УДК 616.33-002.27-074-085.281.9

COLONIZATION OF HELICOBACTER PYLORI IN GASTRIC MUCOSA AT CHRONIC ATROPHIC GASTRITIS

M. Kornyenko, O. Kalenska, O. Kuryk

Summary. *There were analyzed the extent of colonization of Helicobacter pylori in gastric mucosa at chronic atrophic gastritis.The higher extent of colonization of H. pylori it is noted at chronic atrophy gastritis with metaplasia that confirms a bacterium role in emergence of metaplastic changes which potential dysplasia and potential risk of a malignancy.*

Keywords: *chronic atrophic gastritis, Helicobacter pylori, intestinal metaplasia.*

Background. The chronic atrophic gastritis is one of the most important problem of modern gastroenterology as a precancer conditions according to recommendations of WHO [3]. The main aetiology factor of chronic gastritis is Helicobacter pylori (H. pylori) [2]. The main features of H. pylori gastritis are damage of epitelial cells, inflammatory infiltrate to own plate of a mucous membrane and presence of limphoid follicles [4]. The atrophy is defined as reduction of volume of glands or loss by glands of their high-grade function at the expense of a disregeneration when specialized epitelium of gastric glands it is replaced not peculiar to the intestinal epitelium (an intestinal metaplasia) [1]. There are two main atrophy forms – without metaplasia (a true atrophy) and with metaplasia among which allocate full and incomplete metaplasia. The full (small intestine) metaplasia is characterized the existence of all peculiar cells of a small intestine; the most important sign to this metaplasia is existence of Panet cells. The incomplete (colon) metaplasia is characterized by colon cells; Panet cells don't occur. Existence of an intestinal metaplasia may be connect with H. pylori [5]. Therefore definition of extent of colonization of H. pylori in mucosa of stomach at chronic atrophy gastritis with metaplasia and without metaplasia became the purpose of our research.

Material and methods. We investigated 90 cases of gastric biopsy at patients with chronic atrophy gastritis with a true atrophy of gastric mucosa and 120 cases with a metaplastic atrophy. Slices of a mucosa from big, small curvature and a corner of a stomach fixed in 10 % solution of neutral formalin, carried out by the standard techniques, paraffin cuts in thickness of 3–5 microns painted by gematoksilinum-eozinum and by Gimza for identification of H. pylori. For a quantitative assessment of extent of colonization of H. pylori we used the scheme offered by L.I.Aruin: weak degree – to 20 bacteria in some fields of microscope; from 20 to 50 – average degree, and is more than 50 bacteria – high extent of colonization. Histologic preparations investigated under Axioscop 40 (Zeiss) microscope at increase 200.

Results and discussion. At chronic atrophic gastritis without metaplasia (Fig. 1) from 90 studied cases in 48 (53,3 %) were noted existence of limphoid follicles (Fig. 2) in stomach mucosa. In 36 (40 %) cases gastritis was active (1degree activity of inflammatory process (Fig. 3). Bacteria weren't found in 6 cases (6,7 %); in 48 cases (53,3 %)we found weak extent of colonization, in 27 (30 %) – average extent (Fig. 4), and only in 9 (10 %) – high extent of colonization of H. pylori was noted. In group of chronic gastritis with a metaplastic atrophy from 120 gastric biopsy in 64 (53,3 %) cases the full (small intestine) metaplasia was founded (Fig. 5), in 40 cases (33,3 %) – incomplete (colon) metaplasia was noted (Fig. 6), and in 16 cases (13,3 %) the combination of a full and incomplete intestinal metaplasia was noted. In 56 cases (46,7 %) the chronic atrophic metaplastic gastritis was active. The limphoid follicles in mucosa strom were observed in 36 (30 %) cases. In 12 cases (10 %) metaplastic chronic gastritis of H. pylori weren't found, in 40 (33,3 %) cases weak extent of colonization was found, in 44 (36,7 %) average extent and in 24 (20 %) – high extent of colonization of H. pylori was diagnosed.

Conclusions. The higher degree of colonization of H. pylori is noted in chronic atrophy gastritis with intestinal metaplasia. This data confirms the role of bacteria in causing metaplastic changes and the possible occurrence of dysplasia with risk of malignancy.

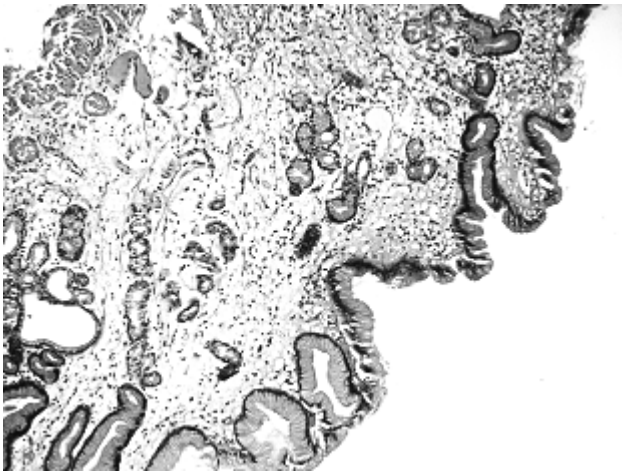

Fig 1. **Chronic atrophic gastritis without metaplasia. Hematoxylin and eosin staining × 100**

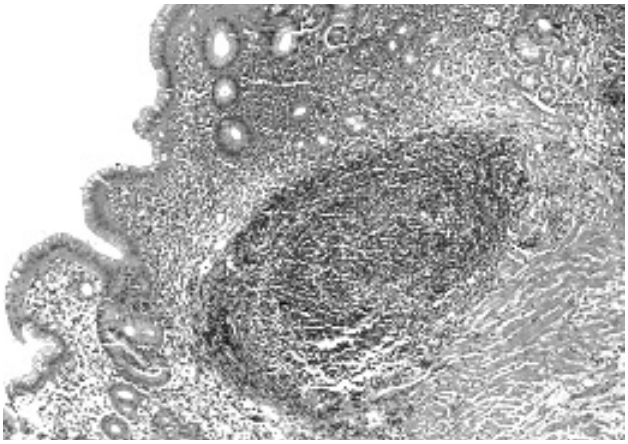

Fig 2. **Chronic atrophic gastritis with limphoid follicles in stomach mucosa. Hematoxylin and eosin staining × 100**

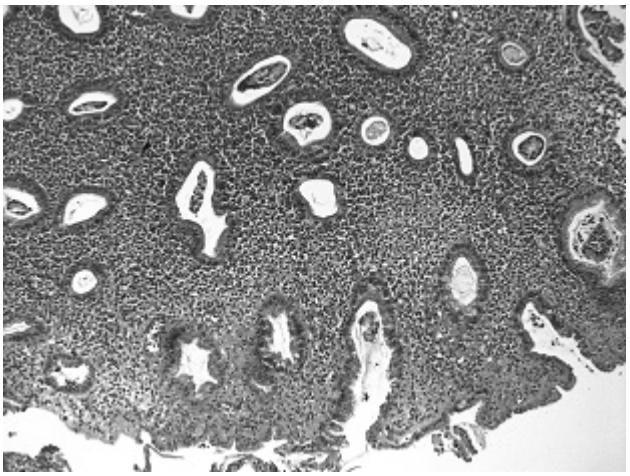

Fig. 3. **Chronic atrophic gastritis with 1 degree activity of inflammatory process. Hematoxylin and eosin staining × 100**

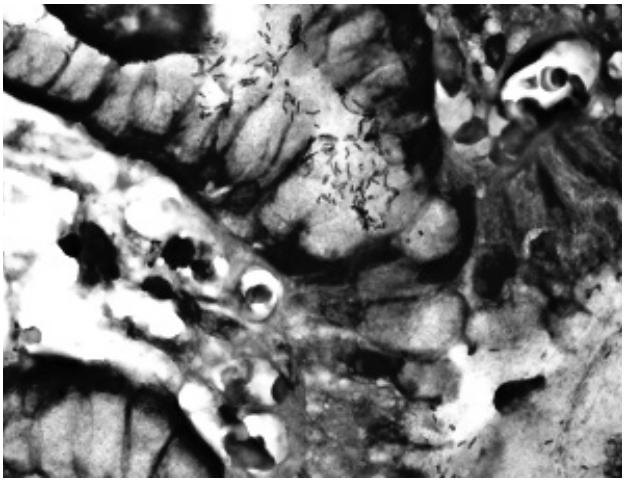

Fig. 4. **Chronic atrophic gastritis with average extent colonization of H. Pylori. Staining by Hymza × 400**

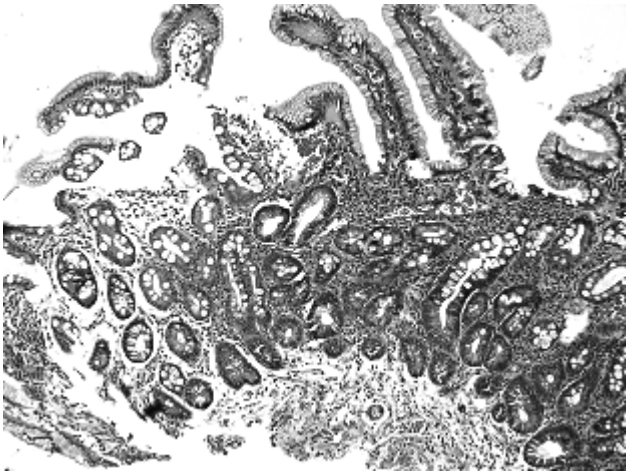

Fig. 5. **Chronic atrophic gastritis with full (small intestine) metaplasia. Hematoxylin and eosin staining × 100**

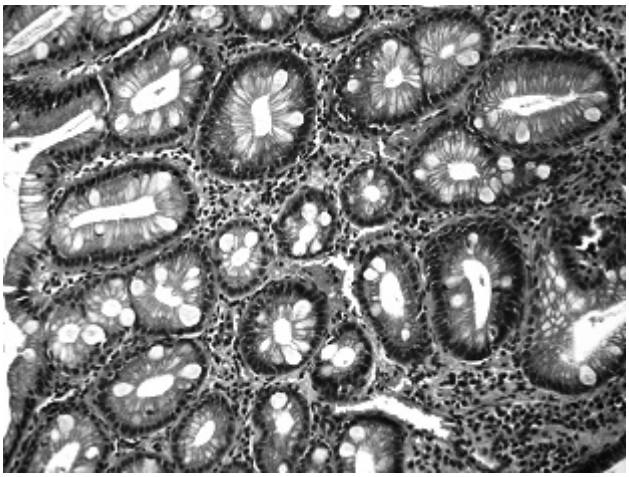

Fig. 6. **Chronic atrophic gastritis with incomplete (colon) metaplasia. Hematoxylin and eosin staining × 200**

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КОЛОНІЗАЦІЯ HELICOBACTER PYLORI В СЛИЗОВІЙ ОБОЛОНЦІ ШЛУНКА ПРИ ХРОНІЧНОМУ АТРОФІЧНОМУ ГАСТРИТІ

М. А. Корнієнко, О. В. Каленська, О. Г. Курик

Резюме. *Проаналізовано ступінь колонізації Helicobacter pylori в слизовій оболонці шлунка при хронічному атрофічному гастриті. Більш високий ступінь колонізації H. pylori відзначається при ХАГ з метаплазією, що підтверджує роль бактерії у виникненні метапластичних змін, на фоні яких може виникати дисплазія з потенційним ризиком малігнізації.*

Ключові слова: *хронічний атрофічний гастрит, Helicobacter pylori, кишкова метаплазія.*

КОЛОНИЗАЦИЯ HELICOBACTER PYLORI В СЛИЗИСТОЙ ОБОЛОЧКЕ ЖЕЛУДКА ПРИ ХРОНИЧЕСКОМ АТРОФИЧЕСКОМ ГАСТРИТЕ

М. А. Корниенко, О. В Каленская, Е. Г. Курик

Резюме. *Проанализирована степень колонизации Helicobacter pylori в слизистой оболочке желудка при хроническом атрофическом гастрите. Более высокая степень колонизации H. pylori отмечается при хроническом атрофическом гастрите с метаплазией, что подтверждает роль бактерии в возникновении метапластических изменений, на фоне которых может возникать дисплазия с потенциальным риском малигнизации.*

Ключевые слова: *хронический атрофический гастрит, Helicobacter pylori, кишечная метаплазия.*