УДК [61+615.1] (06)
ББК 5+52.81
А 43
ISBN 978-985-21-1865-1

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СРАВНИТЕЛЬНЫЙ ГИСТОПАТОЛОГИЧЕСКИЙ АНАЛИЗ ХЕЛИКОБАКТЕРНОГО И НЕХЕЛИКОБАКТЕРНОГО БАКТЕРИАЛЬНОГО ГАСТРИТА

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A COMPARATIVE HISTOPATHOLOGICAL ANALYSIS OF H. PYLORI AND NON-H. PYLORI INDUCED BACTERIAL GASTRITIS

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Резюме. В этом исследовании сравниваются гистопатологические особенности гастрита, вызванного H. pylori (Hp) и не-Hp бактериями в 456 биопсиях. Инфекция Hp (25% случаев) вызывала сильное воспаление, атрофию и метаплазию, особенно у пожилых мужчин. Бактерии, не являющиеся Hp (90% случаев), приводили к более легким изменениям, таким как фовеолярная гиперплазия и образование лимфоидных фолликулов. Результаты демонстрируют различные патологические закономерности между хеликобактерным и нехеликобактерным бактериальным гастритом.

Ключевые слова: Helicobacter pylori, нехеликобактерный бактериальный гастрит.

Resume. This study compares histopathological features of H. pylori (Hp) and non-Hp gastritis in 456 biopsies. Hp infection (25% cases) caused severe inflammation, atrophy, and metaplasia, especially in older males. Non-Hp bacteria (90% cases) led to milder changes like foveolar hyperplasia and lymphoid follicles. Some Hp-negative cases showed autoimmune features. Results demonstrate distinct pathological patterns between Hp and non-Hp gastritis.

Keywords: Helicobacter pylori, non-H. pylori bacteria gastritis.

Relevance. Gastritis remains a globally significant condition with potential progression to severe complications, including gastric cancer. While H. pylori is well-established as the primary etiological agent, emerging evidence suggests that other bacterial species may independently contribute to gastric inflammation. This study addresses critical gaps in understanding the histopathological spectrum of non-Hp bacterial gastritis, which has been understudied despite its clinical prevalence. By systematically comparing mucosal changes between Hp and non-Hp cases, we provide insights into their distinct pathogenic mechanisms. Furthermore, the identification of autoimmune-like features in Hp-negative cases challenges the traditional dichotomy of infectious versus autoimmune gastritis, suggesting a more complex interplay of microbial and host factors. These findings have direct implications for diagnostic accuracy, therapeutic strategies, and the need for expanded microbiological testing in gastritis patients.

Aim: to evaluate the frequency and severity of gastric mucosal damage caused by non-Hp bacteria and compare it with Hp-induced changes.

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Objectives:

1. Assess histopathological patterns in Hp-positive and Hp-negative gastritis.
2. Identify reactive changes associated with non-Hp bacterial colonization
3. Investigate potential autoimmune mechanisms in Hp-negative gastritis.

Material and methods. The study included 456 gastric biopsy specimens collected from the antrum (76.3%), body (17.2%), fundus (14%), and cardiac region (51%) of patients aged 19–94 years in Minsk (January 2024–April 2025). Specimens underwent morphological examination, and data were analyzed using descriptive and inferential statistics in Microsoft Excel.

Results and their discussion.

1. H. pylori-Associated Gastritis

Hp infection was identified in 25% of cases and demonstrated a strong correlation with aggressive histopathological changes:

- Severe Inflammation: Prominent lymphoid infiltration (fig. 1) and neutrophilic activity were hallmarks, reflecting robust immune activation against the bacterium.
- Structural Damage: Glandular atrophy and intestinal metaplasia were frequent, particularly in older males, aligning with Hp's known role in precancerous transformations.
- Demographic Trends: The male predominance in severe Hp cases (31% of the cohort) suggests potential sex-based differences in susceptibility or immune response.

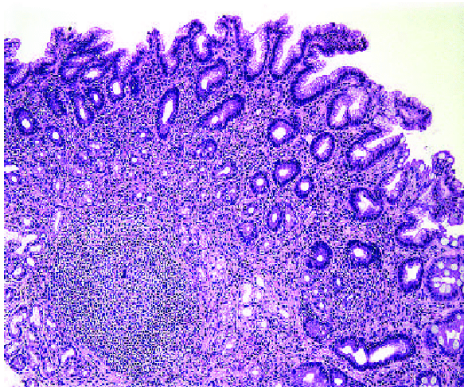

Fig. 1 – Lymphoid infiltration

These findings reinforce Hp's status as a high-risk pathogen for chronic gastritis and gastric carcinogenesis.

2. Non-H. pylori Bacterial Gastritis

Non-Hp bacteria, primarily coccal flora (e.g., Streptococcus anginosus, Enterococcus faecalis), were detected in ~90% of cases and associated with distinct reactive patterns:

- Mild but Chronic Changes: Foveolar hyperplasia (80%) and lymphoid follicles (50%) dominated, indicating low-grade, persistent mucosal irritation (fig. 3, 4).
- Limited Metaplasia: Unlike Hp, non-Hp bacteria rarely caused advanced metaplasia or atrophy, suggesting a less aggressive pathogenic profile.
- Erosions: Focal erosions (20%) were observed, likely due to localized microbial disruption of the epithelial barrier.

The high prevalence of coccal colonization underscores its potential role in gastritis pathogenesis, even in the absence of Hp.

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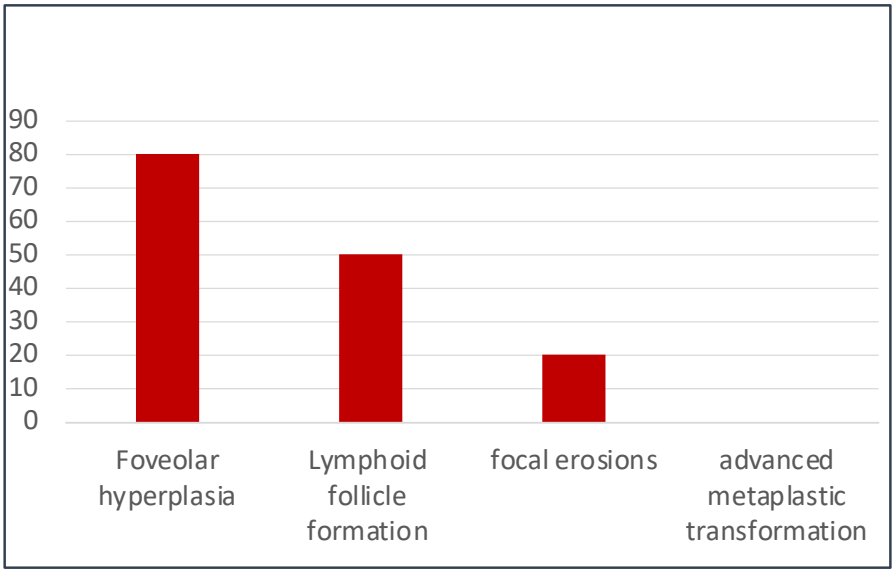

Fig. 2 – Non -H. pylori bacteria and gastric mucosal changes

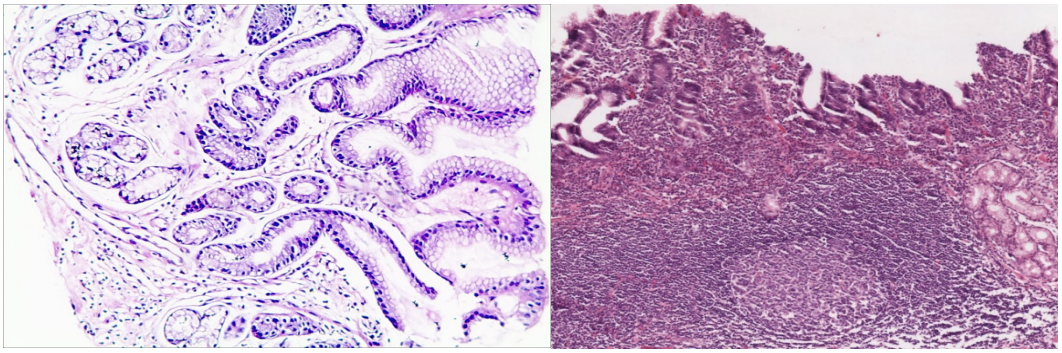

Fig. 3 – Foveolar Hyperplasia, Fig. 4 - Lymphoid follicles

3. Autoimmune Features in Hp-Negative Cases

A subset of Hp-negative specimens exhibited features atypical for infectious gastritis:

- Pyloric Metaplasia: Characterized by pseudo pyloric gland transformation and mucous neck cell proliferation, resembling autoimmune gastritis (fig. 5).
- Neuroendocrine Hyperplasia: Increased enterochromaffin-like (ECL) cells with linear/micronodular patterns, a potential precursor to neuroendocrine tumors (fig. 6).

These findings suggest that some cases of "idiopathic" gastritis may represent undiagnosed autoimmune disease, warranting serological testing (e.g., anti-parietal cell antibodies).

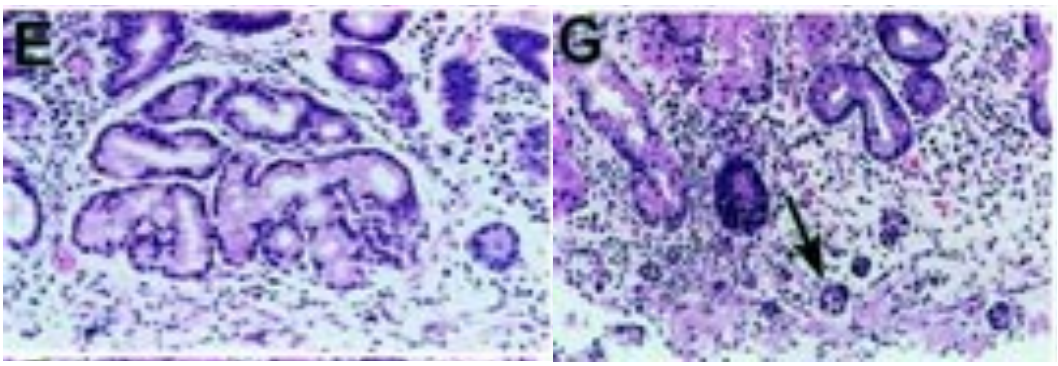

Fig. 5 – Pyloric metaplasia, Fig. 6 - Neuroendocrine Hyperplasia

Common Overlapping Features

Both Hp and non-Hp groups shared:

- Foveolar Hyperplasia: A nonspecific response to mucosal injury.
- Coccal Colonization: Present in both groups, raising questions about its role as a commensal or pathobiont.

Conclusion:

1. Divergent Pathogenic Roles: *H. pylori* drives severe, progressive gastritis with metaplastic and atrophic changes, while non-Hp bacteria (especially coccal flora) induce milder, reactive alterations. The latter may contribute to chronic mild inflammation, warranting clinical attention even in Hp-negative patients.
2. Autoimmune Gastritis Suspected: Pyloric metaplasia and neuroendocrine hyperplasia in Hp-negative cases highlight the need to consider autoimmune mechanisms, particularly in patients with unexplained mucosal damage.
3. Diagnostic Implications: Current diagnostic protocols overly focus on Hp, potentially overlooking non-Hp bacteria and autoimmune etiologies. Expanded testing (e.g., microbial sequencing, serology) could improve diagnostic accuracy.
4. Therapeutic Considerations: Non-Hp gastritis may require alternative management strategies, such as targeted antibiotics or immunomodulatory therapies, depending on the underlying cause.
5. Research Directions: Future studies should:
   - Identify non-Hp species via metagenomics.
   - Clarify the role of coccal flora as commensals or pathogens.
   - Investigate autoimmune markers in Hp-negative gastritis.

This study redefines gastritis as a spectrum of microbial and autoimmune insults, urging a paradigm shift in its clinical evaluation and management.

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