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PERSISTENT GASTRIC MUCOSAL INFLAMMATION AFTER HELICOBACTER PYLORI ERADICATION: MECHANISMS, CLINICAL SIGNIFICANCE AND MANAGEMENT

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Abstract
Helicobacter pylori is a Group 1 carcinogen and a principal cause of chronic active gastritis and downstream premalignant lesions. Successful eradication reduces gastric cancer incidence at the population level, yet post-treatment mucosal recovery is frequently incomplete, with persistent inflammation reported during endoscopic follow-up. This narrative review integrates evidence from clinical follow-up cohorts, randomized trials, and mechanistic studies addressing post-eradication gastric mucosal inflammation, framed using the Updated Sydney System (activity vs chronic inflammation) and OLGA/OLGIM staging. Across longitudinal biopsy studies, neutrophilic activity declines earlier after eradication than chronic mononuclear inflammation, while atrophy and intestinal metaplasia regress slowly and may persist for years. Persisting inflammation after confirmed eradication may reflect residual immune activation, ongoing chemical/reactive injury (e.g., bile reflux, NSAIDs), or altered post-treatment microbial ecology. Dyspeptic symptoms may also persist despite cure, consistent with overlap with functional dyspepsia and non–H. pylori gastropathies. A practical approach emphasizes confirmation of eradication, identification of non–H. pylori drivers, and stage-based risk stratification to guide follow-up and surveillance.

Keywords: Helicobacter pylori; eradication therapy; post-eradication gastritis; Updated Sydney System; OLGA/OLGIM; gastric mucosa.

Introduction

Helicobacter pylori infection is a central driver of chronic active gastritis and a key initiating factor in the multistep pathway that can progress from chronic inflammation to atrophy, intestinal metaplasia, dysplasia, and ultimately gastric adenocarcinoma. In recognition of its carcinogenic potential, H. pylori infection has been classified as a Group 1 carcinogen by the International Agency for Research on Cancer (IARC). [1] Consistent with this, contemporary international consensus documents recommend testing for and treating H. pylori across a range of clinical settings, both to resolve disease and to reduce downstream cancer risk. [2] Within the "gastric precancerous cascade," persistent mucosal inflammation is not merely an

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epiphenomenon but a biological "engine" that promotes epithelial injury, gland loss, and metaplastic remodeling over time.[3] From a preventive perspective, eradication therapy is therefore positioned as a cornerstone intervention. Randomized trial evidence synthesized in recent meta-analysis indicates that H. pylori eradication reduces the incidence of gastric cancer (and likely gastric cancer–related mortality in some settings), supporting population-level and risk-targeted eradication strategies.[4] However, clinical experience and longitudinal biopsy studies highlight an important dissociation between microbiological cure and tissue recovery: successful eradication does not guarantee immediate normalization of gastric mucosal inflammation. In long-term follow-up data, neutrophilic "activity" tends to resolve relatively early after cure, while chronic mononuclear inflammation improves more gradually and may persist at a lower level for years; regression of advanced premalignant lesions is typically slower and less complete than regression of early lesions.[5] This post-treatment spectrum—variably termed post-eradication gastritis, residual inflammation, or persistent gastritis after eradication—is clinically relevant because it intersects with (i) persistent dyspeptic symptoms, (ii) differential diagnosis of non-H. pylori gastropathies (e.g., reactive/chemical injury), and (iii) decisions about risk stratification and surveillance in patients with atrophy and/or intestinal metaplasia. A further challenge is that "persistent inflammation" is not a single-pathway phenomenon. After eradication, the gastric ecosystem and barrier biology can remain altered: emerging evidence suggests that gastric microbial community patterns after eradication may associate with persistent inflammation and with the presence or emergence of atrophy and intestinal metaplasia during follow-up, raising the possibility that post-eradication dysbiosis contributes to continued mucosal immune activation in susceptible hosts.[8] These mechanisms may be particularly relevant in patients with established mucosal remodeling (atrophy/metaplasia), where architectural and functional changes can sustain inflammatory signaling despite clearance of the inciting organism. Given these complexities, standardized histopathology frameworks are essential to interpret post-eradication biopsies reproducibly and to communicate clinically meaningful endpoints. The Updated Sydney System remains the foundational approach to grade gastritis by separating activity (neutrophils) from chronic inflammation (mononuclear infiltrate) and documenting atrophy, intestinal metaplasia, and H. pylori density.[6] For prognosis-oriented reporting, the OLGA staging system integrates severity and topography of atrophy to stratify cancer risk, while the use of intestinal metaplasia–based staging (supporting the OLGIM concept) has been advocated because intestinal metaplasia is often more reproducible than atrophy in routine practice.[7]

Definitions and histologic frameworks

In this review, post-eradication gastric mucosal inflammation refers to histologic inflammatory changes in gastric biopsies obtained after confirmed H. pylori eradication, recognizing that mucosal healing is time-dependent and may be influenced by non-H. pylori injuries. Confirmation should rely on non-serologic tests (e.g., urea breath test or stool antigen test) performed at appropriate intervals and without confounding medications.[12] The Updated Sydney System remains the most widely used framework for reporting gastritis and is

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particularly useful after eradication because it separates "activity" from "chronic inflammation" and records structural remodeling endpoints. It grades (typically none/mild/moderate/marked): Chronic inflammation (mononuclear infiltrate), Activity (neutrophils), H. pylori density, Atrophy, Intestinal metaplasia, and interprets them by topography (antrum vs corpus ± incisura).[6] Biopsy topography matters. For risk stratification and reproducibility, guidelines commonly recommend systematic mapping with at least antrum and corpus biopsies in separate jars, with many protocols also sampling the incisura (Sydney protocol).[6] Active (neutrophilic) inflammation vs chronic inflammation. Neutrophils in the lamina propria and/or within the foveolar/glandular epithelium (including pit abscesses). Interpretation after eradication: Activity usually declines earlier than chronic inflammation; persistent activity should prompt reassessment for ongoing injury or missed/recurring infection. Chronic inflammation Definition: Predominantly lymphocytes and plasma cells in the lamina propria.
Interpretation after eradication: Chronic inflammation may persist at low grade for years despite cure, and its significance depends on time from therapy, topography, and accompanying atrophy/metaplasia stage.[13] Atrophic gastritis is defined by loss of native gastric glands (antral or oxyntic), with replacement by connective tissue (non-metaplastic atrophy) and/or metaplastic epithelium (metaplastic atrophy) on a background of chronic inflammation. Staging (OLGA): The OLGA system stages gastritis by combining severity and topography of atrophy across antrum and corpus into stages 0–IV, which correlate with gastric cancer risk (higher stages, especially III–IV, are higher risk).[21]Metaplasia (replacement by non-native phenotype). Intestinal metaplasia (IM)Definition: Replacement of gastric mucosa by intestinal-type epithelium characterized histologically by goblet cells (± absorptive/Paneth cells). IM is a key endpoint for long-term risk stratification and is often slow to regress after eradication.[21] Staging (OLGIM): Because IM can be more reproducible than "atrophy" in routine practice, IM-based staging is frequently used:Evidence supports using IM as an alternative for OLGA staging when atrophy scoring is challenging.[9] The OLGIM concept has been evaluated against OLGA for cancer-risk ranking and provides clinically useful stratification (though it may be less sensitive in some settings). Distinguishing persistent "gastritis" from reactive/chemical injury. Not all post-eradication abnormalities represent ongoing immune gastritis. Reactive (chemical) gastropathy—often related to NSAIDs, bile reflux, alcohol, or medication effects—has characteristic histology (e.g., foveolar hyperplasia, mucin depletion, lamina propria edema, smooth muscle fibers) and may show little inflammation, so it can confound interpretation of "persistent gastritis." [14]
Mechanisms of persistent inflammation after eradication

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1) Apparent persistence due to ongoing infection (treatment failure, recrudescence, reinfection) or testing confounders. A practical first step is to ensure that "post-eradication inflammation" is not driven by unrecognized ongoing infection. The Maastricht VI/Florence consensus emphasizes confirmation of eradication with non-serologic testing and awareness of test limitations.[2] PPIs can cause false-negative testing (and can suppress bacterial density on biopsy-based tests); classic data show that urea breath tests may become falsely negative during PPI therapy, supporting PPI washout before testing. Where epidemiologic risk is high, recurrence can represent recrudescence (early) or reinfection (later); strain-typed clinical studies demonstrate both phenomena after therapy.[10] 2) Residual immune activation ("immune memory") after bacterial clearance. Even with confirmed eradication, low-grade chronic inflammation can persist long-term. In a cohort evaluated a median ~6 years after successful eradication, mild inactive chronic inflammation was still observed in a subset of patients.[11] Mechanistically, adaptive immune responses may not fully reset: H. pylori–specific Th17 responses and elevated mucosal cytokine signals (e.g., IL-1β) have been reported even in individuals with past infection, supporting an "immune memory" model for persistent inflammation.[12] 3) Baseline mucosal remodeling (atrophy/metaplasia) limits "return to normal"Inflammation, atrophy, and IM evolve on different timelines. Longitudinal follow-up data indicate that preneoplastic lesions regress with time free of infection, but regression is slower and less complete in more advanced lesions, implying that patients with established atrophy/IM may show persistent inflammatory and architectural changes despite cure.[5] This aligns with risk frameworks that treat atrophy/IM as durable markers for follow-up decisions (MAPS II; AGA expert review). 4) Persistent epithelial barrier dysfunction and tight-junction remodeling. H. pylori can directly disrupt epithelial junctional complexes and barrier function through multiple virulence-associated pathways, and junctional remodeling may persist in an inflamed or remodeled mucosa. Reviews detailing these mechanisms highlight tight junction dysregulation and its relevance to ongoing mucosal vulnerability.[13] Barrier disruption provides a biologically plausible bridge from "past infection" to continuing exposure of the lamina propria to luminal irritants (acid, bile acids, microbial products), sustaining inflammation even when H. pylori is no longer detectable.[13] 5) Post-eradication microbiome shifts (dysbiosis) that correlate with inflammation and premalignant lesions. Eradication reshapes the gastric ecosystem. In a randomized study with paired biopsies, specific gastric microbial signatures one year after eradication were associated with persistent/progressive inflammation, atrophy, and intestinal metaplasia, suggesting that non-H. pylori microbes may contribute to ongoing mucosal inflammation after cure.[8] 6) Ongoing chemical/reactive injury: bile reflux, NSAIDs, alcohol, medication effects. Bile reflux gastritis can cause chronic mucosal injury through bile acid–mediated disruption of the mucus barrier and increased epithelial permeability, and it can coexist with or follow H. pylori gastritis, complicating post-eradication interpretation.[15] Similarly, reactive gastropathy—most commonly NSAID-related—may mimic persistent post-eradication pathology and should be actively considered when inflammation does not follow expected trajectories. 7) Alternative diagnoses unmasked after eradication (autoimmune

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atrophic gastritis). In some patients, persistent corpus-predominant inflammation/atrophy reflects autoimmune atrophic gastritis rather than residual H. pylori disease; expert guidance emphasizes topographic biopsies, risk stratification, and evaluation for iron/B12 deficiency and autoimmune markers when suspected.[16]

Clinical manifestations and symptom persistence

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After H. pylori eradication, patients may be asymptomatic despite residual histologic inflammation, while others report persistent or recurrent upper-GI symptoms (typically "dyspepsia"). Symptom persistence should not be interpreted as proof of eradication failure because symptom response correlates poorly with treatment success, and objective "test-of-cure" is recommended irrespective of symptoms.[21] Dyspepsia phenotypes: H. pylori–associated dyspepsia vs functional dyspepsia. The Kyoto Global Consensus emphasizes that H. pylori gastritis can cause dyspepsia in a subset of patients and proposes "H. pylori–associated dyspepsia" as a distinct entity only when eradication is followed by sustained symptom remission. If symptoms persist or recur after confirmed eradication, patients should be considered to have functional dyspepsia (FD). [17] Clinically, this framework is useful for post-eradication follow-up: Sustained improvement (often requiring months, not immediate relief) supports an H. pylori–attributable symptom mechanism.[17] Persistent symptoms shift the focus toward FD mechanisms (visceral hypersensitivity, impaired accommodation, delayed/rapid gastric emptying, psychosocial factors) and toward alternative organic disease (GERD, peptic ulcer if eradication failed, medication-related injury, bile reflux).[17] How often do symptoms improve after eradication? Evidence synthesis indicates that eradication provides a modest but real benefit in dyspepsia outcomes: A contemporary systematic review/meta-analysis reports high-quality evidence that eradication can cure or improve FD symptoms, though the overall effect size is modest.[19] A randomized-trial meta-analysis reported symptom improvement in ~41% with eradication vs ~33% with control, with NNT ~15 for symptom improvement.[20] The 2024 ACG guideline summarizes similar pooled effects (e.g., NNT values for symptom cure/improvement) and emphasizes eradication as one of the few potentially "curative" options for a subset of dyspeptic patients with active infection.[18] Persistent symptoms: common scenarios and clinical interpretation 1) Functional dyspepsia overlap (most common). If dyspepsia persists after confirmed cure, Kyoto consensus supports labeling as FD, and Rome IV criteria provide symptom-based subtypes (postprandial distress syndrome and epigastric pain syndrome), which can overlap.[17] 2) GERD/reflux symptoms after eradication Reflux symptoms may appear after therapy in a subset of patients. A meta-analysis of 27 studies reported overall post-eradication incidence rates around ~15% for endoscopic reflux esophagitis and ~20% for reflux-related symptoms, with regional variation and differences by study design.[21] This is clinically relevant because symptoms labeled "indigestion" may actually be reflux, and management pathways differ.3) Ongoing mucosal injury not driven by H. pylori. Persistent epigastric discomfort can reflect non-H. pylori drivers such as NSAID exposure, bile reflux, alcohol, or reactive gastropathy patterns; in such cases, histology may show changes that do not mirror classic H. pylori activity patterns (and symptom–histology correlation is often weak).4) Missed or recurrent infection (must be excluded) Given poor correlation between symptoms and eradication success, guidelines emphasize a structured post-treatment confirmation strategy rather than symptom-guided assumptions.[18] Practical follow-up logic (symptom-centered)If symptoms persist and eradication status is unknown → test of cure first, then treat FD/GERD or other etiologies

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accordingly.[18]If symptoms persist despite confirmed cure → manage as FD/GERD and evaluate alternative pathology based on age/risk/alarm features and local dyspepsia guidelines. Laboratory and biomarkers

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A) Biomarkers to confirm eradication (mandatory step before interpreting "post-eradication" inflammation) Guidelines recommend confirming eradication using: Urea breath test (UBT) or Fecal antigen test, or Biopsy-based tests when endoscopy is indicated. Key technical requirements (ACG 2024): Perform test-of-cure ≥4 weeks after completion of therapy. Stop PPIs for 2 weeks before UBT or fecal antigen testing (to avoid false negatives). Avoid bismuth and antibiotics for ≥4 weeks before test-of-cure. Do not use serology to establish post-treatment status (antibodies can persist for months–years).[24]These rules matter for "persistent inflammation" interpretation: false-negative cure tests can lead to misclassification of ongoing infection as "residual gastritis."B) Biomarkers for mucosal remodeling risk (atrophy/intestinal metaplasia risk stratification) For patients in whom the main concern is advanced atrophic gastritis/intestinal metaplasia (IM) (rather than active infection), noninvasive biomarker strategies have been evaluated—especially in higher-incidence regions. Serum pepsinogens (PGI, PGII, PGI/PGII ratio) MAPS II states that low pepsinogen I and/or a low PGI/PGII ratio identify patients with advanced atrophic gastritis, and recommends endoscopy for those with low pepsinogen test levels—particularly when H. pylori serology is negative, which may suggest extensive IM and higher gastric cancer risk.[23] MAPS II also notes that pepsinogen thresholds vary by assay/population and should be validated for the specific test used. [18]Gastrin-17 and combined biomarker panels. MAPS II discusses combined serologic approaches (pepsinogen I/II + gastrin-17 + anti-H. pylori antibodies), but highlights that previously published evidence showed little yield from adding gastrin-17 to pepsinogen for detecting atrophy, whereas adding H. pylori serology to pepsinogen may help identify higher-risk individuals in some settings.[23] Important clinical positioning (for MEJDD narrative review): Pepsinogen-based testing is best viewed as a triage tool (who should get endoscopy and staging), not as a replacement for histology-based frameworks (Sydney/OLGA/OLGIM). C) Biomarkers for autoimmune/corpus-predominant atrophic gastritis (and "extra-gastric" consequences) When persistent inflammation/atrophy is corpus-predominant or when there is unexplained anemia, clinicians should consider autoimmune gastritis pathways. AGA Clinical Practice Update (2021) advises: In suspected autoimmune gastritis: check antiparietal cell antibodies and anti–intrinsic factor antibodies to support diagnosis. Evaluate for iron deficiency and vitamin B-12 deficiency in atrophic gastritis irrespective of etiology (especially corpus-predominant), and consider atrophic gastritis in the differential of unexplained iron/B-12 deficiency.[22] These labs are clinically important in post-eradication settings because symptoms such as fatigue or nonspecific dyspepsia may coexist with micronutrient deficiencies due to hypochlorhydria and impaired intrinsic factor function. D) Emerging biomarkers (research use; not ready for routine practice) MAPS II notes several investigational approaches for noninvasive assessment of gastric atrophy risk (e.g., ghrelin, trefoil factors, microRNA panels, volatile organic compounds), but concludes evidence is currently insufficient for routine clinical application. [23]

Endoscopic and morphological dynamics after eradication

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Early phase (weeks to ~3 months): inflammation fades faster than architecture normalizes. After confirmed eradication, endoscopic signs of active H. pylori gastritis (e.g., diffuse redness, nodularity, enlarged folds) often improve first, reflecting reduction of inflammatory activity. In Kyoto endoscopic scoring, total Kyoto scores decrease after eradication, with particularly consistent improvement in diffuse redness, nodularity, and enlarged folds, while features reflecting chronic remodeling improve more slowly.[26] Intermediate phase (~6–12 months): chronic inflammation declines, but "complete normalization" is variable. Longitudinal histology studies show that successful eradication is followed by significant long-term improvements in gastritis, compared with persistent infection, but the speed and completeness of recovery vary between individuals and gastric compartments.[25] Endoscopically, reappearance of "normal" patterns such as regular arrangement of collecting venules (RAC) in the gastric body supports a return toward non-infected mucosa, although RAC is not a standalone test and may be affected by background mucosal change. [28]Late phase (years): atrophy/intestinal metaplasia (IM) often persists and drives residual risk. Even after eradication, preneoplastic lesions regress slowly and incompletely, particularly when baseline atrophy/IM is advanced. In a randomized trial with long-term follow-up, eradication was associated with more regression and/or slower progression of precancerous lesions, supporting the concept that tissue remodeling follows a much longer trajectory than bacterial clearance. [5] Clinically important consequence: patients with histologic IM or severe endoscopic atrophy remain at increased risk of gastric cancer development after eradication, making stage-based follow-up essential.[29]

Determinants of non-resolution

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1) Reinfection, recrudescence, and true recurrence. Residual inflammation may reflect unrecognized ongoing infection (treatment failure/recrudescence) or later reinfection. Strain-typed and follow-up studies demonstrate that recrudescence tends to occur early (within months), whereas reinfection risk is context-dependent and higher in some settings.[30] Follow-up cohorts also report measurable recurrence rates over 1–3 years, emphasizing the need for objective test-of-cure and (when relevant) counseling about reinfection risks.[31] 2) Antibiotic resistance and regimen effectiveness (treatment-failure context). Global increases in macrolide and fluoroquinolone resistance have reduced the reliability of legacy regimens. Major guidelines therefore recommend avoiding clarithromycin- or levofloxacin-containing regimens unless susceptibility is known, and using locally effective quadruple regimens as empiric therapy when resistance patterns are uncertain. [2] 3) Microbiome changes after eradication (post-treatment dysbiosis). Eradication alters the gastric niche; importantly, a randomized study identified specific gastric microbes associated with progressive inflammation, atrophy, and IM one year after eradication, supporting a plausible pathway for "post-eradication inflammation" independent of ongoing H. pylori.[8] 4) PPI exposure (both as a confounder and a biological modifier). Proton pump inhibitors can mask infection by producing false-negative urea breath tests, and can reduce biopsy-based detection sensitivity if used before endoscopy—leading to misclassification of persistent infection as "residual gastritis." [18] Therefore, "non-resolution" should always be interpreted with careful attention to PPI washout and test timing.[18] 5) Bile reflux and reactive/chemical injury. Bile reflux can cause reactive gastritis, modify the histologic phenotype of gastritis, and has been associated with atrophy and intestinal metaplasia, complicating post-eradication interpretation.[32] 6) Autoimmune gastritis differential (corpus-predominant disease). Persistent corpus-predominant inflammation/atrophy after eradication should prompt evaluation for autoimmune atrophic gastritis, especially with iron or vitamin B12 deficiency, and appropriate autoimmune serology (parietal cell/intrinsic factor antibodies) when clinically indicated. [22]

Management and follow-up strategies

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Step 1 — Confirm eradication correctly (do not use symptoms as a "test"). All treated patients should undergo a test-of-cure using urea breath test, stool antigen test, or biopsy-based testing ≥4 weeks after therapy, with PPI held for ~2 weeks and antibiotics/bismuth avoided for ~4 weeks to minimize false negatives.[18] Step 2 — If symptoms or histologic activity persist, systematically search for drivers. A practical post-eradication work-up when inflammation persists should include: Re-check eradication status (with appropriate washout) and consider local resistance/treatment history for possible failure.[18] Review mucosal injury exposures: NSAIDs, alcohol, and features suggesting bile reflux/reactive gastropathy.[15] Consider autoimmune gastritis (corpus-predominant atrophy; anemia/B12/iron deficiency).[22] Step 3 — Risk-stratify by atrophy/IM to decide endoscopic follow-up and surveillance. For patients with gastric intestinal metaplasia (GIM) or advanced atrophy, follow-up should be driven by extent/topography and risk profile, rather than by "presence of mild chronic inflammation". MAPS II (ESGE/EHMSG/ESP) supports surveillance in individuals with advanced atrophy/IM, commonly using ~3-year intervals for higher-risk patterns (and shorter intervals for very high-risk constellations). [15] AGA GIM guideline generally suggests against routine surveillance for all patients, but supports shared decision-making and surveillance consideration in higher-risk groups (e.g., extensive IM, family history, high-incidence ancestry/background).[33] Patients with histologic IM or severe endoscopic atrophy after eradication merit particular attention due to their documented higher post-eradication gastric cancer risk. Step 4 — Address bile reflux/reactive injury when suspected. Management is individualized (diet/behavioral factors, prokinetic strategies when appropriate, mucosal protectants), and selected studies suggest symptom improvement with agents such as ursodeoxycholic acid in bile reflux gastritis; however, evidence quality varies and treatment should be tailored to confirmed reflux/clinical context.

Evidence gaps and future directions

Standardized definition of post-eradication gastritis (what timing, what endpoints—histology vs symptoms vs endoscopy) is still inconsistent across studies, limiting comparability and guideline translation.[22] Optimal follow-up timing and biopsy mapping (especially for OLGA/OLGIM staging after eradication) needs prospective validation across diverse populations and risk settings.[33] Integration of endoscopic scoring with histology: Kyoto classification improves after eradication, but how best to combine Kyoto score trajectories with OLGA/OLGIM for surveillance decisions remains unsettled. [27]Causality of microbiome changes: microbes associated with post-eradication progression of inflammation/atrophy/IM are promising targets, but interventional trials are needed to test whether modifying the microbiome changes outcomes.[8] Management trials for residual inflammation: beyond confirming cure and treating alternative injuries (e.g., bile reflux, NSAIDs), evidence for specific anti-inflammatory or barrier-directed interventions post-eradication is limited and heterogeneous.[18]

Conclusion

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After successful H. pylori eradication, endoscopic inflammatory features often improve earlier than histologic remodeling lesions. Persistent or recurrent inflammation ("post-eradication gastritis") most commonly reflects a spectrum of drivers: missed/recurring infection in the setting of resistance or testing confounders, ongoing chemical injury (especially bile reflux/NSAIDs), microbiome shifts, and autoimmune gastritis in corpus-predominant disease. Evidence-based post-eradication care should prioritize proper test-of-cure, systematic evaluation of alternative etiologies, and risk stratification by atrophy/IM extent (OLGA/OLGIM and/or validated endoscopic frameworks) to guide follow-up and surveillance.[2]

Ethical approval

Not applicable. This manuscript is a narrative review and did not involve human participants or animals.

Conflict of interest

The authors declare no conflict of interest related to this work.

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