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Management Approaches for Patients with Atrophic Gastritis in Outpatient Practice

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Annotation: Atrophic gastritis is a chronic pathological condition characterized by progressive loss of the gastric mucosal glands and decline of gastric secretory function. The eradication of Helicobacter pylori remains the fundamental part of treatment, since the bacterium acts as a major etiological factor. However, increasing antibiotic resistance reduces the efficacy of standard regimens. Rifaximin, a non-absorbed rifamycin derivative, demonstrates potential advantages including low resistance and high intraluminal concentration. This article analyzes the clinical use of rifaximin in outpatient management of atrophic gastritis.

Keywords: atrophic gastritis, Helicobacter pylori, rifaximin, eradication therapy, outpatient management.

Introduction. Atrophic gastritis is defined as a chronic inflammatory disease of the gastric mucosa leading to loss of glandular structures, reduced gastric secretion, and replacement with intestinal metaplasia and epithelial dysplasia [1]. These histopathological changes represent a precancerous condition according to the OLGA/OLGIM classifications. Helicobacter pylori (H. pylori) is the main etiological factor in secondary atrophic gastritis. The microorganism adheres to gastric epithelial cells via adhesins (BabA, SabA, OipA) [2]. Virulence factors CagA and VacA stimulate inflammation and oxidative stress, triggering gland atrophy [3].

H. pylori uses urease to convert urea to ammonia and CO_2, enabling survival in the acidic gastric lumen [4]. Growing antibiotic resistance reduces the success rate of standard triple and quadruple eradication therapy [5]. Therefore, new therapeutic strategies are necessary. Rifaximin is a non-absorbed rifamycin derivative. It inhibits DNA-dependent RNA polymerase, suppressing bacterial replication. Due to absence of systemic absorption and high intraluminal concentration, rifaximin rarely induces resistance [6–7].

Materials and methods. A prospective observational study included 36 patients (21 females, 15 males) with confirmed atrophic gastritis. Diagnosis was established by gastropanel (pepsinogen I/II, gastrin-17), esophagogastroduodenoscopy with biopsy, and urease test. Complete blood count was performed. Median values (Me, Q1–Q3) were calculated. Statistical significance was assessed using Spearman correlation ($p < 0.05$).

Patients were divided into two groups:

- ✓ Standard therapy: clarithromycin + amoxicillin + PPI
- ✓ Modified therapy: rifaximin + clarithromycin + PPI + probiotics

Results. The mean age of females was 44 ± 3.5 years, males — 35 ± 2.5 years. Mean disease duration was 7 years. A decrease in hemoglobin and erythrocytes was found. Patients receiving rifaximin showed normalization of hemoglobin and erythrocytes, reduction of atrophy signs, and higher eradication success.

Correlation analysis:

- ✓ degree of atrophy vs. presence of H. pylori — $rs = 0.392$
- ✓ rifaximin vs. reduction of exacerbations — $rs = 0.561$ ($p < 0.05$)
- ✓ rifaximin vs. hemoglobin improvement — $rs = 0.526$ ($p < 0.05$)

Within 6 months of follow-up, recurrence was observed only in 12% of patients treated with rifaximin, which is more than four times lower compared with standard therapy.

Discussion. Rifaximin demonstrated significant benefit in eradication therapy. Due to minimal absorption and low resistance rates, rifaximin is considered a prospective option for outpatient management of H. pylori–associated atrophic gastritis. Probiotics enhance therapeutic effect by reducing inflammation and improving microbiota balance [7–9].

Conclusion. Rifaximin-based eradication therapy showed higher clinical effectiveness compared with standard triple therapy in patients with H. pylori–associated atrophic gastritis. Rifaximin may be considered as a therapeutic alternative, particularly when resistance to clarithromycin is suspected.

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