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## USING THE OLGA SYSTEM IN CHRONIC ATROPHIC GASTRITIS

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✓ ***Resume,***

***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. The study included 180 patients with dyspepsia symptoms ranging in age from 18 to 80 years old, who were diagnosed with H.pylori - associated gastritis during endoscopic examination with a quick urease test. Nonatrophic fundus gastritis was detected in 99 patients (55.0%), mild atrophy of the mucous membrane of the stomach body was detected in 19 patients (10.56%), moderate atrophy in 36 patients (20.0%) and severe atrophy in 26 patients (14.44%). Thus, our data show that the stage of atrophy, established by the OLGA system, generally corresponds to the stage of atrophy, determined by the visual-analogue scale of the Houston modification of the Sydney system.***

***Key words: chronic atrophic gastritis, intestinal metaplasia, morphology, gastric mucosa, H. pylori.***

## ИСПОЛЬЗОВАНИЕ СИСТЕМЫ OLGA ПРИ ХРОНИЧЕСКОМ АТРОФИЧЕСКОМ ГАСТРИТЕ

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✓ ***Резюме,***

***Целью исследования было изучение эндоскопических и морфологических особенностей слизистой оболочки желудка и кишечника с использованием системы OLGA при хроническом атрофическом гастрите. В исследование были включены 180 пациентов с симптомами диспепсии в возрасте от 18 до 80 лет, у которых был диагностирован ассоциированный с H.pylori гастрит во время эндоскопического исследования с быстрым уреазным тестом. Неатрофический гастрит (во дне) был обнаружен у 99 пациентов (55,0%), легкая атрофия слизистой оболочки желудка была обнаружена у 19 пациентов (10,56%), умеренная атрофия у 36 пациентов (20,0%) и тяжелая атрофия у 26 пациентов (14,44). %). Таким образом, наши данные показывают, что стадия атрофии, установленная системой OLGA, в целом соответствует стадии атрофии, определяемой визуально-аналоговой шкалой хьюстонской модификации системы Сиднея.***

***Ключевые слова: хронический атрофический гастрит, кишечная метаплазия, морфология, слизистая оболочка желудка, H. pylori.***

## OLGA ТИЗИМИНИНГ СУРУНКАЛИ АТРОФИК ГАСТРИТДА ҚЎЛЛАНИЛИШИ

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✓ ***Резюме,***

***Тадқиқот мақсади сурункали атрофик гастритда OLGA тизимидан фойдаланган ҳолда ошқозон ва ичак шиллиқ қаватининг эндоскопик ва морфологик хусусиятларини ўрганиш эди. Тадқоқотда 18 дан 80 ёшгача бўлган, диспепсия аломатлари бўлган, эндоскопик текширув пайтида тез уреаза тести билан H. pylori билан боғлиқ гастрит ташхиси қўйилган 180 бемор иштирок этди. Атрофик бўлмаган туб гасрит 99 беморда (55%), меъда шиллиқ қаватининг енгил атрофияси 19 беморда (10,96%), 36 беморда ўртача атрофия (20%) ва 26 беморда оғир атрофия аниқланди (14,44%). Шундай қилиб, бизнинг маълумотларимиз шуни кўрсатадики, OLGA тизими томонидан ўрнатилган атрофия даражаси, Сидней тизимининг Хюстон модификациясининг визуал-аналогик шкаласи билан аниқланган атрофия босқичига тўғри келади.***

***Калит сўзлар: сурункали атрофик гастрит, ичак метаплазияси, морфология, ошқозон шиллиқ қавати, H. pylori.***

### Introduction

Atrophic gastritis is an urgent problem of modern gastroenterology in our country and around the world, in connection with the transformation into gastric cancer [1,2,4].

Helicobacter pylori infection (HP) and autoimmune gastritis are recognized as the most common etiological factors causing atrophic gastritis [3,6,7]. Moreover, the occurrence of the vast majority of atrophic gastritis is associated with H. pylori. The bacteria H. pylori, persisting on the gastric epithelium, cause chronic Helicobacter pylori gastritis. Long-existing superficial Helicobacter pylori gastritis is transformed into atrophic without appropriate treatment [1,2,5,8].

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 [1,4,9,11].

Stomach cancer (RH) is a global health burden and the fourth most common cause of cancer death in the world [10,12,14]. 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, RG. Patients with precancerous diseases, such as CAH or dysplasia, have a significant risk of developing cancer, and early detection of these lesions is important for screening for cancer [13, 15, 16].

## Material and methods

The study included 180 patients with dyspepsia symptoms ranging in age from 18 to 80 years old, who were diagnosed with H.pylori - associated gastritis during endoscopic examination with a quick urease test. Men accounted for 33.89% of the respondents (61 people), women - 66.11% (119 people). Prior to the endoscopic examination, none of the patients took drugs from the groups of proton pump inhibitors or non-steroidal anti-inflammatory drugs.

The control group consisted of H. pylori-negative volunteers aged 17 to 45 years, in whom there was no history of gastrointestinal tract diseases and gastroduodenal mucosa was assessed as practically unchanged with endoscopy.

The average age of the studied patients was 43.3 ± 7.4 years, while the greatest number of them belonged to the age groups of 31-40 years and 41-59 years.

## Research results

Depending on the presence and severity of atrophy in the stomach, patients were divided as follows. Neatrophic fundus gastritis was detected in 99 patients (55.0%), mild atrophy of the mucous membrane of the stomach body was detected in 19 patients (10.56%), moderate atrophy in 36 patients (20.0%) and severe atrophy in 26 patients (14.44%).

Depending on the presence and severity of atrophy of the mucous membrane of the antrum, the studied patients were divided as follows: non-atrophic gastritis was detected in 12 patients (6.67%), mild atrophy of the gastric mucosa in 35 patients (19.44%), moderate atrophy - in 69 patients (38.33%) and severe atrophy - in 64 patients (35.56%).

When comparing the results of the diagnosis of atrophy in the antrum and in the body of the stomach (table 1), we revealed significant differences: in the body of the stomach, significantly more significant non-atrophic gastritis was detected, and in the antrum - significantly more often than in the body, any degree of atrophy of the gastric mucosa developed (in all cases, P <0.05).

Table 1.

**Statistical analysis of the incidence of atrophy of the mucous membrane in the antrum and body of the stomach**

| Degree of atrophy | Atrophy detection rate (%) | | P |
|---|---|---|---|
| | Body | Antrum | |
| Nonatrophic gastritis | 55,0 | 6,67 | <0,05 |
| Mild atrophy | 10,56 | 19,44 | <0,05 |
| Moderate atrophy | 20,0 | 38,33 | <0,05 |
| Severe atrophy | 14,44 | 35,56 | <0,05 |

Table 2.

**Statistical analysis of the incidence of mucosal atrophy in multifocal and focal chronic gastritis**

| Stage of atrophy | Atrophy detection rate (%) | | | $P_{1-3}$ | $P_{2-3}$ |
|---|---|---|---|---|---|
| | 1. Corpus dominant atrophy | 2. Antrum dominant atrophy | 3. Multifocal atrophy | | |
| Nonatrophic gastritis | 55,0 | 6,67 | 21,7 | <0,05 | <0,05 |
| Mild atrophy | 10,56 | 19,44 | 25,5 | <0,05 | >0,05 |
| Moderate atrophy | 20,0 | 38,33 | 35,6 | <0,05 | >0,05 |
| Severe atrophy | 14,44 | 35,56 | 17,2 | >0,05 | <0,05 |

Depending on the presence and severity of multifocal atrophy of the gastric mucosa (simultaneous presence of atrophic changes in the body and antrum), the studied patients were distributed as follows (table 2): lack of multifocal atrophy - 39 patients (21.7%), a weak degree of multifocal atrophy was detected in 46 patients (25.5%), moderate degree of atrophy in 64 patients (35.6%) and severe multifocal atrophy in 31 patients (17.2%).

The development of intestinal metaplasia in our observations was detected in 89 out of 180 patients (49.4%) and was accompanied by a local disappearance of H. ruli contamination in the metaplasia zones and a decrease in the activity of the inflammatory reaction. Most often, full small bowel metaplasia was found in the preparations, characterized by the presence of absorbent epithelial cells, goblet cells containing sialomucins, and Panet cells. Less often, incomplete small intestinal metaplasia was detected, characterized by the presence of goblet cells containing sialomucins among the integumentary-fossa epithelium of the gastric type. Finally, in a number of cases, we revealed foci of large intestinal metaplasia with the presence of goblet cells separated by high prismatic epithelial cells with an abundant content of sulfomucins.

In all cases, the presence of intestinal metaplasia was accompanied by atrophic changes in the gastric mucosa, expressed to one degree or another. In antrum-dominant chronic atrophic gastritis, intestinal metaplasia of the gastric epithelium was histologically detected in 84 (50%) of 168 patients. In this case, a weak and moderate prevalence of intestinal metaplasia prevailed.

In the group of patients with corpus dominant chronic atrophic gastritis, we identified 33 cases of the development of intestinal metaplasia of gastric epithelium (40.74%) out of 81 patients. Moreover, as in the case of antrumdominant chronic atrophic gastritis, a weak and moderate degree of prevalence of intestinal metaplasia predominated.

In addition to intestinal metaplasia, in patients with corpus dominant atrophic gastritis, we recorded the presence of pseudopiloric metaplasia, or "anthralization," of the gastric mucosa. At the same time, two main localizations of the development of pseudopiloric metaplasia were distinguished: in the area of the actually large curvature of the stomach body and in the area of angular incision of the stomach, where the main gastric glands normally prevail. Pseudo-pyloric mucosal metaplasia in the incision region was detected in 53 of 81 (65.4%) patients with corpus dominant atrophic gastritis, and in the area of great curvature of the body of the stomach, in 10 of 81 (12.3%) patients.

In the group of patients with multifocal atrophy of the gastric mucosa, we identified 81 cases of the development of intestinal metaplasia of gastric epithelium (57.45%) out of 141 patients.

For further studies, we used biopsy specimens from 55 patients who were histologically diagnosed with H. pylori-associated atrophic gastritis.

In the antrum, atrophic changes of various degrees prevailed in 20 of 55 patients (36.36%), of which a weak degree of atrophy was detected in 5 patients, moderate in 9 patients and severe in 6 patients.

Corpus dominant chronic atrophic gastritis was diagnosed in 16 patients out of 55 (29.09%), of which a weak degree of atrophy was determined in 3 patients, moderate in 6 patients and severe in 7 patients.

We diagnosed multifocal chronic atrophic gastritis in 19 of 55 patients (34.55%), of which a weak degree of atrophy was determined in 6 patients, moderate in 6 patients and severe in 7 patients.

Then, the data obtained during histological verification of the stage of atrophy in accordance with the visual-analogue scale of the Houston modification of the Sydney system were entered into the table of the OLGA system. After distributing the obtained data in the table, according to the OLGA system, the following results were obtained (table 3): cases of a weak degree of atrophy, determined in accordance with the visual-analogue scale, corresponded mainly to the first stage of development of atrophy according to the OLGA system 50%, cases of moderate atrophy corresponded mainly to the second stages of development of atrophy according to the OLGA system 38.1%., cases of severe atrophy corresponded mainly to the fourth stage of development of atrophy according to the OLGA system 40%.

Table 3.

**Stage atrophy in chronic atrophic gastritis according to OLGA systems**

| All stages | Число пациентов | St 0 | St 1 | St 2 | St 3 | St 4 |
|---|---|---|---|---|---|---|
| Atrophy | 55 | 0 | 19 | 14 | 9 | 13 |
| mild | 14 | 0 | 7 | 4 | 2 | 1 |
| moderate | 21 | 0 | 6 | 8 | 3 | 4 |
| severe | 20 | 0 | 6 | 2 | 4 | 8 |

Next, we analyzed the prevalence of intestinal metaplasia of the gastric mucosa, depending on the stage of its atrophy according to the OLGA system. As the results showed, a histologically verified weak prevalence of intestinal metaplasia was determined mainly in the first and second stages of atrophy according to the OLGA system, respectively, at 35.29% (table 4).

When analyzing the data obtained, it was found that the presence of inflammatory infiltration of the gastric mucosa in patients with chronic atrophic gastritis corresponded to the second, third or fourth degree according to the OLGA system, while the "zero" and first degrees were not detected.

Such results can be explained by the predominant development of a more pronounced intensity of the inflammatory reaction in one of the stomach sections (corpus-dominant or antrum-dominant gastritis), which, when distributed according to the OLGA system due to the summation of indicators from the body and antrum, led to a higher degree of inflammatory infiltration than when assessed by a visual analogue scale as a separate indicator for each section of the stomach.

Table 4.

**Identification of intestinal metaplasia at different stages of atrophy of the gastric mucosa in chronic atrophic gastritis according to the OLGA system**

| All stages | Number of patients | St 0 | St 1 | St 2 | St 3 | St 4 |
|---|---|---|---|---|---|---|
| Intestinal metaplasia | 42 | 0 | 11 | 15 | 12 | 4 |
| Weak | 17 | 0 | 6 | 6 | 5 | 0 |
| moderate | 18 | 0 | 5 | 7 | 5 | 1 |
| Severe | 7 | 0 | 0 | 2 | 2 | 3 |

## Conclusion

Thus, our data show that the stage of atrophy, established by the OLGA system, generally corresponds to the stage of atrophy, determined by the visual-analogue scale of the Houston modification of the Sydney system. Moreover, an increase in the stage of atrophy of the gastric mucosa, determined by the OLGA system, is associated with an increase in the severity of preneoplastic changes - intestinal metaplasia and dysplasia, which confirms the known relationship of these morphological changes in the cascade of gastric carcinogenesis. The degree of inflammatory infiltration of the gastric mucosa, determined by the OLGA system, also corresponds to the degree of inflammatory infiltration established during the assessment using a visual-analogue scale.

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