevalence in Russia is still high (38.8 %) in spite of downward trend [3]. The importance of gastritis as a disease associated with the risk of developing stomach cancer determines the importance of timely assessment of the prognosis and implementation of preventive measures [2, 4, 5].

Aim: to provide the basic points of the Expert Panel of the Russian Gastroenterological Association with discussion of measures to improve diagnostics, treatment and management of patients suffering from chronic gastritis.

Syndrome of increased epithelial permeability in chronic gastritis pathogenesis

In recent years the Russian experts have developed and implemented the syndrome of increased epithelial permeability (SIEP) paradigm into practical healthcare [6–8]. Mechanisms of integrated three-level system of epithelial barrier protection including pre-epithelial, epithelial and subepithelial protection have been characterized as applied to the gastrointestinal tract [9]. SIEP at epithelial level includes disorder of transcellular (dysfunction of

ion channels) and paracellular permeability (firstly, disturbance of tight junctions functioning) [10, 11]. Considering the key role of *H. pylori* in the genesis of chronic gastritis, the mechanisms of interaction of this microorganism with epithelial defense systems are of interest. It has been shown that *H. pylori* virulence factors CagA provide the adhesion of the bacterium to the mucosa and by directly interacting with junctional proteins of tight junctions disrupt their functioning [12].

The relevance of studying SIEP is also associated with its participation in the processes of metaplasia and carcinogenesis observed in gastritis [13, 14]. The key role in this is played by disturbances in the claudin link of cytoprotection [15]. The indicated pathophysiological mechanisms of SIEP are reflected in the Clinical Guidelines of the Russian Gastroenterological Association (RGA) devoted to gastritis and duodenitis [4, 5].

Determination of the role of SIEP in the genesis of chronic gastritis has served as the basis for the development of "epithelium protective therapy" concept for this disease [7]. Epithelium protective effects have been described for a series of drugs — proton pump inhibitors such as rabeprazole and pantoprazole, colloidal bismuth medical products, and some probiotics.

The largest evidence base is available for rebamipide. It has been shown that rebamipide in chronic gastritis helps to normalize of functioning and interaction of all three levels of protection f gastrointestinal barrier [15]. A number of recent studies have presented a detailed clinical and pharmacological analysis of the effect of this drug in chronic gastritis. Meta-analyses have shown that inclusion of rebamipide into *H. pylori* eradication therapy regimens increases it effectiveness [16]. This drug can help reduce inflammation and atrophic changes. There is even a possibility of reducing the severity of metaplasia in antrum pyloricum if rebamipide is prescribed, though this statement requires additional confirmation [17]. Proven cancer-preventive effects of this drug are of significant practical importance [18]. In addition, rebamipide helps to reduce the symptoms of dyspepsia in the case of combination of gastritis and functional dyspepsia [19].

Gastritis and dyspepsia

The survey results show that in half of the cases, in the presence of dyspepsia symptoms, general practitioners make a preliminary diagnosis of gastritis [20]. In this case, the diagnosis must be morphologically verified and, as a rule, is not accompanied by symptoms. In most cases, indicated symptoms serve as a manifestation of functional dyspepsia. Before establishing a reliable diagnosis of functional dyspepsia, it is necessary to exclude gastritis associated with *H. pylori*. Russian and international recommendations give clear provisions on the need for eradication therapy for *H. pylori* in patients with undiagnosed dyspepsia in order to eliminate the symptoms of dyspepsia and establish a diagnosis of functional dyspepsia [2, 4, 5]. *H. pylori* eradication helps relieve symptoms of functional dyspepsia, but this effect is very modest (number of patients to be treated is 14; 95 % confidence interval (95 % CI): 11–21) [21]. Etiopathogenesis of functional dyspepsia is considered a multifaceted process with complex cause-and-effect relationships. The occurrence of dyspepsia symptoms is caused by many factors, including dietary characteristics, changes in the motility of stomach and duodenum, duodenal eosinophilia, disturbance in the "brain — gastrointestinal tract" axis [22]. Among the "bacterial agents" associated with functional dyspepsia, not only *H. pylori*, but also changes in the microbiota and small intestinal bacterial overgrowth (SIBO) are mentioned. According to a meta-analysis of seven studies involving 1,248 patients, the pool incidence of SIBO in patients with functional dyspepsia was 34.73 % (95 % CI: 24.807–45.383 %), although with significant heterogenicity of results ($p < 0.0001$; $I^2 = 89.91$ %) [23].

The diagnosis of functional dyspepsia sometimes "hides" functional disorders of the gallbladder and sphincter of Oddi or reflux gastritis (biliary gastritis) [24, 25]. The significance of duodenal reflux and reflux gastritis in the occurrence of dyspeptic symptoms or the development of functional dyspepsia requires further study. It is assumed that changes in the qualitative and quantitative composition of the microflora of the stomach and small intestine under the effect of motility disorders of the lower part of gastrointestinal tract, changes in the composition and exposure time of bile components in the cavity of the small intestine and stomach, the subsequent development of SIEP underlie the motor and sensory disorders of the stomach characteristic of functional dyspepsia [26, 27]. The frequent association of functional dyspepsia with reflux gastritis provides a basis for studying the prospects of using ursodeoxycholic acid preparations in these diseases especially considering its beneficial effect on the microbiome [26, 28, 29].

Optimization of *H. pylori* infection eradication therapy

Eradication therapy of *H. pylori* infection plays a key role in the management of patients with chronic Helicobacter gastritis. Target values of antimicrobial therapy imply eradication of the pathogen in more than 90 % of treated patients [2]. There are two possible strategies for choosing a regimen for *H. pylori* eradication therapy: the individualized one — based on determining the susceptibility of *H. pylori* to antibacterial drugs, and empirical strategy — based on information on local resistance of *H. pylori* to clarithromycin and monitoring the effectiveness of regimens in the region. In the Russian Federation, the empirical approach prevails, although the efforts of the medical community are focused on the wider

introduction into practice of molecular genetic methods for determining resistance, which demonstrate high sensitivity and specificity [30].

Triple therapy with clarithromycin and amoxicillin based on the empirical choice remains one of the most commonly prescribed in Russia (56 %), despite its low effectiveness — 80 % [31]. One of the methods for increasing the effectiveness of eradication therapy regimens specified in the Clinical recommendations of RGA is the addition of bismuth tripotassium dicitrate to bismuth-free regimens [4, 5]. According to data of the European Registry on Helicobacter pylori management (Hp-EuReg), triple therapy with clarithromycin boosted with bismuth tripotassium dicitrate results in *H. pylori* eradication in 88 % of cases in the "intention-to-treat" cohort and in 94 % of cases in the "per-protocol" cohort, and with 14-day period of prescription — in 93 % of cases ("intention-to-treat" cohort) [32]. Thus, bismuth tripotassium dicitrate as the fourth component of eradication regimens ensures overcoming of *H. pylori* resistance to clarithromycin.

The addition of rebamipide as a measure to improve eradication efficacy is included in the RGA Clinical Guidelines based on three meta-analyses [4, 5, 16, 33, 34]. Meta-analysis made by D.N. Andreev et al. (2022) analyzed the studies completed in the Russian Federation with the use of Rebagit (manufactured by PRO.MED.CS). In 6 controlled studies (n = 531) the summarized efficiency of eradication was 90.376 % (95 % CI: 86.311–93.560 %) in patients receiving rebamipide, and 81.681 % (95 % CI: 76.499–86.141 %) — in patients who did not receive it. The addition of rebamipide to *H. pylori* eradication regimens significantly increases the effectiveness of treatment (OR = 2.162; 95 % CI: 1.268–3.685; p = 0.005) [16]. Evidence is accumulating that the inclusion of rebamipide into eradication regimens as the fifth component (standard triple therapy + bismuth tripotassium dicitrate + rebamipide) significantly increases the effectiveness of therapy, bringing it closer to rates of more than 95 % [35].

Morphological diagnostics of chronic gastritis

The term "gastritis" defines a spectrum of conditions characterized by histologically confirmed inflammation of the gastric mucosa. Atrophy is defined as a condition characterized by a decrease in density or loss of glands of the corresponding parts of the gastric mucosa and their replacement by extracellular matrix (non-metaplastic atrophy) and/or another type of glands (metaplastic atrophy) [36]. To diagnose and stage gastritis, at least two biopsies from antrum and two — from the body of the stomach are required; biopsies are also taken from any endoscopically altered areas of the mucosa. Biopsy from the angle of the stomach, as recommended by the updated Sidney system, is not mandatory [37]. The histological description should include the topography and extent of atrophic changes, OLGA/OLGIM stages, which correlates well with the risk of stomach cancer and determines the strategy of endoscopic surveillance [37]. It should be noted that when using high-resolution endoscopes with narrow-band imaging mode, it is possible to stage atrophy and intestinal metaplasia using the Kimura — Takemoto, EGA and EGGIM classification systems, which are characterized by a high degree of correlation with OLGA/OLGIM [37].

To make the diagnosis of *H. pylori* more objective, it is recommended to additionally conduct a corresponding immunohistochemical study, which allows avoiding false-positive results with special stains and excluding the presence of other microflora. Immunohistochemical reaction with chromogranin A confirms the presence of hyperplasia of neuroendocrine cells in the body of the stomach, which is an important diagnostic sign of autoimmune gastritis.

The etiological principle should be the key one when establishing the diagnosis of "chronic gastritis". When analyzing data from the Centralized Anatomic Pathology Department of Sechenov University, gastrobiopsies of 3162 patients were studied, who underwent esophagogastroduodenoscopy with biopsy taking in the period from 2017 to 2022. Most often, active *H. pylori*-associated gastritis was diagnosed — 36.7 %; in 28.4 % of cases, chronic atrophic gastritis with intestinal metaplasia was established, in which *H. pylori* infection was not detected, including by immunohistochemical examination. These cases were assessed as post-eradication gastritis. The percentage of atrophic forms of chronic gastritis was 34.8 %. It is possible that such a high figure is associated with programs for monitoring patients with OLGA stages III–IV, which are carried out in the University clinics. In 19.2 % of patients changes in biopsy specimens were minimal or mild and were close to normal histological characteristics of the mucous membrane. Reactive gastropathy occurred in 7.6 % of cases. Autoimmune gastritis took fourth place in terms of frequency of occurrence with a fairly high percentage of observations (8.6 %), which, on the one hand, may be due to an increase in morbidity, and, on the other hand, due to the "concentration" of patients with this diagnosis for dynamic observation in the University clinics [38]. Rare variants of gastritis — lymphocytic gastritis (1.3 %) and gastritis combined with Grohn's disease — accounted for a small percentage of cases, while collagenous and eosinophilic gastritis and Menetrier's disease were observed in isolated patients [38].

Thus, *H. pylori*-associated gastritis still predominates in the Russian population. Studies conducted in recent years in the Russian Federation have demonstrated a decrease in the proportion of the infected individuals in the population: according to epidemiological studies in 2004–2014, 65–92 % of adults were infected [4, 5], in 2017–2019 — about 40 % [3], in Moscow in 2022–2024 — 37.19 % [39].

With the decrease in the prevalence of *H. pylori* infection [3] and the increase in the number of people with eradication of the infection, the structure of chronic gastritis is changing with a significant proportion of post-eradication gastritis. Reactive gastropathy and autoimmune gastritis are common diseases [38]. The high incidence of atrophic gastritis determines the importance of identifying patients at high risk of gastric cancer (stage III–IV according to OLGA) and their endoscopic dynamic monitoring within the framework of the cancer prevention program.

Endoscopic diagnostics of chronic gastritis

Endoscopic examination of a patient to establish the diagnosis of chronic gastritis should be performed using additional imaging technologies and accompanied by a biopsy of the mucous membrane if there is a suspicion of atrophic, metaplastic and neoplastic changes. Erythema and erosions of the gastric mucosa may be a manifestation of both an inflammatory process (gastritis) and reactive changes in the epithelium with minimal inflammatory infiltration (gastropathy) [37, 40]. The most characteristic endoscopic signs of *H. pylori*-associated inflammatory process of the mucous membrane are: diffuse erythema, spotty redness, diffuse edema, enlarged and tortuous folds of the mucous membrane of the stomach body, expansion of the gastric fields, deepening of the grooves, and whitish coating [41, 42]. In *H. pylori*-associated gastritis, visualization of punctate or tortuous whitish pits in the body of the stomach under conditions of narrow-band magnifying endoscopy is possible [43]. *H. pylori*-negative status is reliably characterized by visualization of typical collector venules in the lower third of the body of the stomach along the lesser curvature, as well as the presence of polyps of the fundic glands [44]. In the absence of *H. pylori* infection, erythema bands and hemorrhagic petechiae of the mucous membrane are also often found, which is a frequent manifestation of the reflux nature of the lesion due to the reflux of duodenal contents.

The causes of gastropathy are drugs (primarily NSAIDs and acetylsalicylic acid) [45], alcohol radiation, mechanical factors, and bile in duodenogastric reflux. However, it is difficult to determine the cause of gastropathy during endoscopic examination, so anamnestic data analysis is necessary [46]. In particular, it is difficult to reliably verify pathological duodenogastric reflux, since the presence of bile in the stomach is observed both in normal conditions (in early morning hours, during prolonged fasting) and in pathological conditions (after surgeries, at all stages of the development of gallstone disease, with functional disorders of the upper gastrointestinal tract) [25, 47].

Atrophy of the mucosa is characterized by pallor, decrease height of mucosal folds, and clearer visualization of submucosal vessels [48]. In the case of *H. pylori*-associated atrophic gastritis, the most pronounced atrophic changes are observed in the distal part of the stomach, have a multifocal distribution [49, 50], and often create a visible border with non-atrophic mucosa ("atrophy line") [51]. In contrast, autoimmune atrophic gastritis is characterized by diffuse lesions of the mucous membrane of the proximal stomach ("reverse atrophy" phenomenon) [52]. A sign of autoimmune gastritis when examined in a narrow-band mode with magnification is the absence of the orifices of the glands of the body of the stomach with preserved regular capillaries (the "cast-off skin" sign). Point "glomus-like" lesions associated with hyperplasia of ECL cells are often encountered, as well as the changes in the "white globe appearance" type, which are an accumulation of detritus in cystically dilated glands [53–55].

Intestinal metaplasia may appear endoscopically as either elevated whitish loci (ash-colored nodular change) [56] or mottled patchy erythema [57]. When examined in narrow-band imaging modes, areas of intestinal metaplasia have a uniform appearance of light blue foci on a brown background due to presence of the phenomenon of "light blue crests" [58] on the surface of the epithelium and the accumulation of "white opaque substance" [59] within epithelial structures with an intestinal phenotype.

Functional diagnostic methods for biliary reflux gastritis

Reflux gastritis occurs with duodenogastric reflux of the contents of the duodenum with bile acids, lysolecithin, pancreatic enzymes, which leads to the appearance of clinical symptoms, endoscopic and histological changes indicative of chemical (reactive) gastritis (gastropathy). A distinction is made between primary reflux gastritis, which occurs in patients with slow gastric emptying, impaired antroduodenal coordination, gallbladder dyskinesia, and secondary, which develops after surgical interventions on the stomach and antroduodenal region [47, 60, 61].

There is no doubt that it is necessary to study both the composition of the refluxate that affects the gastric mucosa and the disturbance in the motor function of antroduodenal zone that lead to the development of duodenogastric reflux.

High-resolution antroduodenal manometry is a high-tech method that allows visualization of antroduodenal coordination: contractions of the antrum of the stomach with a frequency of 2–4 cycles per minute, the work of the pyloric canal pump, which, due to the pressure gradient between its proximal and distal sections, passes the contents of the stomach into the duodenum, cyclic contractions of the duodenum itself – 10–12 cycles per minute. Impaired peristalsis leads to the development of pylorospasm, gastroparesis and other conditions [62–64].

In wide clinical practice, it is possible to detect signs of reflux gastritis during EGDS with subsequent histological examination, but they are not

pathognomonic. The presence of bile in the stomach should not be regarded as confirmation of pathologic reflux since it is more often a consequence of regurgitation in response to the examination [65, 66]. Ultrasound examination of the stomach with contrast allows one to see the retrograde movement of the stream of hyperechoic inclusions from the pylorus to the body of the stomach, which can be interpreted as duodenogastric reflux [67].

Daily pH-metry of stomach allows for an objective assessment of the presence of duodenogastric reflux, which is defined as increase in pH in the stomach above 5.0 units, not associated with food intake, that is most often recorded at night. Reflux is considered pronounced if the duration of all refluxes exceeds 10 % of the time of monitoring the pH of the stomach. It should be noted that the value of the pH-metry method is that it allows for the detection of duodenogastric reflux in hypo- or anacid conditions, for example, in patients with atrophic gastritis as well as against the backgrounds of taking antisecretory therapy or after surgical interventions [68, 69].

Thus, antroduodenal manometry and pH-metry make it possible to establish the pathogenetic basis for the development of duodenogastric reflux, select treatment, and evaluate the effectiveness of the therapy. The significance of reflux gastritis as a precancerous disease is due to bile acids, which can contribute to carcinogenesis through various mechanisms. These mechanisms include the induction of intestinal metaplasia, modification of *H. pylori* colonization, and changes in the microbiota of the stomach [70, 71]. Timely diagnostics of reflux gastritis allows timely treatment, which can potentially be an effective approach to the prevention of gastric cancer.

Gastritis after successful eradication of *H. pylori* infection (post-eradication gastritis) and prevention of gastric cancer

In the Clinical Guidelines of RGA, eradication therapy for *H. pylori* in chronic gastritis is postulated as an etiotropic treatment to prevent the progression of atrophy, as a measure of primary prevention of gastric cancer and tertiary prevention for patients who have undergone endoscopic resection of early-stage gastric cancer [4, 5]. The preventive effect of *H. pylori* eradication is most pronounced in the absence of atrophy; patients with severe stages of atrophy, even after successful elimination of *H. pylori*, have an increased risk of developing gastric cancer [2, 4, 5, 37].

A consequence of the widespread eradication therapy for *H. pylori* infection is the appearance of a significant proportion of post-eradication gastritis in the structure of chronic gastritis [38]. When analyzing the results of histologic study of gastric biopsy specimens one year after eradication of the infection, restoration of the structure of the gastric mucosa with a complete reduction of inflammatory changes was noted in 13.2 % of cases, partial regression of inflammatory changes — in 58.6 %, and preservation of the severity of inflammatory changes — in 28.2 % of cases [72, 73]. The persistence of mononuclear inflammatory infiltrate in the post-eradication period is a factor that determines the rate of cellular renewal of the gastric mucosa; a direct correlation has been shown between the apoptosis index, the epithelial cell proliferation index and the severity of mononuclear infiltration of the lamina propria of the gastric stomach mucosa [72, 73].

Thus, it cannot be ruled out that chronic inflammation even after eradication of *H. pylori* infection may contribute to the progression of precancerous changes in the gastric mucosa. The Clinical Guidelines of RGA for the Diagnosis and Treatment of Gastritis and Duodenitis recommend therapy with bismuth tripotassium dicitrate or rebamipide for 4–8 weeks to enhance the protective properties of the gastric mucosa, which should be kept in mind even after successful eradication of *H. pylori* [4, 5].

The effectiveness of rebamipide in reducing histological inflammation has been demonstrated in patients with gastritis with persistent *H. pylori*, which proves the intrinsic anti-inflammatory effect of the drug. In the group of patients receiving rebamipide, a significant reduction in mononuclear infiltration in the antrum and body of the stomach, and neutrophil infiltration in the antrum was noted [74]. In patients with proven successful *H. pylori* eradication treated with rebamipide for 12 months and in the group without treatment, gastritis activity and atrophy scores improved in both cases without differences between them, which was explained by the result of *H. pylori* infection eradication. However, the severity of chronic inflammation of the gastric body was significantly reduced in the rebamipide group compared with the untreated group [75]. Evidence is accumulating of the positive effect of rebamipide on atrophy and intestinal metaplasia [17, 76, 77]. For example, in patients after endoscopic resection of the mucosa for dysplasia or early cancer, the degree of atrophy and the degree of intestinal metaplasia in the antral part of the stomach significantly decreased with rebamipide over 12 months [77]. These data allow us to recommend rebamipide treatment for chronic gastritis after eradication of *H. pylori* infection, including for the purpose of preventing gastric cancer.

Autoimmune gastritis

There are no data on the incidence of autoimmune gastritis in Russia. This makes the studies that show that autoimmune gastritis cannot be considered a rare disease even more important. Autoimmune gastritis based on the presence of antibodies to parietal cells in combination with low levels of pepsinogen I was detected in 26 % of patients with autoimmune thyroiditis and in 13 % of patients with autoimmune liver diseases [78]. In a retrospective analysis

of the morphological study of gastric biopsies over a five-year observation period, autoimmune gastritis accounted for 8.6 %, which, on the one hand, may be due to an increase in the incidence rate, and on the other hand, due to the "concentration" of patients with autoimmune gastritis in the specialized gastroenterology center for dynamic observation [38]. Autoimmune gastritis was often combined with reactive gastropathy in the antral section (35.3 %) and very rarely — with *H. pylori*-associated gastritis (3.3 %) [38]. Serological screening (a panel of determination of pepsinogen I, pepsinogen II, their ratio, gastrin-17 and antibodies to *H. pylori*) in asymptomatic adults followed by endoscopic examination with biopsy allowed us to estimate the adjusted prevalence of autoimmune gastritis as 2.6 % [79]. According to Z.M. Galeeva, 250 patients were included in the Regional Registry of patients with autoimmune gastritis (Kazan); with type I neuroendocrine tumors of the stomach detected in 4.3%.

Autoimmune gastritis is considered as precancerous disease in relation to gastric adenocarcenma [80–82]. It is interesting to note that in the case-control study the presence of any autoimmune disease (33 autoimmune diseases were included, such as autoimmune thyroiditis, type 1 diabetes mellitus, autoimmune liver diseases, etc.) was associated with an increased risk of gastric cancer (OR = 1.10; 95 % CI: 1.01–1.20), but pernicious anemia was characterized by the highest risk (OR = 2.75; 95% CI: 2.19–3.44) [83]. However, in a seven-year follow-up of patients with autoimmune gastritis, in whom *H. pylori* infection was carefully excluded in the medical history and in during the study, no increased risk of gastric cancer was observed. It has been suggested that the increased risk of adenocarcinoma in autoimmune gastritis is associated with previous or current *H. pylori* infection [84].

Thus, autoimmune gastritis provides a number of directions for further research devoted to the assessment of epidemiology, clinical picture and prognosis [85]. The use of rebamipide in real clinical practice for autoimmune gastritis should be analyzed and become the subject of adequately designed studies.

Algorithm for diagnostics and treatment of chronic gastritis

The members of the Expert Panel proposed an algorithm for the diagnosis and treatment of *H. pylori*-associated, post-eradication non-Helicobacter gastritis (Fig).

Resolution of the Expert Panel

1. Diagnostics of gastritis should be aimed at establishing the etiology of this disease and the histological stage, which determines the prognosis, observation tactics and medication therapy.

2. The prevalence and pathogenesis of dyspeptic symptoms and other symptoms in various etiological variants of gastritis require further study, including the use of 24-hour pH-metry, and methods for studying the motility of the upper gastrointestinal tract.

3. For the treatment of dyspepsia and other symptoms in various etiological variants of gastritis and functional dyspepsia it is advisable to prescribe rebamipide as monotherapy and in combination with other drugs.

4. Eradication therapy for *H. pylori* allows for etiologic treatment of gastritis, stopping the progression of atrophy and intestinal metaplasia of the gastric mucosa and effective prevention of gastric cancer. Anti-Helicobacter therapy should be considered in each case of *H. pylori* infection.

5. Data obtained in international and domestic clinical studies allow us to recommend rebamipide for inclusion in *H. pylori* eradication therapy regimens.

6. Patients with severe atrophic gastritis (severe atrophy and/or intestinal metaplasia in the stomach body and in antrum — OLGA/OLGIM III/IV) remain at risk for developing gastric cancer even after successful eradication of *H. pylori* infection and are recommended to have high-quality endoscopic surveillance every 3 years.

7. Experimental and clinical data allow us to consider rebamipide as a promising drug for reducing inflammatory and atrophic changes in the gastric mucosa. The effectiveness of chemoprophylaxis of gastric cancer with rebamipide (including determining the duration of its administration) requires further study.

8. Autoimmune gastritis should not be considered a rare disease; the study of its epidemiology and prognosis is of significant clinical importance.

9. Further research into diagnostic methods, treatment and prognosis assessment for reflux gastritis is needed. The basic treatment for this form of gastritis is ursodeoxycholic acid.

10. The creation of data sets and registries of *H. pylor*-associated, post-eradication and non-Helicobacter gastritis both in Russia and different regions of the Russian Federation will allow us to establish the epidemiology, clarify the prognosis, observation tactics and treatment of gastritis.

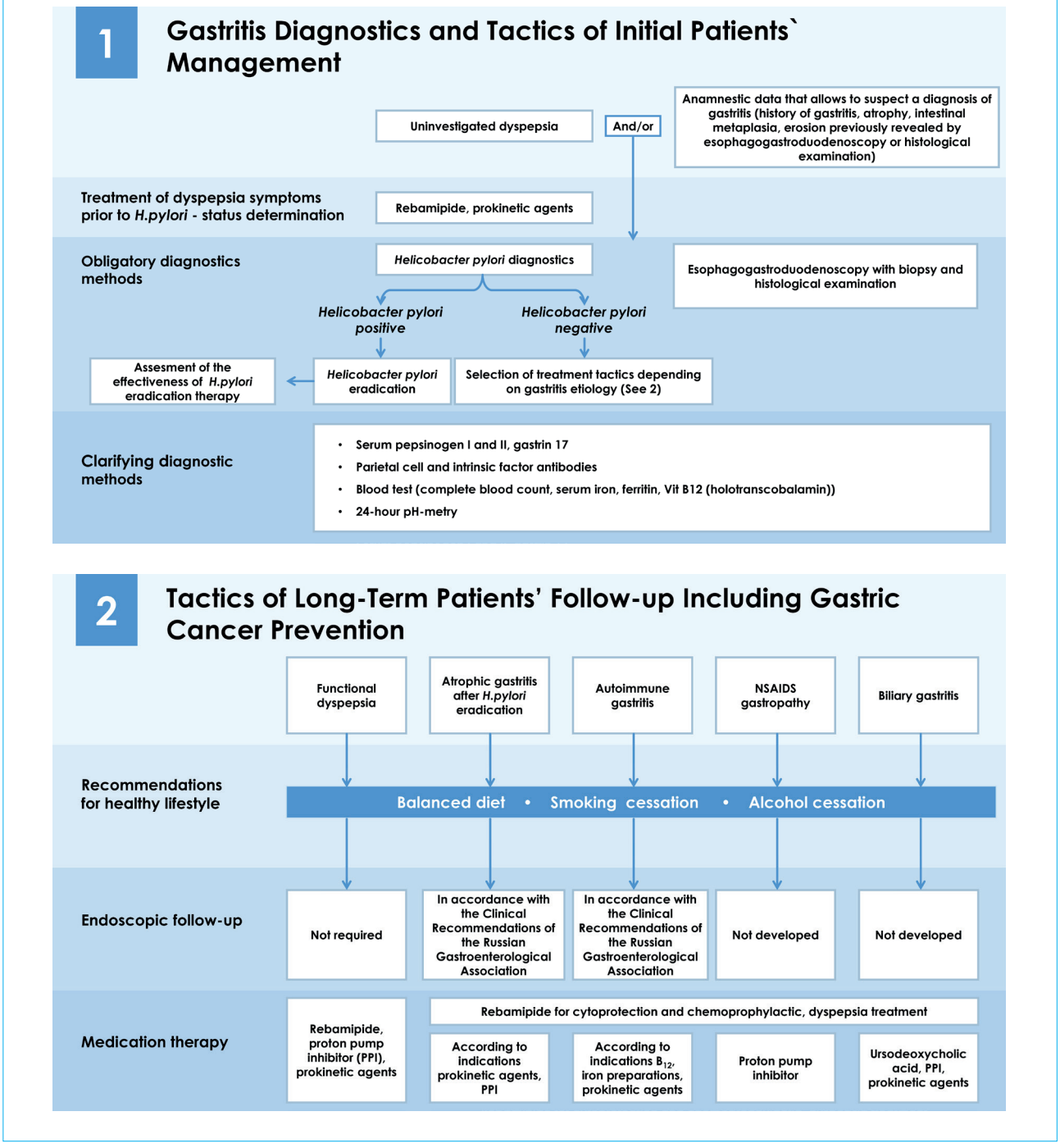

Figure. Algorithm of diagnostics and treatment of *H. pylori*-associated, post-eradication and non-Helicobacter gastritis

Рисунок. Алгоритм диагностики и лечения *H. pylori*-ассоциированного, постэрадикационного и негеликобактерного гастритов

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