

Korolenko R. N., Avramenko A. A. The frequency of detection of the active form of helicobacter pylori infection in patients with chronic non-atrophic gastritis with biliary dyskinesia on the hypotonic hypokinetic type and with preserved function of the gall bladder. Journal of Education, Health and Sport. 2019;9(2):471-479. eISSN 2391-8306. DOI <http://dx.doi.org/10.5281/zenodo.2583214>  
<http://ojs.ukw.edu.pl/index.php/johs/article/view/6652>  
<https://pbn.nauka.gov.pl/sedno-webapp/works/906515>

The journal has had 7 points in Ministry of Science and Higher Education parametric evaluation, Part B item 1223 (26/01/2017).  
1223 Journal of Education, Health and Sport eISSN 2391-8306 7

© The Authors 2019;

This article is published with open access at Licensee Open Journal Systems of Kazimierz Wielki University in Bydgoszcz, Poland

Open Access. This article is distributed under the terms of the Creative Commons Attribution Noncommercial License which permits any noncommercial use, distribution, and reproduction in any medium, provided the original author(s) and source are credited. This is an open access article licensed under the terms of the Creative Commons Attribution Non commercial license Share alike. (<http://creativecommons.org/licenses/by-nc-sa/4.0/>) which permits unrestricted, non commercial use, distribution and reproduction in any medium, provided the work is properly cited.

The authors declare that there is no conflict of interests regarding the publication of this paper.

Received: 05.02.2019. Revised: 11.01.2019. Accepted: 28.02.2019.

UDC 613.33-002.2-008.87+616.361

## **THE FREQUENCY OF DETECTION OF THE ACTIVE FORM OF HELICOBACTER PYLORI INFECTION IN PATIENTS WITH CHRONIC NON- ATROPHIC GASTRITIS WITH BILIARY DYSKINESIA ON THE HYPOTONIC HYPOKINETIC TYPE AND WITH PRESERVED FUNCTION OF THE GALL BLADDER**

**R. N. Korolenko\*, A. A. Avramenko\*\***

**\*City Hospital № 4, Nikolaev**

**\*\*Petro Mohyla Black Sea National University, Nikolaev**

**(Ukraine)**

**aaahelic@gmail.com**

### **Abstract**

The frequency of detection and the degree of dissemination of the gastric mucosa by the active form of Helicobacter pylori in 70 patients with chronic non-atrophic gastritis with biliary tract dyskinesia (BTD) by hypotonic hypokinetic type and in 70 patients with chronic non-atrophic gastritis with preserved function of the bladder were analyzed. In the group of patients with BTD according to the hypotonic hypokinetic type, the frequency of occurrence of the active form of Helicobacter pylori infection in different topographic zones of the stomach ranged from 24.3% to 35.7% with a moderate degree of mucous contamination - (+), while in the group of patients with preserved the function of the gallbladder, the frequency of occurrence of the active form of Helicobacter pylori infection in different topographic zones

of the stomach ranged from 61.4% to 71.1% with a moderate degree of dissemination of the mucous membrane (++)).

**Key words: chronic non-atrophic gastritis, hypotechnical hypokinetic type of hyperplasia of hyperplasia, preserved gallbladder function.**

УДК 613.33-002.2-008.87+616.361

**Частота виявлення активної форми гелікобактерної інфекції у хворих на хронічний неатрофічний гастрит зі дискінезією жовчовивідних шляхів за гіпотонічним гіпокінетичним типом і зі збереженою функцією жовчного міхура**

**Р. М. Короленко, А. О. Авраменко**

#### **Резюме**

Було проаналізовано частоту виявлення і ступінь обсіменіння слизової шлунка активною формою гелікобактерної інфекції у 70-ти хворих на хронічний неатрофічний гастрит з дискінезією жовчовивідних шляхів (ДЖВШ) за гіпотонічним гіпокінетичним типом і у 70-ти хворих на хронічний неатрофічний гастрит зі збереженою функцією жовчного міхура. У групі хворих з ДЖВШ по гіпотонічному гіпокінетичним типом частота виявлення активної форми гелікобактерної інфекції у різних топографічних зонах шлунка коливалася від 24,3% до 35,7% при середньому ступені обсіменіння слизової - (+), в той час як у групі хворих зі збереженою функцією жовчного міхура частота виявлення активної форми гелікобактерної інфекції у різних топографічних зонах шлунка коливалася від 61,4% до 71,1% при середньому ступені обсіменіння слизової - (++)).

**Ключові слова: хронічний неатрофічний гастрит, ДЖВП за гіпотонічним гіпокінетичним типом, збережена функція жовчного міхура.**

**Частота выявления активной формы хеликобактерной инфекции у больных хроническим неатрофическим гастритом с дискинезией желчевыводящих путей по гипотоническому гипокинетическому типу и с сохранённой функцией жёлчного пузыря**

**Р. Н. Короленко, А. А. Авраменко**

**Резюме**

Были проанализированы частота выявления и степень обсеменения слизистой желудка активной формой хеликобактерной инфекции у 70-ти больных хроническим неатрофическим гастритом с дискинезией желчевыводящих путей (ДЖВП) по гипотоническому гипокинетическому типу и у 70-ти больных хроническим неатрофическим гастритом с сохранённой функцией жёлчного пузыря. В группе больных с ДЖВП по гипотоническому гипокинетическому типу частота встречаемости активной формы хеликобактерной инфекции в разных топографических зонах желудка колебалась от 24,3% до 35,7 % при средней степени обсеменения слизистой - (+), в то время как в группе больных с сохранённой функцией жёлчного пузыря частота встречаемости активной формы хеликобактерной инфекции в разных топографических зонах желудка колебалась от 61,4% до 71,1 % при средней степени обсеменения слизистой - (++).

**Ключевые слова:** хронический неатрофический гастрит, ДЖВП по гипотоническому гипокинетическому типу, сохранённая функция жёлчного пузыря.

**Introduction.** The development of chronic *Helicobacter pylori*, which under certain circumstances turns into a destructive form, called peptic ulcer disease, is influenced by various factors [4]. All factors affecting the development of chronic non-atrophic gastritis are characterized by one common property — the effect on the active form of *Helicobacter pylori* infection (HP): both for the factors that kill this form (antibiotics, bismuth preparations) and for the factors that convert the active form of the bacterium into inactive (cocco-shaped) form (proton pump inhibitors) [7, 8, 9, 10, 11, 12, 14, 15, 16, 17]. One of the factors affecting the active form of HP infection is bile reflux [1, 13], but in the available literature there are no data on the effect on this process of this form of biliary tract dyskinesia (BTD), as hypotensive hypokinetic type, and was the reason for the study of this issue.

**Purpose of the study.** To study the frequency of detection of the active form of *Helicobacter pylori* infection in patients with chronic non-atrophic gastritis with biliary dyskinesia by hypotonic hypokinetic type and in patients with chronic non-atrophic gastritis with preserved function of the gall bladder.

**Materials and research methods.** On the basis of the clinical department of the problem laboratory for chronic *Helicobacter pylori* at the Black Sea Petro Mohyla National University and the functional diagnostics of the 4th hospital in the city of Nikolayev, 140 patients with chronic non-atrophic gastritis were comprehensively examined. The first group consisted of 70 patients with BTD on the hypotonic hypokinetic type, the second - 70 patients with the preserved function of the gallbladder. The age of patients ranged from 18 to 72 years old (average age was  $39.6 \pm 0.9$  years). There were 54 men (38.6%), 86 women (61.4%).

The study was carried out in compliance with the basic bioethical provisions of the Council of Europe Convention on Human Rights and Biomedicine (dated 04.04.1997), the Helsinki Declaration of the World Medical Association on the Ethical Principles of Scientific 549 Medical Research with Human Participation (1964-2008), and the MOH Order Of Ukraine No. 690 of September 23, 2009.

Comprehensive examination included: step-by-step enteric pH - metry on VN Chernobrovyi methodology, esophagogastroduodenoscopy (EGDS) with generally accepted method, double HP's testing: test for urease activity and microscopy of stained by Giemsa smears, material for which was taken during endoscopy of 4 topographical zones: from the middle third of the gastric antrum and body division on the big and small curvature with our developed methodology, which allows you to define and the presence of intracellular "Depot" of HP infection (in the presence of a tumor - departing 1 cm from the edge of the tumor) as well as histological studies of the gastric mucosa, the material for which is taken from the same zone and from the edges of the cancer, using a generally accepted method taking into account recent classifications [2, 6]. To calculate the average level of acidity, we used conventional units (CU) [3].

The sequence of the examination: after collecting the anamnesis, the patients were subjected to pH-metry, and then - endoscopy with biopsy sampling for testing HP and histological studies of the gastric mucosa. The study was conducted in the morning, on an empty stomach, 12-14 hours after the last meal. After a comprehensive examination, the patients underwent an ultrasound scan with a food load according to the standard technique [5]. The results of ultrasound became the basis for dividing patients into groups. The obtained data were processed statistically using t-student test with the calculation of average values

(M) and the estimated probability of deviations (m). Changes were considered statistically significant at  $p < 0.05$ . Statistical calculations were performed using Excel spreadsheets for Microsoft Office.

**Research results and discussion.** The data obtained when conducting pH-metry, are shown in table 1.

Table 1.

**Acidity level in patients with chronic non-atrophic gastritis with BTD on hypotonic hypokinetic type and with preserved gallbladder function**

The level of acidity	Frequency of identified different levels of acidity in patients with chronic non-atrophic gastritis with BTD according to hypotonic hypokinetic type and with preserved function of the gall bladder			
	1st group		2nd group	
	Number of patients (n = 70)	%	Number of patients (n = 70)	%
Hyperacidity expressed	8	11,4	11	15,7
Hyperacidity moderate	6	8,6	5	7,1
Normacidity	25	35,7	22	31,4
Hypoacidity moderate	14	20	9	12,9
Hypoacidity expressed	16	22,9	23	32,9
Anacidity	1	1,4	0	0

**Note:** n-the number of studies

In a comparative analysis of the level of acidity in groups in the 1st group, the acidity was  $11.1 \pm 0.63$  CU, in the 2nd -  $11.3 \pm 0.58$  CU, which corresponded to the basal normacidity minimal in both groups.

When conducting EGDS in patients of the 1st group, an active ulcerative process was detected in 2 (2.9%) patients in the duodenum, in 8 (11.4%) patients there were manifestations of past ulcers of the duodenal bulb in as a scar deformity of varying severity. In 9 patients (12.9%), the presence of bile was detected in the stomach cavity.

When conducting EGD in patients of the 2nd group in 4 (5.7%) patients an active ulcerative process was detected in the duodenum, and in 2 (2.9%) patients an active ulcerative process was detected in the stomach; erosive-ulcerative bulbit was detected in 3 (4.3%) patients; in 12 (17.1%) patients there were manifestations of duodenal ulcer ulcers transferred in the past in the form of cicatricial deformity of different severity. In 5 patients (7.1%), the presence of bile was detected in the cavity of the stomach.

When analyzing the data of histological studies in all patients in 100% of cases, the presence of chronic non-atrophic gastritis was confirmed in both the active and inactive stages of varying severity.

When testing for HP, the identification of the active form of Helicobacter pylori infection was different in different groups. The data on the identification and degree of seeding of the gastric mucosa by HP infection by the topographic zones of the stomach are presented in tables 2, 3.

Table 2.

**The frequency of detection and the degree of dissemination of the gastric mucosa by the active form of HP - infection by topographically zones in patients with chronic non-atrophic gastritis with BTD with hypotonic hypokinetic type (n = 70)**

<b>Topographic zones of the stomach</b>	<b>Detection frequency, %</b>	<b>The degree of contamination of the gastric mucosa by HP -infection by topographic zones stomach (+) / (M±M)</b>
1. Antrum, middle one-third, greater curvature	21 (30%)	0,67 ± 0,08
2. Antrum, middle third, small curvature	20 (28,6%)	0,67 ± 0,08
3. Body of the stomach, middle third, greater curvature	17 (24,3%)	0,63 ± 0,10
4. Body of the stomach, middle third, small curvature	25 (35,7%)	0,84 ± 0,10

**Note:** n-the number of studies

Table 3.

**Detection rate and seeding rate of the gastric mucosa by the active form of HP- infection by topographic zones in patients with chronic non-atrophic gastritis with preserved gallbladder function (n = 70)**

<b>Topographic zones of the stomach</b>	<b>Detection frequency, %</b>	<b>The degree of contamination of the gastric mucosa by HP -infection by topographic zones stomach (+) / (M±M)</b>
1. Antrum, middle one-third, greater curvature	44 (62,9%)	1,67 ± 0,10
2. Antrum, middle third, small curvature	43 (61,4%)	1,53 ± 0,10
3. Body of the stomach, middle third, greater curvature	48 (68,6%)	1,81 ± 0,10.
4. Body of the stomach, middle third, small curvature	54 (77,1%)	2,09 ± 0,10

**Note:** n-the number of studies

A comparative analysis of data on the average degree of dissemination of the active form of HP-infection of the gastric mucosa by topographic zones in the examined patients of the 2nd group, the degree of dissemination was significantly higher in all zones than in patients of the 1st group ( $p < 0.05$ ). While in a comparative analysis of data on the average degree of dissemination of the active form of HP infection of the gastric mucosa by topographic zones within the 2nd group itself, a significantly higher degree of dissemination was on the mucosa in the body of the stomach along the lesser curvature ( $p < 0.05$ ).

These results are explainable from the point of view of the effects of bile (especially deoxycholic and chenodeoxycholic acids) and pancreatic juice, which get into the stomach with duodeno-gastric reflux, on the active form of HP infection [1, 13]. When the contractile function of the gallbladder is impaired as hypotonia, bile and pancreatic juice enter the duodenum with a delay, which leads to a violation of antroduodenal coordination and the formation of duodenal-gastric reflux [1, 4]. In this case, the HP infection passes into an inactive (cocci-shaped) form, which, unlike the active form, cannot be fixed on the mucous membranes and is quickly washed off during the meal and due to the peristaltic wave in the intestine, and especially from the mucous membrane of the antrum. smoother relief. In the body of the stomach, where the mucous due to folding is more prominent, inactive forms are less prone to the washing process, and the active forms are more protected from the effects of reflux, especially along the lesser curvature, where contact with bile and pancreatic juice is the least [14]. A decrease in the concentration of active forms on the gastric mucosa leads to a decrease in the concentration of residual ammonia (RA) in the cavity of the stomach and reduces the likelihood of the process of ulceration not only in the duodenum, but also in the stomach [4], which is confirmed by our research.

#### **Conclusions and prospects for further research.**

1. Biliary dyskinesia of the hypotonic hypokinetic type more often than when the function of the gallbladder is preserved, accompanied by duodeno-gastric reflux, which adversely affects the active form of HP infection.

2. Reducing the level of contamination of the gastric mucosa with active forms of HP due to a decrease in residual ammonia reduces the risk of ulceration not only in the duodenum, but also in the stomach.

The prospect of further research is the study of the degree of dissemination by active forms of NR infection of the gastric mucosa in patients with chronic non-atrophic gastritis with BTD with hypertonic hyperkinetic type.

## References

1. Avramenko AA. Vliyanie duodenogastralnogo refluksa na chastotu vyyavleniya aktivnih form helikobakternoj infekcii na slizistoj raznyh zon zheludka u bolnyh hronicheskim neatroficheskim gastritom. *Klinichna ta eksperimentalna patologiya*. 2015; 1 (51): 18 - 21. [Russian].
2. Avramenko AA. Dostovernost stul-testa pri testirovanii bolnykh khronicheskim khelikobakteriozom pri nalichii aktivnykh i neaktivnykh form khelikobakternoy infektsii na slizistoy obolochke zheludka. *Suchasna gastroenterologiya*. 2014; 3 (77): 22–6. [Russian].
3. Avramenko AA, Gozhenko AI, editors. *Helikobakterioz*. Nikolaev: «X-press poligrafiya», 2007. 336 s. [Russian].
4. Avramenko AA, Gozhenko AI, Goydyk VS, editors. *Yazvennaya bolezнь (ocherki klinicheskoy patofiziologii)*. Odessa : OOO «RA «ART-V», 2008. 304 s. [Russian].
5. Dergachyov AI, Kotlyarov PM, editors. *Abdominalnaya ehografiya: spravochnik*. M.: EliksKom, 2005. 352 s., il. [Russian].
6. Kímakovich VY, Nikishaev VI, editors. *Yendoskopiya travnogo kanalu. Norma, patologiya, suchasni klasifikatsiyi*. Lviv: Vidavniststvo Meditsina Svítu, 2008. 208 s., il. 4. [Ukrainian].
7. Maev IV, Kucheryavyj YuA, Andreev DN. Prichiny neeffektivnosti antigelikobakternoj terapii. *RZhGGK*. 2013; 6: 62- 72. [Russian].
8. Maev IV, Samsonov AA, Andreev DN. Evolyuciya predstavlenij o diagnostike i lechenii infekcii *Helicobacter pylori* (po materialam konsensusa Maastricht I V, Florenciya, 2010). *Vestnik prakticheskogo vracha*. 2012; 1: 19 – 26. [Russian].
9. Nikiforova YaV, Tolstova TN, Cherelyuk NI. Osnovnye polozheniya Soglasitelnoj konferencii po diagnostike i lecheniyu *Helicobacter pylori* - Maastricht V (2015). *Suchasna gastroenterologiya*. 2016; 6(92): 119 – 133. [Russian].
10. Fadeenko GD, Nikiforova YaV. Vliyanie kolloidnogo subcitrata vismuta na etiopatogenez hronicheskogo gastrita: novyj vitok izucheniya davnej problemy. *Suchasna gastroenterologiya*. 2015; 6 (86): 74-81. [Russian].
11. Harchenko NV, Tkach SM, editors. *Gastroenterologiya v voprosah i otvetah*. K: OOO «Doktor-Media-Grupp», 2016. 36 s. [Russian].
12. Cimmerman YaS. Problema rastushej rezistentnosti mikroorganizmov k antibakterialnoj terapii i perspektivy eradikacii *Helicobacter pylori*. *Klinicheskaya medicina*. 2013; 6: 14-20. [Russian].



13. Cyrkunov AV, Tarasov VV, Savickij SE. Rol bakterij roda *Helicobacter* pri zbolevaniyah pecheni i zhelchevyvodyashih putej (obzor literatury). *Zhurnal GGMU*. 2005; 1: 13-20. [Russian].
14. Avramenko AO. The effect of proton pump inhibitors on formation of inactive (coccoid) forms of *H. pylori* infection. *Clinical Pharmacy*. 2013; 4: 15 – 17.
15. Dore MP, Leandro G, Realdi G. Effect of pretreatment antibiotic resistance to metronidazole and clarithromycin on outcome of *Helicobacter pylori* therapy. A meta-analytical approach. *Dig.Dis.Sci*. 2000; 45: 68 – 76.
16. Malfertheiner P., Megraud F., O'Morain C.A., Gisbert J.P., Kuipers E.J., Axon A. T., Bazzoli F., Gasbarrini A., Atherton J., Graham D.Y., Hunt R., Moayyedi P., Rokkas T., Rugge M., Selgrad M., Suerbaum S., Sugano K., El-Omar E.M. Management of *Helicobacter pylori* infection – the Maastricht V / Florence Consensus Report. *Gut*. 2016/doi:10.1136/gutjnl-2016-312288.
17. Peitz U, Sulliga M, Wolle K. High rate of posttherapeutic resistance after failure of macrolide – nitroimidazole triple therapy to cureline therapies in a randomized study. *Aliment. Pharmacol. Ther*. 2002; 16: 315 – 322.