Review Article

Helicobacter pylori in Dental Calculus: Its Role in Gastritis Recurrence and Treatment

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Abstract

Helicobacter pylori (*H. pylori*) is a Gram-negative, microaerophilic, spiral-shaped bacterium and a well-established causative agent of chronic gastritis, peptic ulcers, and gastric adenocarcinoma. Traditionally regarded as a purely gastric pathogen, *H. pylori* has been increasingly identified in extragastric sites, most notably within the oral cavity-particularly in dental calculus. The biofilm-protected environment of dental calculus enables *H. pylori* to evade systemic antibiotics and host immune defences, facilitating persistence and acting as a reservoir for gastric reinfection following otherwise successful eradication therapy. This paper reviews the mechanisms of oral colonisation, the role of the oral reservoir in gastritis recurrence, the virulence factors that amplify mucosal inflammation, and current as well as innovative therapeutic strategies targeting both gastric and oral niches. A dual-site eradication strategy-combining periodontal therapy with systemic antibiotics-is proposed to improve treatment outcomes and reduce recurrence rates of gastritis, thereby preventing progression to gastric ulcers and malignancies.

Keywords: Helicobacter pylori, dental calculus, oral reservoir, gastritis recurrence, oral submucous fibrosis, biofilm, dual-site eradication, periodontal therapy.

Introduction

Helicobacter pylori is a Gram-negative, flagellated, microaerophilic bacterium that selectively colonises the gastric mucosa and is implicated in one of the most prevalent chronic bacterial infections globally, affecting approximately 44% of the world's population[1]. Its causal association with type B chronic gastritis, peptic ulcer disease, mucosa-associated lymphoid tissue (MALT) lymphoma, and gastric adenocarcinoma has led to its classification by the International Agency for Research on Cancer (IARC) as a definitive Group 1 carcinogen.

For decades, clinical efforts focused exclusively on the gastric niche. However, accumulating microbiological and molecular evidence has established the oral cavity-particularly dental plaque and calculus - as a significant and clinically relevant extragastric reservoir[2]. Dental calculus, the calcified product of dental plaque, provides a structurally complex and metabolically active biofilm environment that shields *H. pylori* from antimicrobial agents and immunological clearance.

The clinical significance of this oral reservoir lies in its capacity to seed gastric reinfection following apparently successful antibiotic eradication therapy. Standard triple or quadruple therapy regimens achieve only 70–85% eradication rates, with oral colonisation implicated as a major driver of the remaining treatment failures and recurrences[3].

This review comprehensively examines the mechanisms of oral *H. pylori* colonisation and biofilm formation, its contribution to gastritis recurrence and virulence, the challenges of current treatment paradigms, and emerging therapeutic strategies that integrate periodontal management into *H. pylori* eradication protocols.

Oral colonisation of H. Pylori

Prevalence in the Oral Cavity and Dental Calculus

H. pylori has been identified in multiple oral niches including dental plaque (both supragingival and subgingival), saliva, tonsillar tissue, and dental calculus. The prevalence of oral *H. pylori* is variable across studies, influenced by detection methodology, geographic region, and the periodontal health status of subjects[2]. Polymerase chain reaction (PCR)-based detection studies consistently report higher sensitivity than culture-based methods, with oral *H. pylori* detection rates ranging from 25% to over 78% in gastric *H. pylori*-positive individuals.

Dental calculus serves as a particularly favourable microenvironment for *H. pylori* colonisation due to its dense three-dimensional biofilm architecture, mineral content, and protection from mechanical disruption. Studies report co-infection rates between gastric and oral *H. pylori* ranging from 40% to 60%, with higher prevalence observed in patients with chronic gastritis or active peptic ulceration[4]. Subgingival calculus, in particular, offers an anaerobic or microaerophilic microenvironment that closely mirrors the

conditions of the gastric mucosa, promoting bacterial survival and metabolic activity.

Mechanisms of Oral Colonisation

The primary mode of *H. pylori* acquisition is person-to-person transmission via the faecal-oral or oral-oral routes, with dental plaque and saliva constituting the principal vehicles in the oral-oral route[1]. Adhesin molecules on the bacterial outer membrane-including BabA (blood group antigen-binding adhesin), SabA (sialic acid-binding adhesin), and Oip A (outer inflammatory protein A)- mediate attachment to epithelial cell surfaces and salivary glycoproteins, enabling colonisation of the oral mucosa and integration into dental plaque biofilms.

Once integrated into the biofilm matrix of dental calculus, *H. pylori* undergoes phenotypic adaptation, including a transition to the coccoid form - a metabolically quiescent, environmentally resistant morphological variant that is less susceptible to antibiotic treatment and maintains the capacity to revert to the virulent spiral form under permissive conditions.

Biofilm Formation and Resistance Mechanisms

H. pylori forms structured biofilms within dental calculus that confer multifactorial resistance to eradication:

- **Extracellular Polymeric Substance (EPS) Matrix:** The EPS matrix - composed of polysaccharides, proteins, lipids, and extracellular DNA - acts as a physical barrier that significantly impedes antibiotic diffusion to inner biofilm layers, reducing effective drug concentrations below minimum inhibitory levels.
- **Efflux Pump Upregulation:** Biofilm-associated *H. pylori* upregulates multidrug efflux pumps (including TolC and HefABC systems) that actively expel antimicrobial agents, contributing to intrinsic resistance beyond that achieved by impermeability alone.
- **Metabolic Quiescence:** Slow-growing or dormant bacterial cells within biofilms exhibit reduced susceptibility to antibiotics that target actively dividing bacteria, explaining why conventional bactericidal agents demonstrate reduced efficacy against biofilm-resident strains.
- **Genetic Resistance Acquisition:** The biofilm microenvironment promotes horizontal gene transfer, facilitating acquisition of resistance determinants-including point mutations in 23S rRNA (conferring clarithromycin resistance) and gyrA/gyrB mutations (conferring fluoroquinolone resistance)-between co-resident bacteria[3].

These compounding resistance mechanisms render oral *H. pylori* significantly more refractory to systemic antibiotic eradication than planktonic gastric strains, fundamentally challenging the efficacy of standard treatment protocols in patients with oral colonisation.

Contribution to Gastritis Recurrence

Reinfection Pathways

The oral cavity functions as a dynamic reservoir for gastric reinfection following successful pharmacological eradication of the gastric *H. pylori* burden. Two primary reinfection pathways have been proposed: (i) direct seeding via salivary flow containing oral *H. pylori* into the stomach, and (ii) gastro-oesophageal reflux facilitating retrograde transfer of oral bacteria into the gastric milieu[4]. In both pathways, the dental calculus reservoir serves as the perpetual source of viable organisms.

Epidemiological studies corroborate this model: patients with untreated periodontal disease and high oral *H. pylori* load demonstrate significantly higher gastric reinfection rates compared to those with good oral hygiene[5]. A landmark prospective study reported a recurrence rate of 32.5% in patients who received systemic eradication therapy without concurrent periodontal intervention, compared to 5% in patients who received combined systemic and periodontal treatment, underscoring the mechanistic importance of the oral reservoir.

Virulence Factors Exacerbating Gastric Inflammation

Oral *H. pylori* strains frequently harbour virulence determinants that directly amplify gastric mucosal pathology:

- **CagA (Cytotoxin-Associated Gene A):** The cagA gene encodes a 120-145 kDa oncoprotein that is translocated into gastric epithelial cells via the Type IV secretion system (T4SS). Once internalised, CagA undergoes tyrosine phosphorylation by Src and Abl kinases, disrupting multiple cellular signalling pathways including those involving SHP-2 phosphatase, leading to cell elongation, loss of polarity, and oncogenic transformation. CagA-positive strains are associated with a two- to five-fold increased risk of gastric adenocarcinoma.
- **VacA (Vacuolating Cytotoxin A):** VacA is a pore-forming toxin that inserts into mitochondrial membranes and induces vacuolation of gastric epithelial cells, activates autophagy, and promotes apoptosis. VacA also suppresses T-cell activation and proliferation, enabling immune evasion by colonising *H. pylori* strains.

- **Co-infection with Periodontal Pathogens:** Synergistic interactions between *H. pylori* and key periodontal pathogens such as *Porphyromonas gingivalis*, *Fusobacterium nucleatum*, and *Treponema denticola* amplify inflammatory responses in the gastric mucosa. *P. gingivali* lipopolysaccharide synergises with *H. pylori*-induced NF-κB activation, elevating pro-inflammatory cytokine release (IL-1β, IL-8, TNF-α) and augmenting mucosal injury[6].

Diagnostic approaches for Oral H. Pylori

Accurate detection of oral *H. pylori* is essential for guiding dual-site treatment decisions. Current and emerging diagnostic modalities include:

- **Polymerase Chain Reaction (PCR):** The gold standard for oral *H. pylori* detection due to its high sensitivity (>90%) and specificity. Targets include the 16S rRNA gene, cagA, vacA, and glmM (ureC) gene loci. Quantitative PCR (qPCR) enables bacterial load quantification.
- **Culture:** Definitive identification with antibiotic sensitivity testing is possible but technically challenging due to the fastidious growth requirements of *H. pylori* (microaerophilic atmosphere, enriched media). Sensitivity for oral specimens is lower than for gastric biopsies.
- **Rapid Urease Test (RUT):** Detects urease activity in dental plaque or calculus samples. Rapid and cost-effective but less sensitive than PCR.
- **Salivary PCR:** Non-invasive, patient-friendly, and suitable for post-treatment monitoring of oral eradication efficacy. Sensitivity varies by sample collection technique.
- **Immunohistochemistry and FISH:** Fluorescence in situ hybridisation (FISH) using species-specific probes allows direct visualisation of *H. pylori* within calculus sections and can simultaneously detect resistance genes.

Post-treatment confirmation of both gastric and oral eradication should employ the ^{13}C-urea breath test (UBT) for gastric clearance and salivary PCR for oral clearance, conducted at a minimum of four weeks after completion of antibiotic therapy.

Challenges in treatment and therapeutic strategies

Limitations of Current Eradication Therapies

Standard triple therapy—consisting of a proton pump inhibitor (PPI) combined with clarithromycin and amoxicillin administered for 7–14 days - historically achieved eradication rates exceeding 90%. However, rising clarithromycin resistance (>25% in many countries) has reduced current efficacy to 70–85%, with further reduction in patients harbouring oral *H. pylori* reservoirs due to biofilm-mediated antibiotic protection[3]. Bismuth quadruple therapy (PPI + bismuth + tetracycline + metronidazole) and sequential or concomitant regimens have been developed as rescue protocols but similarly fail to address the oral reservoir.

The consequence of incomplete eradication extends beyond recurrence of dyspeptic symptoms: persistent *H. pylori* colonisation maintains a state of chronic active gastritis that progressively advances through the Correa cascade—from atrophic gastritis to intestinal metaplasia, dysplasia, and adenocarcinoma. Each episode of reinfection from the oral reservoir resets and perpetuates this oncogenic progression.

Dual-Site Eradication Strategies

The most clinically significant advance in *H. pylori* management has been the conceptualisation and validation of dual-site eradication - simultaneously targeting both gastric and oral reservoirs. Evidence-based approaches are summarised in the table below:

Therapeutic Approach	Mechanism of Action	Clinical Evidence (Recurrence Risk Reduction)
Periodontal therapy (Scaling and Root Planing)	Mechanical debridement removes calculus-associated *H. pylori* biofilm; reduces total oral bacterial load	63% reduction in gastric recurrence risk[1]
Chlorhexidine gluconate mouth rinse (0.12–0.2%)	Disrupts EPS biofilm matrix; broad-spectrum antimicrobial activity against oral flora including *H. pylori*	Significant adjunctive reduction in oral *H. pylori* load; enhances systemic eradication outcomes
Probiotics (*Lactobacillus* spp., *Bifidobacterium*)	Competitive exclusion at adhesion sites; production of bacteriocins and organic acids inhibiting *H. pylori* growth; immune modulation	Reduces reinfection risk by 30–50%; improves eradication rates when combined with triple therapy[5]

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Antimicrobial peptides (AMPs)	Membrane-disrupting cationic peptides (e.g., LL-37, HNP-1) penetrate biofilm EPS matrix and disrupt bacterial membrane integrity with reduced resistance development	Promising in vitro and animal model data; clinical trials in progress[3]
Photodynamic Therapy (PDT)	Light-activated photosensitisers generate localised reactive oxygen species that destroy bacterial membranes; effective against biofilm-resident organisms without antibiotic resistance induction	Early clinical studies show significant oral *H. pylori* load reduction; requires clinical validation in larger cohorts

Novel and Emerging Approaches

Beyond the established strategies outlined above, several emerging modalities hold promise for improving *H. pylori* eradication outcomes by specifically targeting the oral biofilm:

- **Nanoparticle Drug Delivery:** Nanoparticle carriers (silver nanoparticles, liposomal antibiotic formulations) penetrate biofilm EPS matrices more effectively than free antibiotic molecules, delivering bactericidal concentrations to inner biofilm strata inaccessible to conventional therapy.
- **Phage Therapy:** Bacteriophages specific to *H. pylori* have been identified and may be deployed as lytic agents selectively targeting the pathogen within oral and gastric biofilms without disrupting the commensal microbiome.
- **Quorum Sensing Inhibition:** Disrupting quorum sensing signalling pathways that regulate biofilm formation may render *H. pylori* more susceptible to conventional antibiotics by preventing biofilm maturation.
- **Personalised Microbiome Restoration:** Faecal microbiota transplantation (FMT) and targeted oral microbiome restoration using commensal probiotic consortia represent frontier approaches to competitively exclude *H. pylori* from both oral and gastric niches.

Clinical Recommendations

Based on current evidence, the following integrated clinical protocol is recommended for the management of *H. pylori*-associated gastritis, particularly in patients with a history of recurrent infection or treatment failure:

- **Pre-treatment Oral Assessment:** All patients diagnosed with *H. pylori*-positive gastritis should undergo comprehensive oral examination and periodontal charting. Oral *H. pylori* status should be assessed using salivary or subgingival plaque PCR prior to initiating systemic eradication therapy.
- **Integrated Treatment Protocol:** Systemic antibiotic eradication regimens (bismuth quadruple therapy preferred in high-resistance regions) should be initiated concurrently with full-mouth professional periodontal therapy including supragingival and subgingival scaling and root planing. Adjunctive chlorhexidine 0.12% twice-daily rinsing should be continued for the duration of antibiotic therapy.
- **Probiotic Supplementation:** Administration of *Lactobacillus reuteri* or *Lactobacillus acidophilus*-containing probiotics concurrently with antibiotic therapy reduces adverse gastrointestinal effects, improves eradication rates, and provides competitive exclusion in both oral and gastric niches.
- **Post-treatment Eradication Confirmation:** At 4–6 weeks post-completion of therapy, ^{13}C-urea breath test should confirm gastric eradication, and salivary PCR should confirm oral clearance. Incomplete oral clearance warrants targeted periodontal re-treatment.
- **Long-term Surveillance and Prevention:** Patients with a history of *H. pylori* infection should undergo six-monthly professional dental prophylaxis and annual *H. pylori* reassessment. Patient education on oral hygiene optimisation, avoidance of salivary sharing practices, and smoking cessation (a known risk factor for both periodontal disease and *H. pylori* persistence) should be provided.

Discussion

The recognition of the oral cavity-and dental calculus specifically-as an extragastric reservoir of *H. pylori* has fundamental implications for the clinical management of gastric disease. The persistently high global recurrence rates of *H. pylori*-associated gastritis, despite pharmacologically effective eradication regimens, are difficult to explain without invoking an oral reservoir as a source of reinfection[4]. The evidence reviewed in this article provides a mechanistic framework for this phenomenon and supports a paradigm shift in eradication strategy.

A critical, and often overlooked, implication of oral *H. pylori* colonisation is the role of dental professionals in the interdisciplinary management of this infection. Gastroenterologists prescribing eradication therapy without concurrent periodontal intervention may be treating only one site of a dual-site infection, contributing to the treatment failure burden. Conversely, dentists identifying untreated periodontal disease and heavy calculus deposits in patients with recurrent gastric symptoms are ideally positioned to

initiate a referral pathway and provide adjunctive periodontal therapy as part of a coordinated eradication strategy.

Existing literature acknowledges the heterogeneity of oral *H. pylori* detection rates, partly attributable to variability in detection methods, sample sites, and patient populations. The development and standardisation of sensitive, non-invasive salivary PCR protocols would substantially advance the feasibility of oral screening in clinical practice. Furthermore, genotypic characterisation of oral *H. pylori* strains-including cagA status and vacA allele typing-would enable personalised risk stratification and identification of patients at elevated risk of malignant progression[6].

Conclusion

The persistence of *H. pylori* in dental calculus represents a significant, clinically actionable challenge in the management of recurrent gastritis and peptic ulcer disease. The oral biofilm environment confers multifactorial antibiotic resistance, protects the organism from immunological clearance, and enables salivary or reflux-mediated reseeding of the gastric mucosa following systemic eradication therapy. Virulence factors harboured by oral strains - including CagA and VacA - perpetuate gastric mucosal inflammation and drive oncogenic progression along the Correa cascade.

Addressing this challenge demands a multidisciplinary model of care in which gastroenterologists, periodontists, and general dentists collaborate to implement dual-site eradication strategies encompassing systemic antibiotic therapy, professional periodontal debridement, adjunctive antimicrobial rinsing, and probiotic supplementation. Novel approaches including nanoparticle antibiotic delivery, photodynamic therapy, and phage therapy hold promise for targeting biofilm-resident *H. pylori* and warrant rigorous clinical validation.

Ultimately, the integration of oral health assessment and management into *H. pylori* eradication protocols represents a rational, evidence-grounded strategy to improve treatment success rates, reduce recurrence, and mitigate the long-term risk of gastric malignancy in affected individuals.

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