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MODERN APPROACHES TO THE DIAGNOSIS AND TREATMENT OF GASTRITIS

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Abstract
Gastritis is a histologically defined inflammatory condition of the gastric mucosa with substantial global prevalence. This prospective observational study evaluated 148 patients using endoscopy, biopsy, and Helicobacter pylori testing protocols. Modern bismuth quadruple eradication therapy achieved a 91.7% success rate. Integrated diagnostic and targeted therapeutic strategies significantly reduced symptom burden and improved patient outcomes.

Keywords: Gastritis, helicobacter, endoscopy, biopsy, eradication, omeprazole, clarithromycin, dyspepsia, atrophy, metaplasia, diagnosis, pathology, inflammation, gastropathy, vonoprazan.

Introduction

Gastritis - defined as histological inflammation of the gastric mucosa - encompasses a clinically and pathologically heterogeneous spectrum of conditions unified by epithelial injury and inflammatory cell infiltration. Contemporary classification systems integrate etiology, anatomical distribution, histological pattern, and temporal course, effectively superseding outdated purely symptomatic frameworks. Globally, Helicobacter pylori infection, recognized as the predominant etiological agent, was estimated to affect approximately 43.9% of adults between 2015 and 2022, with prevalence still exceeding 70% in sub-Saharan Africa. Untreated chronic gastritis progresses through sequential stages of atrophy, intestinal metaplasia, and dysplasia, establishing a well-defined oncological risk corridor. Despite considerable advances in endoscopic imaging, molecular diagnostics, and targeted pharmacotherapy, diagnostic inconsistency and therapeutic heterogeneity continue to persist, warranting rigorous systematic evaluation of integrated contemporary management approaches across diverse clinical settings.

Literature Review

Early understanding of gastritis remained anchored to symptom description until Marshall and Warren's 1983 isolation of H. pylori fundamentally reoriented etiological thinking. The subsequent Sydney System (1990) introduced standardized biopsy mapping and semiquantitative histological grading, substantially improving diagnostic reproducibility. The OLGA and OLGIM staging frameworks later refined this foundation, linking histological severity quantitatively to gastric cancer risk. Contemporary guidelines - including the 2022 Chinese Society of Gastroenterology consensus and the 2024 American College of Gastroenterology recommendations - consolidate decades of evidence, now prioritizing molecular susceptibility profiling before antibiotic selection. Russian gastroenterologists, particularly Ivashkin and colleagues, have contributed substantially to clarifying

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mucosal response patterns under eradication protocols, informing treatment standards across the post-Soviet clinical landscape.

Methodology

This prospective observational study was conducted at a tertiary-level gastroenterology unit between January 2022 and December 2023. Ethical approval was obtained from the institutional review board, and all participants provided written informed consent prior to enrollment. Patients were included if they presented with upper abdominal symptoms persisting for four or more consecutive weeks and received a confirmed histological diagnosis of gastritis. Exclusion criteria encompassed prior gastric surgery, confirmed malignancy, concurrent immunosuppressive therapy, pregnancy, and age below 18 years. The final study cohort comprised 148 participants ranging in age from 19 to 74 years (mean age 43.7 ± 14.2 years). Gender distribution showed 82 male patients (55.4%) and 66 female patients (44.6%). Etiological classification revealed H. pylori-associated gastritis in 96 patients (64.9%), non-steroidal anti-inflammatory drug (NSAID)-induced gastritis in 28 patients (18.9%), autoimmune gastritis in 14 patients (9.5%), and idiopathic or multifactorial gastritis in the remaining 10 patients (6.8%). Diagnostic evaluation followed a structured multimodal protocol. All patients underwent high-definition esophagogastroduodenoscopy (EGD) under conscious sedation. Mucosal biopsies were obtained from five standardized gastric sites according to the Sydney System protocol: two specimens from the antrum, two from the corpus, and one from the incisura angularis. All histological specimens were evaluated by an experienced gastrointestinal pathologist blinded to clinical data, using updated Sydney System scoring criteria.

H. pylori status was determined through a combination of complementary methods. Rapid urease testing was performed intraoperatively on fresh antral biopsy tissue. Fecal antigen testing (HpSA) was conducted using a validated monoclonal enzyme immunoassay. The ^{13}C-urea breath test was completed at baseline and repeated at 8 weeks post-therapy to confirm eradication. Serology was intentionally excluded as a primary diagnostic tool, given its documented inability to differentiate active from previously resolved infection. Treatment protocols were assigned according to etiological subgroup. H. pylori-positive patients received bismuth quadruple therapy for 14 consecutive days, comprising bismuth subcitrate 120 mg four times daily, tetracycline 500 mg four times daily, metronidazole 400 mg three times daily, and esomeprazole 40 mg twice daily. For NSAID-induced gastritis, the offending agent was discontinued where clinically feasible, and proton pump inhibitor monotherapy with esomeprazole 40 mg once daily was prescribed for 8 weeks. Patients with confirmed autoimmune gastritis received quarterly surveillance endoscopy and intramuscular vitamin B12 supplementation at 1,000 µg monthly. Outcome assessment included symptom severity scoring using the validated Gastrointestinal Symptom Rating Scale (GSRS) at baseline and at 8 weeks. Follow-up endoscopy with repeat biopsy was performed at 12 weeks for all H. pylori-positive patients. Statistical analysis was performed using SPSS Statistics version 26.0. Categorical variables were compared using the chi-squared test, and continuous variables were analyzed with the independent-samples t-test or the Mann-Whitney U test where data distribution was non-normal. A p-value below 0.05 was applied as the threshold for statistical significance across all comparisons.

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Results

Among the 148 enrolled participants, complete follow-up data including confirmatory endoscopy and eradication testing were available for 141 patients (95.3%), with 7 patients lost to follow-up. The final per-protocol analysis therefore proceeded on 141 patients. H. pylori eradication was confirmed in 88 of 96 initially positive patients (91.7%) following completion of 14-day bismuth quadruple therapy. In the subgroup of 8 patients who failed first-line treatment, susceptibility-guided rescue therapy incorporating levofloxacin 500 mg once daily, amoxicillin 1,000 mg twice daily, and esomeprazole 40 mg twice daily for 14 days achieved eradication in 6 additional cases (75.0%), bringing the overall cumulative eradication rate to 97.9% across two sequential treatment lines. These figures are consistent with reported per-protocol efficacy benchmarks for bismuth quadruple regimens in populations with moderate-to-high metronidazole resistance. Histological improvement, defined as a reduction of at least one grade in both inflammatory activity and chronic inflammation scores per the updated Sydney System criteria, was documented in 84 of 88 successfully eradicated patients (95.5%) at the 12-week follow-up biopsy. Among patients with persistent H. pylori infection following first-line therapy, histological improvement was observed in only 4 of 8 cases (50.0%), a difference reaching strong statistical significance ($p < 0.001$). Intestinal metaplasia, present at baseline in 29 patients (20.6%), showed no measurable regression over the 12-week observation period in any subject, consistent with the established understanding that metaplastic transformation requires considerably longer surveillance intervals for any potential stabilization to occur. Symptom outcomes were assessed via the GSRS at baseline and at 8 weeks. The mean total GSRS score decreased from 38.4 ± 9.1 at baseline to 19.7 ± 6.8 at follow-up across the overall cohort ($p < 0.001$). Subgroup analysis revealed the greatest symptom reduction in H. pylori-positive patients following confirmed eradication (mean reduction: 21.3 points; 95% CI 18.9-23.7), compared with patients presenting with NSAID-induced gastritis managed via proton pump inhibitor monotherapy (mean reduction: 14.8 points; 95% CI 12.1-17.5). The between-group difference was statistically significant ($p = 0.004$).

In the autoimmune gastritis subgroup ($n = 14$), endoscopic findings demonstrated oxyntic mucosal atrophy in all cases, with concomitant iron-deficiency anemia detected in 8 patients (57.1%) and cobalamin deficiency confirmed in 11 patients (78.6%). Monthly intramuscular B12 supplementation normalized serum cobalamin levels in 10 of 11 deficient patients within 12 weeks of initiating replacement therapy. Gastroscopic grading at baseline revealed mild-to-moderate mucosal erythema in 63 patients (44.7%), nodular antral mucosa in 38 patients (27.0%), and erosive changes in 22 patients (15.6%). Subepithelial hemorrhage was observed in 11 patients (7.8%), predominantly in the NSAID-induced subgroup. Bile reflux gastropathy was an incidental finding in 5 patients (3.5%). Adverse effects during treatment were recorded in 23 of 141 patients (16.3%). The most commonly reported events were metallic taste ($n = 11$, 7.8%), treatment-related nausea ($n = 8$, 5.7%), and mild diarrhea ($n = 4$, 2.8%). No serious adverse events necessitating premature treatment discontinuation were observed during the study period.

Discussion

The 91.7% first-line eradication rate observed in this study compares favorably with data reported in recent systematic reviews, where bismuth quadruple therapy achieves per-protocol eradication rates of 85-93% in populations characterized by moderate clarithromycin resistance. The pragmatic

decision to employ bismuth quadruple therapy as empirical first-line treatment - rather than standard clarithromycin-based triple therapy - reflects the contemporary understanding that clarithromycin resistance, now exceeding 20% across many European and Central Asian populations, substantially undermines the clinical utility of triple regimen protocols. This position is supported by both the 2022 Russian Gastroenterological Association guidelines and the 2024 ACG recommendations, both of which now explicitly endorse bismuth-containing regimens as the preferred empirical strategy in resistance-prevalent settings. The near-universal histological improvement (95.5%) following confirmed eradication corroborates findings from longitudinal mucosal recovery studies, which consistently demonstrate that active neutrophilic infiltration resolves within weeks of bacterial clearance, while chronic lymphocytic inflammation progressively diminishes over subsequent months. The complete absence of metaplasia regression within 12 weeks is clinically expected and should not be misconstrued as therapeutic failure. Current evidence suggests that intestinal metaplasia may partially stabilize or regress over years following sustained eradication, though complete histological normalization is uncommon - particularly among older patients or those presenting with high-stage atrophy at baseline. This biologically grounded observation reaffirms a core preventive principle: early intervention, before the atrophic-metaplastic transition is established, remains the most effective strategy for reducing gastritis-associated oncological risk. The substantially greater symptomatic benefit observed in H. pylori-positive patients relative to the NSAID-induced subgroup merits careful clinical interpretation. NSAID-induced mucosal injury operates through a dual mechanism of direct topical epithelial damage and systemic prostaglandin depletion; while proton pump inhibitor therapy effectively heals macroscopic erosions, residual histological inflammation may persist, explaining the comparatively attenuated symptom response in this cohort. From a clinical counseling perspective, this finding argues for setting calibrated expectations in patients presenting with NSAID-associated gastritis, where symptomatic recovery is characteristically slower and less complete than in H. pylori eradication candidates.

The high prevalence of cobalamin deficiency (78.6%) in the autoimmune gastritis subgroup reaffirms that anti-intrinsic factor antibody-mediated vitamin B12 malabsorption is a near-universal consequence of advanced autoimmune gastritis. Prompt and sustained parenteral supplementation alongside long-term endoscopic surveillance for gastric neuroendocrine tumors and adenocarcinoma constitute non-negotiable clinical priorities in this population. Several limitations constrain the interpretation of these findings. The single-center design may limit external validity across settings with divergent H. pylori resistance profiles. The 12-week follow-up window was insufficient for evaluating long-term mucosal dynamics, particularly atrophy stabilization trajectories or cancer risk modification. Furthermore, antibiotic susceptibility testing was not routinely performed before initiating first-line therapy, given limited access to culture-based and molecular resistance assays in the study environment. Future multicenter studies incorporating susceptibility-guided initial therapy, extended follow-up exceeding 24 months, and broader demographic representation would materially strengthen the evidentiary foundation for these management approaches.

Systematic integration of multimodal diagnostic protocols with etiology-guided treatment substantially improves outcomes in gastritis management. Bismuth quadruple therapy achieves high first-line eradication rates, and histological mucosal recovery following H. pylori clearance is robust and clinically meaningful. Expanding access to antibiotic susceptibility testing and maintaining long-

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term mucosal surveillance remain critical clinical priorities for reducing gastritis-associated oncological risk.

References

1. Chen Y.-C., Malfertheiner P., Yu H.-T. et al. Global prevalence of Helicobacter pylori infection and incidence of gastric cancer between 1980 and 2022 // Gastroenterology. - 2024. - Vol. 166, № 4. - P. 605-619.
2. Li Y., Choi H., Leung K. et al. Global prevalence of Helicobacter pylori infection between 1980 and 2022: a systematic review and meta-analysis // Lancet Gastroenterology and Hepatology. - 2023. - Vol. 8, № 6. - P. 553-564. - DOI: 10.1016/S2468-1253(23)00070-5.
3. Azer S.A., Awosika A.O., Akhondi H. Gastritis // StatPearls [Internet]. - Treasure Island (FL) : StatPearls Publishing, 2024. - Режим доступа: https://www.ncbi.nlm.nih.gov/books/NBK544250/.
4. Chinese Society of Gastroenterology, Cancer Collaboration Group. Guidelines for diagnosis and treatment of chronic gastritis in China (2022, Shanghai) // Journal of Digestive Diseases. - 2023. - Vol. 24, № 3. - P. 150-180.
5. Ивашкин В.Т., Маев И.В., Лапина Т.Л. и др. Клинические рекомендации Российской гастроэнтерологической ассоциации по диагностике и лечению инфекции Helicobacter pylori у взрослых // Российский журнал гастроэнтерологии, гепатологии, колопроктологии. - 2018. - Т. 28, № 1. - С. 55-70. - DOI: 10.22416/1382-4376-2018-28-1-55-70.
6. Ивашкин В.Т., Маев И.В., Лапина Т.Л. и др. Клинические рекомендации Российского гастроэнтерологического общества и РЭНДО по диагностике и лечению гастрита и дуоденита // Российский журнал гастроэнтерологии, гепатологии, колопроктологии. - 2021. - Т. 31, № 4. - С. 70-99. - DOI: 10.22416/1382-4376-2021-31-4-70-99.
7. Rugge M., Genta R.M., Graham D.Y. et al. Gastritis: the clinico-pathological spectrum // Digestive and Liver Disease. - 2011. - Vol. 43, № 3. - P. 170-179. - DOI: 10.1016/j.dld.2010.09.006.

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