

**U.K. Abdullaeva**Bukhara State Medical Institute, Uzbekistan, Bukhara  
e-mail: abumkur14@gmail.com

## RETROSPECTIVE ANALYSIS OF GASTRITIS ASSOCIATED WITH CHRONIC ATROPHIC H. PYLORI IN PATIENTS WITH NOCARDIAL GASTRIC CANCER IN BUKHARA CITY AND PREVENTION OF ATROPHIC PROCESSES OF GASTRIC MUCOSA

Atrophic gastritis is understood as a progressive inflammatory process of the gastric mucosa, characterized by the loss of gastric glands. *Helicobacter pylori* infection (HP) and autoimmune gastritis are recognized as the most common etiological factors causing atrophic gastritis. The results of a retrospective analysis of the prevalence of chronic atrophic gastritis associated with *H. pylori* in patients with non-cardiac gastric cancer in Bukhara city were described. In addition, an algorithm for early diagnosis of atrophic processes of the gastric mucosa has been developed. When the incidence rate of chronic atrophic gastritis associated with *H. pylori* was analyzed in patients with non-cardiac gastric cancer in the city of Bukhara, it was noted that it tended to increase for 5 years from 2015 to 2019. Based on the research results, a personal algorithm for the formation of risk groups of atrophic processes of the gastric mucosa was developed. A personalized algorithm allows forming risk groups for atrophic processes of the gastric mucosa based on serological parameters, as well as taking into account data on genetic predisposition.

**Keywords:** chronic atrophic gastritis, gastric mucosa, *H. pylori*, non-cardiac gastric cancer.

### Introduction

Atrophic gastritis is an urgent problem of modern gastroenterology in our country and around the world as it may transform into gastric cancer [1, 2]. The clinical and morphological feature of atrophic gastritis is a decrease in the number of specialized glandulocytes that provide secretory function of the stomach, and their replacement with simpler cells, including those that produce mucus. Extensive atrophy of the mucous membrane of the body of the stomach, as a rule, is associated with hyposecretion of hydrochloric acid and impaired pepsinogen production [3, 4]. Moreover, the occurrence of the vast majority of atrophic gastritis is associated with Long-existing superficial *H. pylori* gastritis is transformed into atrophic without appropriate treatment [5, 6].

Atrophic gastritis clinically, as a rule, does not manifest itself for a long time, therefore, the diagnosis of chronic gastritis is more morphological than clinical [7, 8]. Gastric cancer (GC) is a global health burden and the fourth most common cause of death from cancer in the world. A sequential histopathology cascade for the development of gastric adenocarcinoma of the intestinal type – from normal gastric epithelium to chronic gastritis, chronic atrophic gastritis (CAG) and intestinal metaplasia (IM), followed by dysplasia and, finally, GC. Patients with precancerous diseases, such as CAG or dysplasia, have a significant risk of developing cancer, and early de-

tection of these lesions is important for screening for cancer [8, 9]. For CAG population screening, the endoscopic mass screening program has been shown to be effective in countries with a predominant GC, such as Korea and Japan. Overview of modern concepts of gastric metaplasia and gastric cancer. An endoscopic screening program reduced mortality rate associated with cancer by 47% as part of a case-control study in Korea. The effectiveness of the Korean National Cancer Program in reducing stomach cancer mortality [10].

The aim of the study was to study the endoscopic and morphological features of the mucous membrane of the stomach and intestines using the OLGA system in chronic atrophic gastritis.

### Materials and Methods

During 2015-2019, a retrospective analysis of 152 patients diagnosed with non-cardiac gastric cancer was conducted at the Bukhara branch of the Republican Specialized Oncology and Radiology Scientific and Practical Medical Center based on their medical records and medical history. Based on the research results, a personal algorithm for the formation of risk groups of atrophic processes of the gastric mucosa was developed.

The results obtained during the study were subjected to statistical processing using SPSSv.15.0 (2007) and MS Excel software package for Windows

XR. The following values and criteria were calculated using standard methods of variation series: arithmetic mean value (M), arithmetic mean error ( $\mu$ ). Student's t-test was used to assess the statistical significance of differences between two selected indicators.

### Results

According to the results of the analysis, the incidence of chronic atrophic H. pylori-related gastritis in patients with non-cardiac gastric cancer was 66.1% in 2015, 68.9% in 2016, 69.1% in 2017, and 69.1% in 2018. – 68.6%, in 2019 – 69.5%.

Detection of atrophic changes of the gastric mucosa during preventive medical examinations in the city of Bukhara is very low, in 2015 it was 0.3%, and in 2019 it increased to 3%, which may be related to the beginning of the widespread use of EFGDS along with biopsy in people at risk.

The gender distribution of chronic atrophic H. pylori-associated gastritis in patients with non-cardiac gastric cancer revealed an annual increase in the number of men and a decrease in the number of women. Thus, in 2015, the number of men and women with non-cardiac gastric cancer was 68.4% and 32.6%, respectively, and in 2019, it was 72.8% and 27.2%.

The most common chronic atrophic H. pylori-associated gastritis in patients with non-cardiac gastric cancer was found at the age of 50-65 years, in women at the age of 66-80 years. It should be noted that individual cases of this pathology occurred at the age of 25-35 years, which indicates a younger age of atrophic changes in patients with non-cardiac gastric cancer.

According to WHO recommendations, H. pylori

is the first factor of carcinogenesis. Bukhara region is one of the regions with high prevalence of H. pylori. Accordingly, the incidence of non-cardiac gastric cancer in these regions may be directly related to the prevalence of H. pylori. Therefore, in our study conducted in the city of Bukhara, a high incidence of chronic H. pylori-associated atrophic gastritis was found in patients with non-cardiac gastric cancer.

#### *Preventing the formation of atrophic processes of the gastric mucosa*

Based on the results obtained during the study, a personal algorithm for the formation of risk groups of atrophic processes of the gastric mucosa was developed. According to this algorithm, risk groups were divided into 3: low, medium and high risk of atrophic processes of the gastric mucosa.

#### *Low risk group of atrophic processes of the gastric mucosa*

In the low-risk group, if H. pylori is negative, pepsinogen I, pepsinogen II, pepsinogen I/pepsinogen II is normal, if there is no genetic predisposition to non-cardiac gastric cancer, if he does not smoke, if he does not consume high-salt products, he is considered as a healthy stomach, and EFGDS is not necessary. If H. pylori (+), pepsinogen I, pepsinogen II, pepsinogen I/pepsinogen II are normal or increased, chronic H. pylori-associated nonatrophic gastritis, if H. pylori (+), pepsinogen I is increased; pepsinogen II, pepsinogen I / pepsinogen II normal or increased, with frequent use of nonsteroidal anti-inflammatory drugs, there is a risk of erosive ulceration of the stomach and duodenum, and in both cases, eradication therapy is necessary. And EFGDS is prescribed according to the guidelines, ie, smoking, hereditary predisposition to non-cardiac gastric cancer and intake of high-salt products (Figure 1).

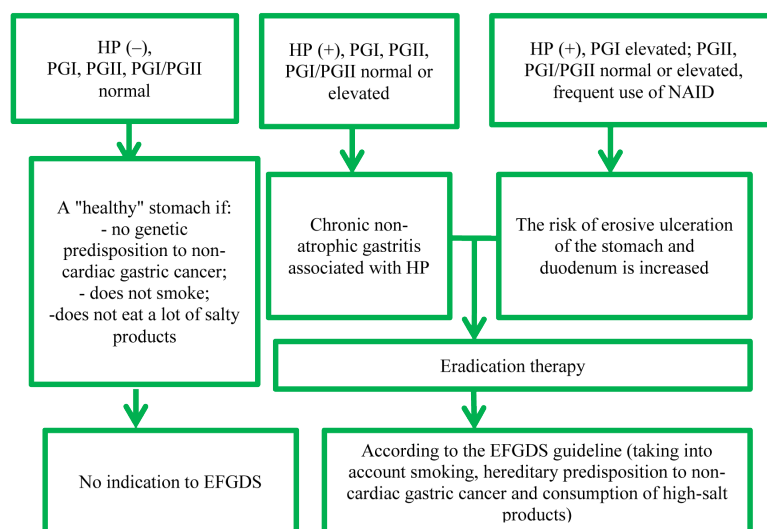
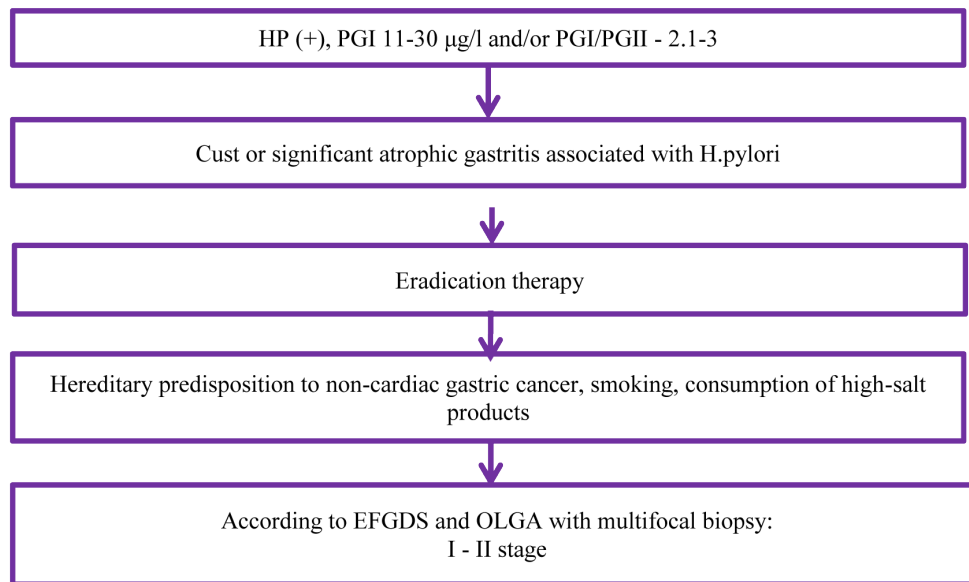


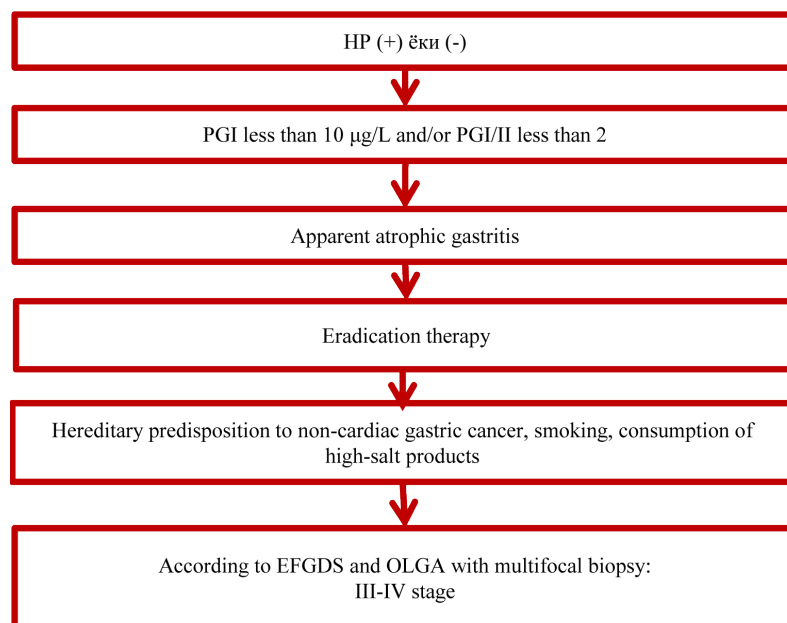
Figure 1 – Low risk of atrophic processes of the gastric mucosa

In the risk group, if HP+, PGI 11-30 µg/l and PGI/PGII – 2.1-3, HP is evaluated as associated slow or significant atrophic gastritis, in which eradication therapy is necessary, if the patient has a hereditary predisposition to non-cardiac gastric cancer. and if he eats a lot of salty products, EFGDS is performed along with multifocal biopsy, and according to OLGA, it is evaluated as gastritis level 1 or 2 (Fig. 2).



**Figure 2** – Average risk of atrophic processes of the gastric mucosa

In the high-risk group, regardless of whether HP (+) or HP (-), PGI is less than 10 µg/l and PGI/II is less than 2, it is evaluated as a single atrophic gastritis, eradication therapy is necessary, if the patient has a history of non-cardiac gastric cancer. If there is a tendency to eat a lot of salty products, EFGDS is performed along with multifocal biopsy, and according to OLGA, it is estimated as 3 or 4 stages of gastritis (Fig. 3).



**Figure 3** – High risk of atrophic processes of the gastric mucosa

Thus, a personalized algorithm allows forming risk groups for atrophic processes of the gastric mucosa based on serological parameters, as well as taking into account data on genetic predisposition. People diagnosed with atrophic processes of the gastric mucosa require observation using endoscopic and morphological studies.

### Discussion

To date, there is a certain understanding of the serological diagnosis of atrophic processes of the gastric mucosa in the world. Over the years, studies by various authors have confirmed that atrophic fundal gastritis can be successfully determined by serum pepsinogen-I or the ratio of pepsinogen-I to pepsinogen-II. It has been shown that serological screening using a set of biomarkers can serve as an early indicator of pre-tumor pathology and gastric cancer [16]. Thanks to national cancer control programs and mass screening, which includes serological methods, Japan has the highest stomach cancer survival rate in the world at 53%, compared to less than 20% in other countries. At the same time, the proportion of early cancer detected in the total composition of patients with gastric cancer is increasing [7].

A number of results have been achieved in research conducted in the world to study the early diagnosis of atrophic processes of the gastric mucosa, including: it was found that the low level of pepsinogen-I and pepsinogen-I / pepsinogen-II are predictive for the risk of gastric cancer [8]; it was found that the method of early detection of atrophic gastritis through the level of pepsinogens allows to reliably determine the presence of atrophy of the gastric body and assess its severity [8]; mass screening of gastrin –

17 and pepsinogen – I serological markers has been proven to create an opportunity to characterize the risk of atrophy of the gastric mucosa [9]. It has been found that the use of serum biomarkers such as IgG antibodies, pepsinogens produced against *H. pylori* can reduce the cost of gastric cancer screening and the public health burden [10].

### Conclusion

In the program developed for the early diagnosis of atrophic processes of the gastric mucosa, CAG risk factors are listed, each indicator is evaluated with points, and the severity of atrophy is determined based on the total sum. According to this algorithm, risk groups are divided into 3: with low, medium and high risk of atrophic processes of the gastric mucosa. Thus, if there is a low risk of atrophic processes of the gastric mucosa, EFGDS is performed based on the doctor's decision and taking into account the anamnesis and the clinic. At moderate and high risk of atrophic processes of the gastric mucosa, it is recommended to conduct a large number of biopsies with EFGDS and subsequent stratification of the risk of atrophic processes of the gastric mucosa taking into account the morphological systems OLGA and OLGIM. If *H. pylori* are detected, eradication therapy is necessary.

In conclusion, it can be said that based on these results, serological methods are compatible with morphological methods in terms of their diagnostic value. But in patients, if the atrophic processes are at the 2-3 level, nothing can replace the morphology. Serological methods can be used for screening or observation of patients with mild forms of gastritis, i.e., non-atrophic gastritis or slow or significant atrophy of atrophic gastritis.

### References

1. Abdullaeva U.K. Predicting the risk of atrophic transformation in gastritis associated with chronic *Helicobacter pylori* // abstract of PhD dissertation on medical sciences. – Tashkent. 2021. – 46 p.
2. Abdullaeva U.K. Predicting the risk of atrophic transformation in chronic gastritis using serum pepsinogen // World journal of pharmaceutical research, Faculty of Pharmacy Medical University, Bulgaria. – Vol. 8, Iss. 13. – 2019. – P. 219-228.
3. Abdullaeva U.K., Sobirova G.N., Karimov M.M., Aslonova I.J. The prevalence and possibilities of prevention of noncardial gastric cancer in the Bukhara region // American journal of medicine and medical sciences. – 2020, 10(9). – P. 679-681.
4. Sobirova G.N., Abdullaeva U.K., Nosirova M.S., Aslonova I.J. Evaluation of the gastrointestinal mucosa by the OLGA system in chronic atrophic gastritis // Journal of critical reviews, Kuala Lumpur, Malaysia. – Vol. 7, Iss. 2, – 2020. – P. 409-413.
5. Dinis-Ribeiro M., Kashin S.V., Kuvaev R.O., Nadezhin A.S. et al. // Draft Recommendations of the Russian Endoscopic Society for Endoscopists, Gastroenterologists, Therapists, Oncologists and Surgeons. – 2012. – No. 44. – P. 74-94.
6. Sugano K., Tack J., Kuipers E.J. et al. Kyoto global consensus report on *Helicobacter pylori* gastritis // Gut. – 2015. – Vol. 64 (9). – P. 1353-1367.
7. Sun X., Bi Y., Nong B., et al. Linked color imaging confers benefits in profiling *H. pylori* infection in the stomach. // Endosc Int open. – 2019;7(7): E885-92.
8. Syrjänen K. A Panel of Serum Biomarkers (GastroPanel®) in Non-invasive Diagnosis of Atrophic Gastritis. Systematic Review and Meta-analysis // Anticancer Res. – 2016. – Vol. 36 (10). – P. 5133-5144.
9. Takeda T., Asaoka D., Nojiri S., et al. Linked color imaging and the Kyoto classification of gastritis: evaluation of visibility and inter-Rater reliability. // Digestion. – 2019:1-10.
10. Telaranta-Keerie A., Kara R., Paloheimo L. et al. Prevalence of undiagnosed advanced atrophic corpus gastritis in Finland: an observational study among 4,256 volunteers without specific complaints // Scand J Gastroenterol. – 2010. – Vol. 45. – P. 1036-1041.