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LETTER TO THE EDITOR

Surveillance strategies for precancerous gastric conditions after *Helicobacter pylori* eradication: There is still need for a tailored approach

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Abstract

Prevailing evidence declares that *Helicobacter pylori* (*H. pylori*) eradication therapy could shift precancerous gastric conditions (PGC) and positively confines gastric cancer (GC) risk during long-term endoscopic follow-up. Nonetheless, there is a yet unsolved controversy regarding the best-individualized surveillance strategies following *H. pylori* eradication, based on malignant risk stratification. This last dispute is due to the uncertainty of contemporary evidence and the role of *H. pylori* inflammatory changes in underestimating PGC at the index endoscopy. However, the current state of the art suggests that it is reasonable that high-quality endoscopy with histological assessment for the most accurate diagnosis of PGC may be delayed in selected high-risk patients without alarm signs for malignancy, following the eradication of *H. pylori*. Notwithstanding, these aspects need to be further examined in the next future to establish and optimize the most beneficial and cost-effective strategies for recognizing and managing *H. pylori*-positive patients with PGC in the short- and long-term follow-up. Accordingly, additional studies are yet required to sharpen the hazard stratification of patients with the greatest chance of GC evolution, also recognizing the evolving racial, ethnic, immigration factors and the necessity of novel biomarkers to limit GC development or accomplish a diagnosis of malignancy at an early stage.

Key Words: *Helicobacter pylori*; Endoscopic surveillance; Atrophic gastritis; Intestinal metaplasia; Dysplasia; Gastric cancer

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Core Tip: Prevailing evidence affirms that *Helicobacter pylori* (*H. pylori*) eradication therapy could shift precancerous gastric conditions and positively confines gastric cancer risk during long-term endoscopic follow-up. Nonetheless, there is a yet unsolved dispute concerning the most useful individualized surveillance strategies following *H. pylori* eradication, based on malignant risk stratification. These aspects should be examined in the next future to establish and optimize the most cost-effective strategies for recognizing and managing *H. pylori*-positive patients with precancerous gastric conditions in the short- and long-term follow-up. Accordingly, new studies are required to sharpen the hazard stratification of patients with the greatest chance of progressing into gastric cancer.

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TO THE EDITOR

We read with great interest the review of Weng *et al*[1], pointing out the most recent literature supporting the impact of *Helicobacter pylori* (*H. pylori*) on the gastric mucosa alterations. Specifically, the authors assumed that, despite some controversy, current evidence suggests that *H. pylori* eradication treatment could reverse atrophic gastritis (AG) and intestinal metaplasia (IM) and favorably limits the appearance of gastric cancer (GC), particularly in long-term surveillance[1].

However, there is still unresolved debate regarding the best-individualized follow-up strategies, based on malignant risk stratification, due to uncertainty of current evidence and the role of *H. pylori* inflammatory changes in underestimating IM extension and dysplastic lesions at the index endoscopy (Table 1)[2-12].

In a recent article focused on the crucial role of high-resolution endoscopy with narrow-band imaging (NBI) for the optimal detection of IM, Dinis-Ribeiro M *et al*[13] criticized the recent U.S. guidelines that discourage short-interval endoscopic surveillance of patients with IM[14]. They supported and elaborated on the rationale behind the suggested 3-year-interval endoscopic surveillance of high-risk subjects with more extensive IM[13,14], for detecting early gastric neoplasia that, due to dismal prognosis of GC and increased aging of the population, can improve patient's survival [14]. Additionally, they stated that "*The majority of patients with gastric IM, those who during high-quality endoscopy were shown to have IM of limited severity and extent, confined to the antrum, and have a negative family history for GC do not require surveillance*"[13]. Notwithstanding, maybe this affirmation seems to neglect genetic/epigenetic/racial factors, personal habits and underlying comorbidity roles (*i.e.*, alcohol consumption, smoking, autoimmune and metabolic diseases) that can hold distinctive malignant potential, theoretically affecting subsequent endoscopic surveillance.

Notably, a recent prospective cohort study[12], including 85 Italian patients with *H. pylori*-related active gastritis, undergoing upper gastrointestinal endoscopy 6 mo following eradication therapy, demonstrated that high-resolution endoscopy with NBI doubled the rate of identifying histological low-grade dysplasia (LGD) missed at pre-treatment endoscopy, in a high-risk subgroup which had extensive atrophy and IM at baseline. In over 40% of patients, visible gastric lesions with LGD were found following *H. pylori* eradication was not identified at their first pre-treatment endoscopy, thus suggesting that inflammatory changes associated with active *H. pylori* infection hinder the correct detection of gastric LGD lesions[12].

Of interest, in cases of indefinite gastric dysplasia, or with "not visible" dysplasia diagnosed randomly throughout the stomach without endoscopic evidence of visible lesions, the prevailing guidelines recommend a necessary endoscopic reassessment using high-resolution endoscopy with NBI to rule out dysplasia on missed visible lesions[12,15].

Moreover, some authors consider high-resolution surveillance endoscopy with NBI as "*sufficient for a diagnosis of extensive IM or premalignant stomach even without biopsy sampling*"[13]. There is an established association between the endoscopic grading of

Table 1 Characteristics of patients included in the eleven selected studies applying endoscopic surveillance shorter than two years for the evaluation of precancerous gastric conditions following *Helicobacter pylori* eradication

	van der Hulst RW *et al*[2], 1997	Tucci A *et al* [3], 1998	Sung JJ *et al* [4], 2000	Annibale B *et al* [5], 2000	Ohkusa T *et al*[6], 2001	Oda Y *et al*[7], 2004	Annibale B *et al* [8], 2002	Yamada T *et al*[9], 2003	Iacopini F *et al* [10], 2003	Wambura C *et al* [11], 2004	Panarese *et al*[12], 2020
Study	Prospective	Retrospective	Prospective, randomized, placebo controlled trial	Observational, prospective study	Single-blind, uncontrolled prospective trial	Retrospective	Retrospective	Retrospective	Observational, prospective study	Observational, prospective study	Observational, prospective study
Country	Netherlands	Italy	China	Italy	Japan	Japan	Italy	Japan	Italy	Japan	Italy
Mean age, yr	49.2	50	51 (Median)	48.7	54	51	46 (Median)	52.6	55	51.2	56.1
Male, %	54	50	49.5	14.3	73	89.8	22.5	64.4	75	74.7	37.6
Overlap AAG	NA	0	NA	48.6	NA	NA	55	NA	NA	NA	26.3
Mean follow-up, mo	12	12	12	6-12	12-15	1-2	6-12	22	12	12	6
Total, *n*	106	10	226	25	115	59	40	87	40	107	85
Resolution of gastric acute/chronic inflammation in the antrum *n* (%)	S	10/10 (100)	S	25/25 (100)	NA	S	S	S	S	S	81/85 (95.3)
Resolution of gastric acute/chronic inflammation in the corpus, *n* (%)	S	NA	S	25/25 (100)	NA	S	S	S	S	S	81/85 (85.3)
Resolution of gastric acute/chronic inflammation in the fundus *n* (%)	NA	10/10 (100)	NA	NA	NA	NA	NA	NA	NA	NA	NA
Improvement of AG in the antrum, *n* (%)	NS	NS	NS	NS	34/38 (89)	NS	NS	NS	NS	S	NS
Improvement of AG in the corpus, *n* (%)	NS	NA	NS	NS	34/38 (89)	NS	8/40 (20) AG reversed	S	NA	S	NS

Improvement of AG in the fundus, *n* (%)	NA	S	NA	NA	NA	NA	NA	S	NA	NA	NA
Improvement of IM in the antrum, *n* (%)	NS	S	S	NS	28/46 (61)	NS	NS	NS	NS	NS	NS
Improvement of IM in the corpus, *n* (%)	NS	NA	NS	NS	28/46 (61)	NS	NS	NS	NA	S	NS
Improvement of IM in the fundus, *n* (%)	NA	NS	NA	NA	NA	NA	NA	NA	NA	S	NA
ECL pattern regression, *n* (%)	NA	NA	NA	8/15 (53.3) patients with AG in the body (12 mo after curing *H. pylori*)	NA	NA	NA	NA	NA	NA	36/39 (92.3)
LGD regression (or progression), *n* (%)	NA	NA	NA	1/1 (100) regression in a patient with AG in the body (12 mo after curing *H. pylori*)	NA	NA	NA	NA	NA	NA	The proportion of patients with histological diagnosis of LGD on random biopsies did not significantly change after *H. pylori* eradication [15 (17.6) *vs* 9 (10.6)]; the detection of LGD on visible lesions significantly increased after *H. pylori* eradication [0 (0) *vs* 19 (22.3)]
Conclusions	The usefulness of *H. pylori* eradication to regress precancerous lesions following 12 mo follow-up is uncertain	The natural history of AG can be modified by the eradication of *H.pylori*	At 12 mo, *H. pylori* eradication can block the histological progression of gastric mucosa alterations	*H. pylori* infection may be cured in patients with AG in the body with a partial reversing of its adverse outcomes on acid secretion and body ECL cell hyperplasia	After successful *H. pylori* eradication, precancerous lesions improved in most patients	After *H. pylori* eradication, neutrophil infiltration in the gastric mucosa improved relatively soon, while AG and IM did not display such tendency	In patients with AG of the body and *H. pylori* infection, the assessment of histological data after eradication is essential. In patients with maintaining body atrophy after *H. pylori* elimination, there is no association with the reversal of body atrophy, even at long-term surveillance	AG in the corpus can be improved after 12 mo following *H. pylori* eradication	*H. pylori* positive patients with AG, the overall oxidative damage of the gastric mucosa is more severe than that in *H. pylori* positive patients with nonatrophic gastritis	Eradication of *H. pylori* may decrease the risk of GC, due to the importance of *H. pylori* infection in the contributory role of gastritis in COX-2 expression and the dissociation between the processes of regression in gastritis and the reduction in COX-2	HR-WLE with NBI can be more reliable in diagnosing LGD on visible lesions after *H. pylori* elimination, presumably due to the removal of the underlying confounding effects of inflammatory and mucosal lymphoproliferative changes induced by *H. pylori* chronically active infection. Aged patients and those with autoimmune diseases (especially AAG) could be at higher risk for *H. pylori* persistent infection

AAG: Autoimmune gastritis; AG: Atrophic gastritis; IM: Intestinal metaplasia; ECL: Enterochromaffin-like cell; LGD: Low-grade dysplasia; *H. pylori*: *Helicobacter pylori*; NA: Not available; NS: Not significant; S: Significant improvement;

HR-WLE: High-resolution wight light endoscopy; NBI: Narrow band imaging.

gastric intestinal-metaplasia (EGGIM) and operative link on gastritis/intestinal-metaplasia assessment (OLGIM) stages in the assessment of the presence/extent of IM [12,13], and EGGIM stages ≥ 5 with OLGIM III/IV predicts early GC risk[12,13], although its reproducibility needs to be further confirmed in larger prospective studies as also expressed in the U.S. guidelines[14].

Nevertheless, even if feasible as a surveillance program in specialized referral centers, this strategy may not be widely applicable in endoscopy units that do not have access to such technologies. A targeted bioptic mapping seems more adequate for identifying mucosal gastric areas at risk of malignant transformation[12], despite the existing risk of overestimating OLGIM in patients with mild/focal IM. Concomitant *H. pylori*-related gastritis may limit the accuracy of EGGIM classification at the time of the initial endoscopy.

Advanced histological atrophy stages, even after *H. pylori* eradication, carry the highest risk for developing gastric neoplasia[12-15]. Nevertheless, recent long-term cohort studies from Eastern countries reported late development of GC during 5-14 years monitoring also in patients with none/mild gastric atrophy or antral IM, irrespectively of *H. pylori* eradication[12,16,17].

Notably, even with a high-resolution endoscope, if morphological changes do not appear, genetic and epigenetic changes in epithelial cells cannot be detected[18]. Specifically, epigenetic alterations (*i.e.*, aberrant DNA methylation), accumulate in cancers and also in normal-appearing tissues surrounding cancers[18]. Indeed, cross-sectional studies prove that aberrant methylation levels in normal tissues may be associated with cancer risk, particularly in chronic inflammation-associated cancers. Additionally, the relationship between miR-124a-3 DNA methylation abnormalities and similar trends for EMX1 and NKX6-1, have been judged extremely relevant predictors of developing authentic metachronous GCs[18].

Therefore, it is reasonable that high-quality endoscopy with histological assessment for the most accurate diagnosis of PGC[12] may be delayed, in selected high-risk patients who are symptomatic but have no alarm hallmarks for malignancy, after eradication of *H. pylori* diagnosed according to prior results of non-invasive tests had been achieved and serological autoimmunity biomarkers had been performed (*e.g.*, autoimmune AG-AAG), rather than applying the prevailing guidelines suggestion of operating targeted biopsies at initial endoscopy for histological estimation and determination of *H. pylori* status[12]. Such an approach is likely to enhance the PGC detection rate, especially for dysplastic lesions, reducing the confounding effect of *H. pylori*-related gastritis or AAG, and complies with the European guidelines[15], which recommend immediate high-quality endoscopy after the diagnosis of dysplasia without endoscopically visible lesions[12,15].

Therefore, we believe that further large prospective multicenter studies are still needed to identify additional risk factors of gastric malignancy development.

Moreover, multiple and evolving racial, ethnic, and immigration factors, may affect the risk of gastric neoplasia[19,20], calling also for the necessity of novel biomarkers for tailoring surveillance strategies to different patients.

These aspects should be considered in the next future to better define and optimize cost-effective strategies for identifying and managing *H. pylori*-positive patients with PGC in the short- and long-term follow-up.

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