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A COMPARATIVE HISTOPATHOLOGICAL ANALYSIS OF HELICOBACTER PYLORI AND NON-HELICOBACTER PYLORI-INDUCED GASTRITIS

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Introduction. Gastritis refers to inflammation of the gastric mucosa, frequently associated with Helicobacter pylori (Hp) infection. This bacterium is the leading microbial cause of chronic gastritis and related gastroduodenal disorders. However, Hp is not the only microorganism capable of triggering gastric inflammation. Other bacteria have been known to disrupt the gastric epithelial balance leading to gastritis which includes Fusobacterium nucleatum, Helicobacter heilmannii, Streptococcus anginosus, Enterococcus faecalis, Escherichia coli and Klebsiella pneumoniae.

Objective: we aim to identify how often non-Hp bacteria damage the gastric mucosa and compare these effects to Hp's known pathogenic mechanisms, helping clarify their distinct pathological roles in gastritis development.

Materials and methods. Our research materials were 456 gastric biopsy specimens collected from hospitals across Minsk from 01.01.2024 to 15.04.2025, primarily from the antrum and body, with occasional samples from the cardiac region of the stomach. Methods were morphological and statistical. Data processing was carried out in Excel.

Results and their discussion. The patient cohort spanned ages 19 to 94, with nearly equal gender distribution. This revealed distinct histopathological patterns associated with Hp infection compared to non-Hp bacterial flora (predominantly coccal bacteria). The cohort comprised predominantly middle-aged to elderly with a female predominance. Hp infection was detected in 25% of cases and correlated with severe inflammatory changes, including highly marked lymphoid infiltration, neutrophilic activity, atrophy, and intestinal metaplasia–particularly in older males. In contrast, non-Hp bacteria (noted in ~90% of cases as coccal flora) were associated with milder but prevalent reactive changes, such as foveolar hyperplasia (80%), lymphoid follicles (50%), and erosions, without the advanced metaplasia seen in most Hp cases. Cases with coccal flora but no Hp showed pyloric metaplasia or neuroendocrine hyperplasia, suggesting autoimmune gastritis.
Notably, both groups shared features like foveolar hyperplasia and coccal colonization, suggesting that non-Hp bacteria may contribute to low-grade mucosal injury independently.

Conclusion. These findings highlight the need to consider non-Hp bacterial influences in gastritis pathogenesis, particularly in Hp-negative cases with unexplained inflammation or reactive alterations. Further research should explore the microbial identity and mechanisms of these non-Hp bacteria in gastric mucosal damage.