



Dyspepsia following *Helicobacter pylori* eradication: Shifts in etiology and clinical challenges

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Abstract

This editorial discusses a landmark 2025 retrospective cohort study by Suzuki *et al* using > 23000 endoscopic records from Japan's *Helicobacter pylori* (*H. pylori*) eradication program. The study addressed the clinically significant phenomenon of persistent dyspeptic symptoms after successful bacterial eradication. Comprehensive analysis revealed that 28.7% of the patients continued to experience functional dyspepsia (defined as self-reported upper abdominal pain or bloating) after eradication, a prevalence rate indistinguishable from that of the never-infected control groups. Notably, Suzuki *et al* demonstrated no significant epidemiological association between dyspepsia and the risk of gastric cancer. These findings suggest that persistent symptoms may originate from multifactorial mechanisms, including post-eradication alterations in gastric acid secretion, visceral hypersensitivity, and potential gastric microbiome changes. These evidence-based insights support the implementation of stratified management approaches based on individual symptom patterns and sociodemographic characteristics, moving beyond uniform eradication protocols. This study recommends reducing emphasis on dyspepsia symptoms in revising endoscopic screening guidelines to minimize unnecessary interventions and improve medical resource efficiency. This research contributes significantly to mitigating public health anxiety regarding *H. pylori* infection, prevents indiscriminate antibacterial overuse, and provides a robust scientific foundation for developing more rational, evidence-based clinical decision-making frameworks for *H. pylori*-associated dyspepsia management.

Key Words: *Helicobacter pylori*; Persistent dyspepsia; Therapeutic strategies; Etiology; Epidemiology

Core Tip: Although Japan has made significant progress in *Helicobacter pylori* eradication and gastric cancer deaths have declined annually, many patients still suffer from dyspepsia. This challenges the traditional “test-and-eradicate” approach and places a heavy burden on patients. Suzuki *et al*’s large-scale study ($n = 23250$) revealed similar post-eradication dyspepsia prevalence (28.7%) to that in uninfected individuals, suggesting a shift from infection to functional factors, such as gastric acid secretion and visceral hypersensitivity. These findings call for individualized symptom-based management, rather than repeated eradication.

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INTRODUCTION

Japan has a high prevalence of gastric cancer (GC), with approximately 50000 annual deaths, prompting early detection and prevention efforts. *Helicobacter pylori* (*H. pylori*) infection is implicated in most GC cases in Japan[1]. The pathogenic potential of *H. pylori* is heterogeneous, and virulence factors, such as cytotoxin-associated gene A protein, serve as crucial molecular determinants underlying diverse clinical outcomes[2].

In 2000, Japan introduced *H. pylori* eradication therapy into its National Health Insurance (NHI) to treat peptic ulcers associated with *H. pylori* infections[3]. In February 2013, Japan’s NHI extended the coverage of *H. pylori* eradication therapy to patients with *H. pylori*-positive chronic gastritis; a global first aimed at reducing GC incidence and mortality, which has declined thereafter[4].

In Japanese clinical practice, the guidelines for dyspepsia advocate eradication therapy as the first-line treatment modality for all *H. pylori*-positive cases[5]. However, many patients continue to experience persistent dyspepsia post-eradication[6], highlighting the limitations of an “infection-centric” approach and ambiguous relationship between dyspepsia and *H. pylori* infection status. Previously, large-scale population-based investigations into the prevalence of dyspepsia and endoscopic manifestations among post-*H. pylori* were lacking. Suzuki *et al*[7] aimed to address this gap in the epidemiological understanding of dyspepsia in the post-eradication era, thereby providing a robust evidence base for clinical practice and formulation of public health policies.

KEY FINDINGS OF THE STUDY

This large-scale population-based study analyzed data from > 23000 adults who underwent gastroscopic screening to systematically probed the epidemiological characteristics of dyspepsia and its associated endoscopic manifestations in the era of *H. pylori* eradication.

The study findings indicated that despite successful *H. pylori* eradication, the symptoms associated with dyspepsia remained widespread. The prevalence rate in the eradication and non-infected groups was 28.7%, which was significantly higher than that in the currently infected or spontaneously eradicated groups (25.8%). Multivariate regression analysis identified crucial factors that were independently associated with dyspepsia, including aged < 60 years, women, gastric ulcers, duodenal ulcers, erosive esophagitis, history of gastric surgery, and successful *H. pylori* eradication. Notably, no significant association was detected between GC, esophageal cancer, and symptoms of dyspepsia.

Endoscopic examination revealed that > 90% of patients with dyspepsia exhibited no organic lesions. This suggests that the primary etiology of dyspeptic symptoms may no longer be traditional organic diseases, such as ulcers and cancers, but rather functional dyspepsia (FD). Notably, this study broadly defined dyspepsia as self-reported upper abdominal pain or bloating, which encompasses but is not strictly limited to Rome IV-defined FD.

This study revealed remarkable age- and sex-related disparities. The prevalence rate among individuals aged < 60 years was significantly higher than that among the elderly population (33.5% *vs* 27.0%) and conspicuously higher in women than in men.

By adopting rigorous data collection methodologies and statistical analysis techniques, this study offers significant references for dyspepsia diagnosis and treatment and a scientific foundation for public health departments in the long-term management of *H. pylori* eradication, impacting clinical practice and public health.

IMPLICATIONS

Implications for clinical practice

Dyspeptic symptoms should not be considered a reliable basis for screening upper gastrointestinal malignancies. This finding is consistent with the conclusions of several international studies[8]. Modern endoscopic screening protocols detect most malignant tumors at the asymptomatic stage. Overreliance on symptom-driven decision-making for endoscopic referral risks missing early-stage cancers, which are often asymptomatic, leads to the inefficient use of endoscopic resources, and increases the financial burden on healthcare systems. Thus, screening strategies should be based on age and risk factors, rather than symptoms[9].

Implications for patient management

Persistent FD after *H. pylori* eradication warrants further clinical attention. The study revealed that the prevalence of dyspepsia in the eradicated group (28.7%) was comparable to that in the never-infected group and higher than that in the currently infected or naturally eradicated groups (considering that spontaneous eradication is exceedingly rare, this group likely approximates an active infection). Although eradication effectively eliminates infection, it does not address the underlying pathophysiological mechanisms of FD, such as visceral hypersensitivity or impaired gastric accommodation[10]. Furthermore, eradication therapy can dynamically affect symptom expression. Factors, such as post-eradication gastric acid recovery, short- and long-term impacts of antibiotics[11] on gut microbiota[12], and the “indication confusion” effect, in which patients seek care because of persistent symptoms, may collectively contribute to the complex symptom profiles observed in cross-sectional studies[13]. Therefore, patient management should be forward-looking and longitudinal rather than relying solely on post-eradication assessment. Successful *H. pylori* eradication should not be viewed as a treatment endpoint. Clinicians should implement a comprehensive management approach, including dietary modifications, proton pump inhibitors (PPIs), neuromodulators, or psychological interventions, as per the latest FD guidelines[14]. This approach fosters realistic expectations and may reduce unnecessary healthcare use owing to unresolved symptoms.

Implications for public health

Efforts should focus on reducing public anxiety, alleviating the economic burden of medical care, and rationally allocating healthcare resources. This study challenges the traditional paradigm that *H. pylori* eradication equals complete symptom resolution, providing evidence for optimizing national healthcare resource allocation. The findings indicated no significant association between dyspepsia and GC, and > 92.3% of the patients showed no organic lesions upon endoscopic examination. Moreover, using dyspepsia as an absolute indication for endoscopic screening results in many unnecessary procedures[15]. Consequently, public health systems should take two complementary approaches. First, endoscopic resources should be prioritized for high-risk populations, such as those with severe gastric atrophy or a family history of GC. Second, it promotes a tiered healthcare delivery model, in which primary care physicians emphasize cost-effective interventions, such as symptom management and lifestyle modification[16]. Additionally, eradicating *H. pylori* is not the “magic key” to solving all stomach discomfort. This may help guide the public to have more rational treatment expectations, thereby indirectly alleviating unnecessary health concerns. Targeted health education can reduce public anxiety and minimize unnecessary healthcare use. Ultimately, these strategies support a shift from a single-disease control model to a comprehensive framework that integrates symptom management and resource optimization. This transformation may enhance population health outcomes, while reducing the burden on medical insurance systems and ensuring their long-term sustainability.

DISCUSSION ON STUDY LIMITATIONS

This retrospective cohort study provides valuable insights but has several methodological limitations that warrant careful interpretation. First, the classification of *H. pylori* infection status, based on endoscopic evidence of gastric atrophy and self-reported eradication history, groups individuals with current infections and those who have naturally cleared the infection, resulting in a heterogeneous exposure category. Additionally, relying solely on self-reported eradication history without confirmation through objective tests, such as urea breath tests, may introduce bias in classification errors. This may affect the validity of the “eradication post cohort”. Although this approach may enhance the feasibility of large-scale studies, it hampers the ability to disentangle the distinct contributions of active infection and post-infectious mucosal alterations to dyspeptic symptoms.

Second, as an observational study, it can only establish associations rather than causal relationships. A critical unresolved question is whether FD arises as a consequence of eradication therapy itself or whether individuals who pursue treatment are inherently more susceptible to FD, with *H. pylori* infection acting as a contributing factor. This concern is compounded by the likelihood that symptomatic individuals are more inclined to undergo testing and receive treatment, potentially introducing a selection bias.

Additionally, the study lacked data on key potential confounders known to influence the onset and persistence of FD, including psychological comorbidities, dietary habits, and socioeconomic stressors[17]. Another notable limitation is the absence of information on the time elapsed since eradication, which is crucial considering that symptom trajectories may be shaped by dynamic physiological processes, such as the recovery of gastric acid secretion and reconstitution of the gut microbiome following antibiotic exposure[18]. Emerging evidence suggests that antibiotic-induced disruption of the gastrointestinal microbiota may contribute to the development and prolongation of dyspeptic symptoms[19].

CONCLUSION

Suzuki *et al*[7] indicated that dyspeptic symptoms may remain prevalent even after successful eradication of *H. pylori*, challenging the conventional belief that bacterial eradication alone is sufficient to resolve dyspepsia. Our findings provide valuable baseline data and strategic insights for future studies. There are areas that warrant further investigation. First, conducting prospective longitudinal studies to monitor dynamic changes in gastric acid secretion, intestinal microbiota [20], and visceral sensitivity following eradication therapy, thereby elucidating the temporal patterns and underlying mechanisms of symptom evolution, such as *H. pylori* infection, and its eradication significantly affects gastric acid secretion and gastrin levels[21]. *H. pylori* infection induces gastric mucosal inflammation, releases cytokines, and recruits inflammatory cells, thereby altering the sensitivity of internal nerve endings and promoting the development of visceral hypersensitivity[22]. The various effects of multiple antibiotics, such as clarithromycin, metronidazole, levofloxacin, and amoxicillin, often combined with PPI, during eradication treatment lead to dysbiosis of the gastrointestinal microbiota, manifested as reduced diversity and changes in the abundance of specific bacterial populations[23]. Second, strain virulence profiling should be integrated with host-specific characteristics to identify subpopulations that may derive dual benefits from GC prevention and symptom relief, thereby laying the foundation for individualized therapeutic approaches. Third, strategies for optimizing the allocation of endoscopic resources by prioritizing high-risk individuals, such as the elderly, those with severe gastric atrophy, or a family history of GC, and exploring the efficacy and cost-effectiveness of non-eradication treatment approaches within primary care settings. Continued research can yield more diverse and precisely tailored management strategies for patients experiencing persistent post-eradication symptoms, thereby advancing the refinement and personalization of clinical practice. Fourth, establish an intelligent predictive model by integrating multi-omics and clinical big data[24,25]. By consolidating high-dimensional datasets, including patient genomics, gut metagenomics, metabolomics, and electronic health records, and leveraging advanced machine learning and deep learning algorithms, a system capable of accurately predicting the risk of persistent dyspepsia following eradication therapy could be developed[26]. This approach facilitates the early identification of high-risk individuals and offers data-driven decision support for intensified symptom management in select post-treatment patients. Fifth, advanced artificial intelligence-powered real-time analysis and computer-aided diagnostic systems are available for endoscopic imaging[27]. The application of computer vision in gastroscopy enables automated, objective, and quantitative evaluation of gastric mucosal features, such as atrophy and intestinal metaplasia[28]. These systems go beyond supporting early neoplastic detection[29]; and enhance the recognition of subtle mucosal changes associated with functional symptoms, such as microinflammation and abnormal vascular patterns, thereby elucidating the potential functional etiologies of dyspepsia. This capability reduces dependence on inconclusive “no abnormalities detected” reports and transforms endoscopy from a purely morphological assessment tool into one that supports functional and pathophysiological correlation.

FOOTNOTES

Author contributions: Lu JG is responsible for the concept conception and initial draft writing; Gao YZ is responsible for supervision, guidance and review.

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